RELATIONSHIP BETWEEN MUSCLE STIFFNESS OF THE SUPERFICIAL SHOULDER MUSCULATURE AND SUBACROMIAL SPACE DISTANCE

Pamela Rachelle Young

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Approved by:

Joseph Myers

Troy Blackburn

Elizabeth Hibberd

Timothy Mauntel

Terri Jo Rucinski

ABSTRACT

Pamela Rachelle Young: Muscle Stiffness of the Superficial Shoulder Musculature and its Relationship to Subacromial Space Distance
(Under the direction of Dr. Joseph Myers)

Side-to-side differences in subacromial space distance, muscle stiffness, and pectoralis minor length (PML) and the predictive ability of these physical characteristics to predict subacromial space distance in overhead athletes were investigated. Fifty collegiate overhead athletes completed one testing session of bilateral measurements of the subacromial space distance, muscle stiffness, and PML. The dominant arm exhibited a shorter PML (p=0.02) and greater stiffness of the teres minor (1.50kg: p<0.005; 1.75kg: p<0.005; 2.0kg: p<0.005), posterior deltoid (1.50kg: p<0.005; 1.75kg: p=0.02; 2.0kg: p<0.005), and lower trapezius (1.50kg: p=0.04; 1.75kg: p=0.03; 2.0kg: p=0.03) compared to the non-dominant arm. Neither stiffness nor PML predicted subacromial space distance in either limb of healthy overhead athletes. These side-to-side differences could provide clinicians with a screening tool to identify individuals with asymmetries. Further research is needed to determine the relationship between stiffness of the superficial shoulder musculature and subacromial space distance in overhead athletes with subacromial impingement syndrome.

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CHAPTER I

INTRODUCTION

An Overview

Shoulder pain is common among overhead athletes, particularly among competitive baseball, volleyball, tennis, and swimming athletes (Borich et al., 2006). The prevalence of shoulder pain among competitive overhead athletes is reported to be between 10-30% (Diederichsen et al., 2009). It is common for overhead athletes to describe a vague sense of discomfort, often an achy pain that developed over time, in their shoulder. This has often been attributed to several different pathological findings with subacromial impingement syndrome (SAIS) being one of the more frequently reported causes of shoulder pain (McClure, Bialker, Neff, Williams, & Karduna, 2004).

Subacromial Impingement Syndrome

SAIS accounts for 44-65% of all shoulder pain related doctors' visits (de Witte et al., 2011; McClure et al., 2004; Michener, McClure, & Karduna, 2003; Umer, Qadir, & Azam, 2012). SAIS was first described by Neer (Neer, 1983) as three progressive stages of rotator cuff tendinopathy. Stage I involves inflammation of the subacromial bursa and the rotator cuff, particularly the supraspinatus, with minor evidence of tendon degeneration and typically affects people under age 25. Stages II and III involve structural changes due to

repetitive overload and are more common in people ages 25 and older (Neer, 1983). Collegiate overhead athletes primarily experience the early symptoms of SAIS and it is relatively uncommon for Stage III impingement to occur in the collegiate athlete (Cowderoy, Lisle, & O'Connell, 2009).

Currently, SAIS is classified into two main categories: structural and functional (Page, 2011). Structural impingement, or primary impingement, stems primarily from anatomical factors that predispose the athletic shoulder to subacromial impingement including acromion morphology and coracoacromial ligament thickening (Bigliani & Levine, 1997; Magaji, Singh, & Pandey, 2012; Neer, 1983; Tibone et al., 1985). The current method of treatment for primary impingement includes surgical intervention with subacromial decompression and/or anterior acromioplasty (Bigliani & Levine, 1997; Magaji et al., 2012; Neer, 1983). However, the overhead athlete more commonly experiences the effects of functional rather than structural impingement due to the repetitive nature of his/her sport (Cowderoy et al., 2009; Page, 2011). Functional impingement, or secondary impingement, is the compression of the long head of the biceps tendon, the subacromial bursa, and/or the supraspinatus tendon between the humeral head and the acromion process as a result of superior migration of the humeral head during elevation of the arm (Cools, Cambier, & Witvrouw, 2008; Desmeules, Minville, Riederer, Cote, & Fremont, 2004; Diederichsen et al., 2009; Ludewig & Cook, 2000; Neer, 1983; Page, 2011). Secondary impingement manifests as a result of altered glenohumeral and scapular kinematics (Burkhart, Morgan, & Kibler, 2003; Cools et al., 2008; Diederichsen et al., 2009; Ludewig & Cook, 2000; Maenhout, Van Eessel, Van Dyck, Vanraes, & Cools, 2012; McClure et al., 2004; Page, 2011). Recent literature suggests posterior capsule and muscle tightness, resulting in decreased internal

rotation range of motion (ROM), has been linked with SAIS (Maenhout et al., 2012; Myers, Laudner, Pasquale, Bradley, & Lephart, 2006; Tyler, Nicholas, Roy, & Gleim, 2000). The overall effect of each of these etiologies is a narrowing of the subacromial space distance, ultimately increasing the likelihood of pathological compression of the structures within (Burkhart et al., 2003; Maenhout et al., 2012).

Subacromial Space Distance

The subacromial space is defined as the space between the humeral head and coracoacromial arch (Cowderoy et al., 2009; Neer, 1983). The coracoacromial arch is formed by the acromion process, the coracoid process, and the coracoacromial ligament (Bigliani & Levine, 1997; Cowderoy et al., 2009; Michener et al., 2003; Neer, 1983). The subacromial bursa, supraspinatus tendon, and long head of the biceps tendon lie within this space and are susceptible to pathological compressions with subacromial space distance reductions (Michener et al., 2003). At 0° of flexion and abduction, healthy shoulders demonstrate a subacromial space distance of approximately 10mm which narrows to approximately 5mm with further arm elevation to 60° and 120° of abduction (Flatow et al., 1994; Ludewig & Cook, 2000). Shoulders with impingement demonstrate even further reductions of this space at 90° of shoulder abduction (mean 1.4 mm ± 1.1 mm) (Graichen, Bonel, et al., 1999). Narrowing of the subacromial space distance has been partially attributed to abnormal glenohumeral and scapular kinematics, such as increased superior translation of the humeral head (Deutsch, Altchek, Schwartz, Otis, & Warren, 1996), decreased internal rotation (Borich et al., 2006; Maenhout et al., 2012), increased anterior scapular tilting (Borich et al., 2006; Hébert, Moffet, McFadyen, & Dionne, 2002; Ludewig & Cook, 2000), increased

scapular upward rotation (Karduna, Kerner, & Lazarus, 2005), and increased protraction of the scapula (Solem-Bertoft, Thuomas, & Westerberg, 1993). Altered scapular kinematics that are related to subacromial impingement have also been linked with altered muscle activity and a shortened pectoralis minor length (PML). Graichen et al. (Graichen et al., 1998; Graichen, Stammberger, Englmeier, Reiser, & Eckstein, 1999) identified increased muscle activity of the shoulder abductors while Borstad et al. (Borstad & Ludewig, 2005) identified a shortened PML as contributing factors to a narrower subacromial space distance. In addition to these known contributors, it is likely that muscle stiffness of the superficial shoulder musculature may also play a role in reducing the subacromial space distance (Hung, Hsieh, Yang, & Lin, 2010). A narrower subacromial space distance increases the risk of injury because the limited available space increases the compressive contact of the aforementioned structures, ultimately predisposing the shoulder to SAIS.

Muscle Stiffness and Abnormal Shoulder Kinematics

Muscle stiffness is the resistance of tissue to change in position or length and is defined as the ratio of change in force to change in length (Blackburn, Norcross, & Padua, 2011; Hung et al., 2010; Huxel et al., 2008; Myers & Lephart, 2000; Oatis, 1993; Olds, McNair, Nordez, & Cornu, 2011). Much of the research in regards to stiffness and the shoulder concerns either the pathological "frozen," or stiff shoulder (Hung et al., 2010), or the benefits of muscle stiffness in relation to pathological instability of the glenohumeral joint (Huxel et al., 2008; Olds et al., 2011). Stiff shoulder occurs as the result of muscular and capsular contracture which ultimately limits total glenohumeral ROM. In a recent study,

Hung et al. (Hung et al., 2010) reported significant glenohumeral internal rotation deficits in participants with stiff shoulder.

Several other studies have examined the influence of muscle stiffness in subjects with glenohumeral instability. These studies found dynamic muscle stiffness at the shoulder is essential for maintaining glenohumeral stability during functional activity (Huxel et al., 2008; Myers & Lephart, 2000; Olds et al., 2011). Patients with recurrent glenohumeral instability have demonstrated significantly less active muscle stiffness and a relative increase in dislocation episodes (Olds et al., 2011). Active muscle stiffness also assists in resisting stretching episodes, heightens muscle spindle sensitivity, and reduces the amount of delay prior to reflexive stabilization of a joint, overall creating a more functionally stable joint (Myers & Lephart, 2000).

Research clearly identifies the cascade of subacromial impingement as a progression from posterior shoulder tightness to internal rotation deficits (Hung et al., 2010) to altered glenohumeral and scapular kinematics (i.e. increased scapular upward rotation (Karduna et al., 2005), anterior tilting (Borich et al., 2006), and internal rotation (Ludewig & Cook, 2000)) and finally to subsequent reductions in subacromial space (Graichen, Bonel, et al., 1999; Maenhout et al., 2012). Because glenohumeral internal rotation deficits are theorized to be a major contributing factor to alterations in kinematics and ultimately reduction in subacromial space distance, stiffness in muscles that function to externally rotate the shoulder (thus limiting internal rotation range of motion) may potentially be correlated to decreased subacromial space distance and a greater risk of SAIS. Theoretically, stiffness of the infraspinatus, teres minor, and posterior deltoid would contribute to limited internal rotation ROM (Hung et al., 2010), stiffness of the latissimus dorsi would potentially limit

glenohumeral abduction and external rotation of the humerus, and increase scapular upward rotation during abduction (Laudner & Williams, 2013), stiffness of the upper trapezius would create an elevated scapular posture, stiffness of the lower trapezius would increase scapular upward rotation, and stiffness of the pectoralis major and pectoralis minor would limit external rotation of the humerus and scapular posterior tilting during shoulder abduction (Terry & Chopp, 2000). Therefore, it is possible to theorize that each of these can contribute to a functional narrowing of the subacromial space distance. Theoretically, muscle stiffness of the superficial shoulder musculature could be the predisposing factor that instigates this cascade of injury.

Purpose and Clinical Relevance

Research clearly identifies the cascade of subacromial impingement as a progression from posterior shoulder tightness to internal rotation deficits (Hung et al., 2010) to altered glenohumeral and scapular kinematics (i.e. increased scapular upward rotation (Karduna et al., 2005), anterior tilting (Borich et al., 2006), and internal rotation (Ludewig & Cook, 2000)) and finally to subsequent reductions in subacromial space (Graichen, Bonel, et al., 1999; Maenhout et al., 2012). Because posterior shoulder tightness and alterations in glenohumeral and scapular kinematics are related to reductions in subacromial space distance, stiffness in the muscles that can contribute to abnormal glenohumeral and scapular kinematics may potentially be correlated to decreased subacromial space distance and an increase in SAIS. While there is a theoretical link between muscle stiffness and subacromial space distance, to date there are no previous studies that identify this relationship in either healthy or non-healthy shoulders; therefore, it is important to first determine if there is a

relationship within the healthy overhead athlete's shoulder. The purpose of this study was to evaluate side-to-side differences in subacromial space distance, muscle stiffness, and PML, as well as to determine the ability of these physical characteristics to predict subacromial space distance. Understanding the contribution of each of these to subacromial space distance may provide clinicians with valuable information regarding potential risk factors for decreasing subacromial space distance and developing SAIS. Through a better understanding of these possible risk factors, clinicians could develop better intervention and prevention programs that could ultimately reduce the likelihood of instigating the subacromial impingement cascade of injury.

Research Questions

RQ 1: What are the relative contributions of superficial shoulder musculature stiffness and PML to subacromial space distance?

RQ 2: Is there a difference in muscle stiffness values between dominant and non-dominant shoulders?

RQ 3: Is there a difference in subacromial space distance between dominant and non-dominant shoulders?

RQ 4: Is there a difference in pectoralis minor length between dominant and non-dominant shoulders?

Variables

- Predictor:
    - Muscle stiffness

        - Teres minor
        - Infraspinatus
        - Posterior deltoid
        - Upper trapezius
        - Lower trapezius
        - Latissimus dorsi
    - Pectoralis minor length
- Criterion:
    - Subacromial space distance
- Independent:
    - Dominant Arm (DOM)
    - Non-dominant Arm (NON)
- Dependent:
    - Muscle Stiffness
        - Teres minor
        - Infraspinatus
        - Posterior deltoid
        - Upper trapezius
        - Lower trapezius
        - Latissimus dorsi
    - Pectoralis minor length

- Subacromial space distance

Hypotheses

H1: There will be a set of variables that significantly predict subacromial space distance with relative contributions from greatest to smallest as:

- Pectoralis minor length
- Posterior deltoid stiffness
- Infraspinatus stiffness
- Teres minor stiffness
- Upper trapezius stiffness
- Latissimus dorsi stiffness
- Lower trapezius stiffness

H2: The dominant arm will demonstrate greater muscle stiffness compared to the non-dominant arm.

H3: The dominant arm will demonstrate lesser subacromial space distance compared to the non-dominant arm.

H4: The dominant arm will demonstrate a shorter pectoralis minor length compared to the non-dominant arm.

Null Hypotheses

H1: Greater muscle stiffness of the infraspinatus, teres minor, upper and lower trapezius, and latissimus dorsi and a shorter pectoralis minor length will not predict a narrowing of the subacromial space distance.

H2: There will be no significant difference in muscle stiffness values between dominant and non-dominant arms.

H3: There will be no significant difference in subacromial space distance values between dominant and non-dominant arms.

H4: There will be no significant difference in pectoralis minor length values between dominant and non-dominant arms.

Statistical Hypotheses

- Hypothesis 1:
  - H_0: $r = 0$
  - H_A: $0 \geq r \geq -1.0$
- Hypothesis 2:
  - Muscle Stiffness H_0: $\mu_{Dom} = \mu_{Non}$
  - Muscle Stiffness H_A: $\mu_{Dom} > \mu_{Non}$
- Hypothesis 3:
  - Subacromial space distance H_0: $\mu_{Dom} = \mu_{Non}$
  - Subacromial space distance H_A: $\mu_{Dom} < \mu_{Non}$
- Hypothesis 4:
  - Pectoralis Minor Length H_0: $\mu_{Dom} = \mu_{Non}$
  - Pectoralis Minor Length H_0: $\mu_{Dom} < \mu_{Non}$

Operational Definitions

- Healthy shoulders: Participants without any history of shoulder surgery and without current or history of shoulder injury within the previous year.
- Shoulder injury: Shoulder impairments in either the dominant or non-dominant arm which limited their normal activities for three consecutive days within the past six months.
- Dominant arm: The arm with which the participant would throw a ball for maximal distance.

- Subacromial space distance: The space between the proximal humerus, most lateral portion of the acromion, and coracoacromial ligament.
- Muscle stiffness: The resistance of muscle tissue to changes in length or position. The ratio of change in force to the change in muscle length.
- Pectoralis minor length: The measurement of the pectoralis minor from the sternocostal junction of the fourth rib to the coracoid process.

Assumptions

- Participants will follow directions when completing the tasks required during the study.
- A myotonometer is a valid and reliable tool used to measure muscle stiffness.
- A Vernier caliper is a valid and reliable tool used to measure pectoralis minor length.
- A digital inclinometer is a reliable measure of glenohumeral range of motion.

Delimitations

- Only subjects between the ages of 18-25 years will be used in order to control for possible degenerative changes that occur with age.
- The shape of the acromion will not be investigated.

Limitations

- The 2D US measurements of subacromial space cannot capture the effects on subacromial space during 3D movement normal to the athletic shoulder.
- Myotonometer measurements can be compromised by proximity of other muscles.

CHAPTER II

A REVIEW OF THE LITERATURE

Introduction

Shoulder pain is frequently reported among collegiate overhead athletes, particularly among those involved in swimming, baseball, volleyball, and tennis due to the demands of their sport (Diederichsen et al., 2009). Lo et al. (Lo, Hsu, & Chan, 1990) reported that the prevalence of shoulder pain in Chinese athletes involved in upper arm sports was 43.8% with 66.1% of them were under the age of 25 and 41.9% having competed at the elite or collegiate level. Of the athletes reporting shoulder pain as their primary complaint, volleyball and swimming ranked the highest with tennis, basketball, and badminton equally distributed with 10 athletes each. One of the more common injuries reported in conjunction with shoulder pain is shoulder impingement (McClure et al., 2004). This pathology can be debilitating to an athlete's performance, activities of daily living, and overall feelings of well being. The pathological anatomical and biomechanical contributing factors to subacromial impingement have been addressed throughout the literature. These contributors include acromion morphology (Bigliani & Levine, 1997), abnormal glenohumeral and scapular kinematics (Deutsch et al., 1996; Ludewig & Cook, 2002; Yamaguchi et al., 2000), and posterior shoulder tightness (Maenhout et al., 2012; Myers et al., 2006). The current literature has only recently proposed the interaction effect of the anatomical and biomechanical contributors to a

narrowing of the subacromial space distance. The purpose of this review of the literature is to analyze and discuss each of these factors as well as others that may be considered predisposing risk factors for developing subacromial impingement. This review of the literature will seek to demonstrate the gaps in knowledge and understanding of how subacromial space is directly affected by modifiable physical characteristics of the shoulder.

Muscle Stiffness

Muscle stiffness is the resistance of tissue to change in position or length and is defined as the ratio of change in force to change in length (Blackburn et al., 2011; Oatis, 1993). This infers that stiffer muscles surrounding the shoulder girdle may limit the amount of free movement of the scapula and humerus as compared to more compliant/less stiff muscles, ultimately affecting normal glenohumeral and scapular kinematics. However, research has elucidated the need for dynamic muscle stiffness as it relates to dynamic stability of the shoulder. Dynamic muscle stiffness at the shoulder is essential for maintaining glenohumeral stability during functional activity, protecting the joint from instability episodes (Huxel et al., 2008; Myers & Lephart, 2000; Olds et al., 2011). Huxel et al. (Huxel et al., 2008) noted that shoulder stiffness was 77% greater with active contraction as compared to passive rest regardless of joint position and suggested that moderate levels of torque production and stiffness remain relatively constant. The authors went on to suggest that consistent levels of stiffness are more desirable and can contribute to supplementing joint stability, particularly within the unstable joint. Olds et al. (Olds et al., 2011) observed a lower level of stiffness in unstable shoulders at 30% and 50% maximal voluntary strength levels with perturbations into horizontal abduction. Myers et al. (Myers & Lephart, 2000)

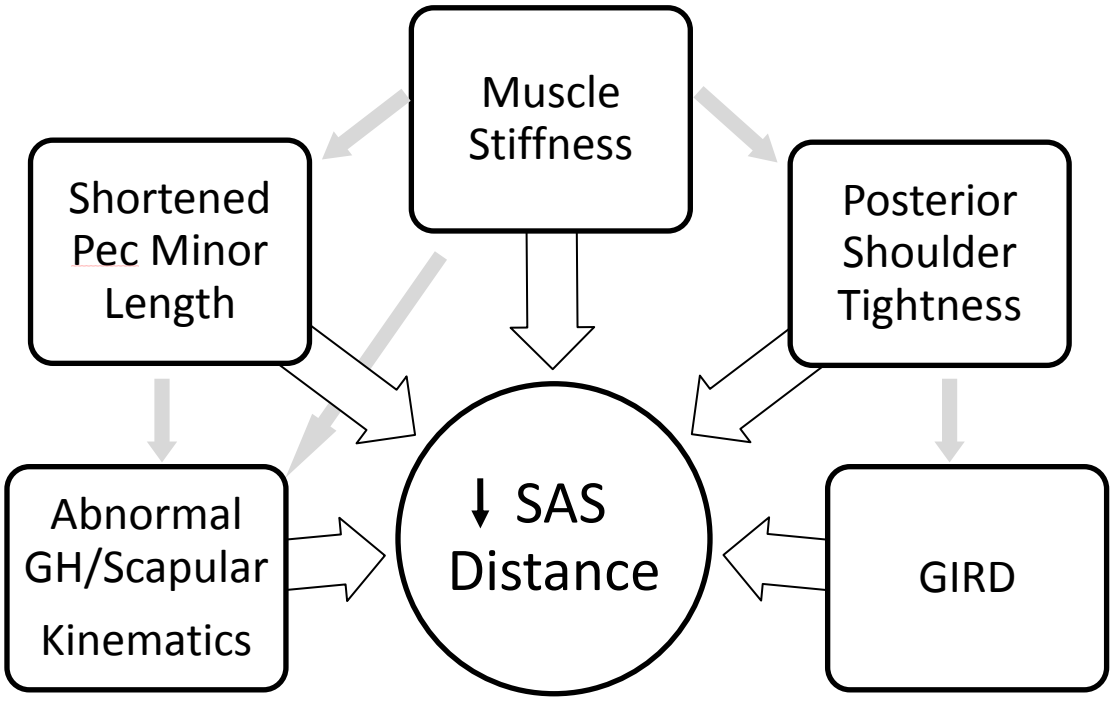
comments on the influence of the sensorimotor system on the functional stability of the shoulder and suggests that the preparatory muscle activation component of neuromuscular control contributes to increasing active muscle stiffness and subsequently improves dynamic glenohumeral stability. Less research has been conducted considering the effects of muscle stiffness on the stable glenohumeral joint and scapulothoracic joint and particularly how it relates to subacromial impingement syndrome (SAIS).

Determining the influence of stiffness of muscles acting on the glenohumeral and scapulothoracic joints is essential to further understanding of SAIS. In particular, the stiffness of the infraspinatus, teres minor, posterior deltoid, upper and lower trapezius, and latissimus dorsi. Each of these muscles contributes to overhead motion and may affect subacromial space distance (Table 1). Greater stiffness of each of these muscles, theoretically, will create abnormal glenohumeral and scapular kinematics during overhead movements. For instance, greater infraspinatus, teres minor, and posterior deltoid stiffness will create glenohumeral internal rotation deficits (GIRD) (Hung et al., 2010). Greater latissimus dorsi stiffness and lower trapezius stiffness will increase scapular upward rotation (Karduna et al., 2005; Laudner & Williams, 2013) and greater upper trapezius stiffness will posture the scapula in a position of elevation. A shortened PML, which may be as a result of pathological increases in tissue stiffness, also contributes to greater anterior tilting and internal rotation of the scapula, subsequently decreasing the subacromial space distance (Borstad & Ludewig, 2005; Ludewig & Cook, 2000). Theoretically, these limitations induced by tissue stiffness can functionally narrow the subacromial space. Overall, this paper proposes a cascade of injury that stems from muscle stiffness of the superficial shoulder musculature and ultimately leads to SAIS (Figure 1).

TABLE 1: Muscles and their Function

Muscle	Function
Infraspinatus	Externally rotates the humerus; cuffs the humeral head into the glenoid fossa
Teres Minor	Externally rotates the humerus; cuffs the humeral head into the glenoid fossa
Posterior Deltoid	Extends and externally rotates the humerus
Upper Trapezius	Elevates and upwardly rotates the scapula
Lower Trapezius	Depresses and upwardly rotates the scapula
Latissimus Dorsi	Adducts, extends, and internally rotates the humerus
Pectoralis Minor	Protracts and downwardly rotates the scapula

FIGURE 1: Proposed Subacromial Impingement Cascade of Injury



Subacromial Impingement Syndrome

Epidemiology

The term "shoulder impingement" encompasses three main pathologies of the shoulder: 1) internal impingement, 2) coracoid impingement, and 3) subacromial impingement. Internal impingement is the compression of the articular surface of the supraspinatus and infraspinatus between the humeral head and posterior superior glenoid rim with the shoulder in a position of 90° of abduction and external rotation (Davidson, Elattrache, Jobe, & Jobe, 1995). Coracoid impingement is the compression of the subscapularis tendon between the coracoid process and lesser tuberosity of the humerus typically with the shoulder in a position of glenohumeral elevation, horizontal adduction, and internal rotation (Okoro, Reddy, & Pimpelnarkar, 2009). Subacromial impingement is the compression of the long head of the biceps tendon, the supraspinatus, and the subacromial bursa between the humeral head and the acromion process. Although each of these impingements may be present in the overhead athlete, the focus of this research project is to evaluate the relationship between subacromial space distance and the development of SAIS.

SAIS is a common pathology of shoulder pain accounting for 44-65% of all shoulder pain related doctor's visits (de Witte et al., 2011; McClure et al., 2004; Michener et al., 2003; Umer et al., 2012). SAIS commonly affects populations in which a primary function of daily activities includes repetitive overhead activity. This is most commonly seen in competitive overhead athletes, particularly those involved in swimming, tennis, baseball, and volleyball (Borich et al., 2006), and in the industrial workplace, particularly among construction workers, welders, and steelworkers (Ludewig & Cook, 2000). Tibone et al. (Tibone et al., 1985) highlighted the prevalence of SAIS within overhead dominant sports. In a study

involving 35 shoulders, the authors identified 17 pathological shoulders in baseball, 6 in swimming, and 4 in tennis, with the remaining distributed between football, skiing, surfing, and racquetball. Shoulder pain, often linked with SAIS, in USA competitive swimming has been reported at rates as high as 38-75% (McMaster & Troup, 1993). Other studies have also directly examined the incidence of shoulder impingement in competitive baseball athletes (Mihata et al., 2012; Myers et al., 2006).

Pathoanatomy and Biomechanics of the Shoulder

The glenohumeral joint and scapulothoracic joints are the primary joints involved in SAIS. The alteration of normal movement at these joints contributes to the development of SAIS in the overhead athlete. The ball and socket glenohumeral joint has six degrees of freedom allowing a variety of movement necessary for activities of daily living. This is particularly important in facilitating the motions commonly utilized in overhead dominant athletics. Throwing and hitting athletes often operate out of a position of abduction and external rotation, a position often implicated in pathologic conditions such as SAIS. Normal glenohumeral kinematics requires external rotation in order for the greater tuberosity to clear the acromion and therefore enable optimal shoulder flexion and abduction (Flatow et al., 1994; Neagle & Bennett, 1994). The infraspinatus, teres minor, and posterior deltoid function as primary external rotators as well as humeral head stabilizers and experience a resultant increase in eccentric load during the deceleration phase of throwing. The deltoid and supraspinatus are the primary movers for humeral abduction. These muscles work in concert with each other in order to abduct the humerus while the infraspinatus, teres minor, and subscapularis function as opposing forces that simultaneously keep the humeral head centered on the glenoid fossa (Sharkey & Marder, 1995). Within the first 30-60° of elevation,

concurrent superior translation of the humeral head 1-3mm on the glenoid fossa occurs in order to facilitate elevation of the glenohumeral joint. For the remainder of the movement, the humeral head remains relatively centered on the glenoid fossa (Neumann, 2010; Terry & Chopp, 2000; Umer et al., 2012). These dynamic force couples, the deltoid and supraspinatus in conjunction with the other three rotator cuff muscles, serve to stabilize the humeral head on the glenoid fossa effectively limiting the amount of pathological superior humeral translation that would contribute to reducing the subacromial space distance and increasing the risk of SAIS (Terry & Chopp, 2000).

Normal scapulothoracic (ST) joint function is a crucial component of enabling normal movements of the shoulder in overhead activity. In order to achieve optimal shoulder elevation the scapula must elevate, upwardly rotate, externally rotate, and posteriorly tilt. The primary muscles responsible for these movements are the trapezius, rhomboids, levator scapulae, serratus anterior, and pectoralis minor (Terry & Chopp, 2000). The trapezius is a broad tri-portioned muscle that extends from the base of the skull to the scapular spine, clavicle, acromion, and spinous processes of the lower thoracic vertebrae, functioning as a scapular retractor and upward rotator. The rhomboids work concurrently with the middle trapezius as scapular retractors, while the levator scapulae work in conjuction with the upper trapezius to upwardly and internally rotate the scapula. The serratus anterior originates on the first nine ribs and inserts from the superior to inferior angle on the scapula. Contraction of the serratus anterior causes protraction and upward rotation of the scapula. The pectoralis minor also originates on the ribs and inserts at the coracoid process of the scapula and functions to protract, and downwardly rotate the scapula. These normal scapular movements establish an appropriate separation between the acromion and the humeral head, ultimately

maintaining normal subacromial space distance (Hébert et al., 2002; Ludewig & Braman, 2011; Terry & Chopp, 2000).

The subacromial space is defined as the space between the humeral head and the coracoacromial arch. The coracoacromial arch is formed by the acromion process, the coracoid process, and the coracoacromial ligament (Bigliani & Levine, 1997; Cowderoy et al., 2009; Michener et al., 2003; Neer, 1983). The subacromial space houses three primary structures often compromised in SAIS including the supraspinatus tendon, the long head of the biceps tendon, and the subacromial bursa (Michener et al., 2003). In a healthy shoulder, a normal subacromial space distance is between 6-14mm, but is affected by normal overhead movements. At 30° of abduction, the subacromial space is at its maximum width, whereas it narrows to its minimum at 120°, with the majority of spatial reductions occurring between 60° and 120° of abduction. Rotation at 90° of abduction also has a significant effect on subacromial space distance. The subacromial space is at its maximum width in internal rotation and at its minimum in external rotation. However, the vector of the minimal distance of the subacromial space in internal rotation passes directly through the supraspinatus tendon at the location where most rotator cuff tears occur, indicative of greater risk of injury during internal rather than external rotation (Graichen, Stammberger, et al., 1999). The width of this space is affected by overhead movements and subsequently can affect the aforementioned structures.

One example of functional overhead movement is exemplified in the baseball pitch. The throwing motion involves complex coordination of movement of the humerus and scapula. During the cocking phase the humerus is abducted, externally rotated, and horizontally abducted while the scapula retracts in order to form a stable base for the

humerus to act upon. The acceleration phase begins when the humerus begins to internally rotate in order to generate and transfer force to the ball upon release. Maintaining a position of abduction, the humerus internally rotates while the scapula protracts, preserving that stable base for the humerus, and begins the conversion of eccentric to concentric force at the anterior shoulder and concentric to eccentric force at the posterior shoulder. The final phase of the throwing motion is the violent and forceful deceleration phase. The humerus begins its migration from horizontal abduction to horizontal adduction while continuing its internal rotation moment about the shoulder. Meanwhile the scapula continues to protract and the posterior shoulder muscles create a forceful eccentric contraction to slow down the rotational velocity generated during the acceleration phase (Dillman, Fleisig, & Andrews, 1993; Meister, 2000).

These dynamic and functional motions at the shoulder ultimately affect the subacromial space. When the humerus abducts and the scapula upwardly rotates and protracts as seen in the throwing motion, the subacromial space naturally narrows, but maintains a width that will not predispose the internal structures to pathological compression (Graichen, Stammberger, et al., 1999; Ludewig & Cook, 2002). During abduction, normal translations of the humerus on the glenoid involve a superior humeral glide approximately 1-3mm within the first 30-60° of glenohumeral elevation (Ludewig & Cook, 2002; Umer et al., 2012). For the remainder of the movement, the humeral head remains relatively centered on the glenoid fossa. However, functional narrowing of the subacromial space can become injurious with alterations in glenohumeral and scapular kinematics.

Etiology

SAIS occurs as the result of both anatomical and biomechanical variations in the

glenohumeral and scapulothoracic joints. SAIS is often divided into two categories based on these anatomical versus biomechanical differentiations that predispose the athletic shoulder to pathological impingement: 1) Primary and 2) Secondary impingement. Primary impingement is the result of variations in the coracoacromial arch that impinge on the structures occupying the subacromial space. Secondary impingement, however, occurs as the result of a cascade of biomechanical abnormalities at the shoulder. The most common cause of secondary impingement is the instability of the glenohumeral joint commonly observed in the high school and collegiate overhead throwing athlete (Cowderoy et al., 2009; Tyler et al., 2000).

The structural changes of the coracoacromial arch associated with primary impingement most frequently involve variations in the inherent shape of the acromion process. Bigliani et al. (Bigliani & Levine, 1997) classified three different types of acromion morphology: Type I (flat), Type II (curved), and Type III (hooked). Research has also identified a pseudo-Type III acromion morphology resulting from an increase in osteoblastic activity at the anterior acromion contributing to the formation of an exostosis. This spurring of the anterior acromion is not typically present in the younger athletic shoulder, but rather is seen in middle aged adults (Cowderoy et al., 2009). Subacromial impingement has been attributed to the encroachment of the acromion process into the subacromial space (Neer, 1983). The hooked acromial morphology protrudes into the subacromial space thereby increasing the compressive forces on the structures located within that space (Bigliani & Levine, 1997). Subacromial decompression and anterior acromioplasty are common surgical techniques utilized to reduce the compressive forces applied on the subacromial structures by the acromion process (Bigliani & Levine, 1997; Magaji et al., 2012; Neer, 1983). Coracoid

ligament thickening is another less common anatomical variation that can contribute to impingement of the structures within the subacromial space (de Witte et al., 2011). Surgical intervention is the only option for correcting bony abnormalities; therefore, the focus of this study will be on the modifiable muscular characteristics commonly implicated in SAIS.

Secondary impingement, unlike primary impingement, involves biomechanical abnormalities that lead to compression of the structures within the subacromial space. Secondary impingement can be further subdivided into two other categories: intrinsic and extrinsic impingement. Intrinsic impingement is the degeneration of the rotator cuff, particularly the supraspinatus, as a result of overuse, tensile overload, and/or insufficient stability and excessive mobility of the glenohumeral joint. This ultimately engenders imbalances of the scapular muscles and abnormal scapulohumeral rhythm contributing to ischemic changes in the supraspinatus tendon (de Witte et al., 2011; Michener et al., 2003). Extrinsic impingement is the narrowing of the subacromial space thereby causing a mechanical compression of the rotator cuff, subacromial bursa, and long head of the biceps tendon (de Witte et al., 2011; Umer et al., 2012). These typically stem from alterations in the biomechanics and kinematics of the glenohumeral and scapulothoracic joints.

Altered Glenohumeral and Scapular Kinematics

Alterations in glenohumeral kinematics often involve pathological superior translations of the humeral head on the glenoid fossa; an alteration often observed within individuals with impingement. Individuals with impingement demonstrate excessive superior translation of 1.0-1.2mm as evidenced on radiographic images (Deutsch et al., 1996). Those unaffected by impingement and those with stage II impingement demonstrate a centrally located starting position of the humerus on the glenoid fossa (mean -0.4mm; mean -0.2mm)

as compared to those with stage III impingement (full rotator cuff tears) that presented with the humerus located above the glenoid's center (mean +0.3mm) (Deutsch et al., 1996). Other studies have also identified excessive and abnormal superior humeral head translation during glenohumeral elevation in subjects with impingement (Ludewig & Cook, 2002; Yamaguchi et al., 2000).

In addition to alterations in humeral head movement, aberrations in scapular kinematics are related to SAIS. SICK scapula, first defined by Burkhart et al. (Burkhart et al., 2003), refers to **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement. There are three primary patterns of scapular dyskinesis and Type III is most often related to SAIS. In Type III SICK scapula, the malpositioned scapula sits in a protracted and anteriorly tilted position making the inferomedial border appear more prominent and makes the affected shoulder appear lower than the contralateral side. As a result of this protraction and anterior tilt, the pectoralis minor and short head of the biceps become adaptively tight and short serving to maintain and increase the malposition of the scapula. This altered scapular kinematic decreases the available subacromial space and subsequently increases the risk of impingement (Burkhart et al., 2003).

Abnormal muscle activation of the serratus anterior, upper and lower trapezius, rotator cuff, and middle deltoid contributes to alterations in scapular kinematics such as decreased posterior tipping, increased upward rotation, and elevation of the scapula during glenohumeral abduction increasing the risk of impinging the subacromial structures (Ludewig & Cook, 2000). Upper crossed syndrome, first described by Vladimir Janda, refers to the muscle imbalances between the anterior and posterior muscles acting on the thoracic

and cervical spine. These imbalances of tight pectorals, suboccipitals, upper trapezius, and levator scapulae, and weak cervical flexors, rhomboids, and lower trapezius create a forward head and rounded shoulders posture often implicated in SAIS (Janda, 1988; Page, 2011). Individuals with greater forward head and rounded shoulders posture demonstrate greater anterior tilting, internal rotation, and upward rotation of the scapula as well as concurrent reductions in serratus anterior activation (Thigpen et al., 2010). A shortened PML orients the scapula in a more protracted position. Protraction of the scapula diminishes the subacromial space thereby increasing the amount of contact pressure on the structures within (Borstad & Ludewig, 2005). Internal rotation of the scapula also decreases the subacromial space and is a patterned behavior in shoulders with symptoms of SAIS (Ludewig & Cook, 2000). Recent research has also identified an increase in latissimus dorsi tightness, or stiffness, in swimmers that contributes to greater upward rotation of the scapula during the humeral elevation that occurs during the repetitive performance of the swimming strokes (Laudner & Williams, 2013). Greater scapular upward rotation decreases the amount of subacromial clearance and subsequently increases subacromial contact forces (Karduna et al., 2005). A study by McClure et al. (McClure, Michener, & Karduna, 2006) demonstrated slightly greater upward rotation in subjects with SAIS. Interestingly enough, other studies have found that shoulders with impingement typically demonstrate decreased scapular upward rotation (Ludewig & Cook, 2000; Su, Johnson, Gracely, & Karduna, 2004) and this may be a compensatory reaction in order to decrease the amount of subacromial contact occurring during humeral elevation.

Research has also identified posterior shoulder tightness as a predominant factor contributing to the pathological cascade of SAIS. Tightness, or stiffness, of the posterior

shoulder stems from a tight posterior capsule, posterior rotator cuff, and posterior deltoid (Harryman et al., 1990; Myers et al., 2006; Tyler et al., 2000). Stiffness of the infraspinatus, teres minor, and posterior deltoid has a high correlation with GIRD in patients with pathological stiff shoulder (Hung et al., 2010) and GIRD is correlated with a greater number of shoulder injuries within throwing athletes (Myers et al., 2006). In a study by Tyler et al. (Tyler et al., 2000), participants (non-throwers) with subacromial impingement in their dominant arm demonstrated significant internal rotation deficits (mean of -22.29°) as compared contralaterally, as well as greater posterior capsule tightness than the control group. It has also been suggested that anterior and superior humeral head translation on the glenoid fossa increases as a result of posterior capsular tightness. One cadaveric study operatively tightened the posterior capsule and demonstrated a significant increase in anterior translation (mean of 7.27mm) and slight increase in superior translation (mean of 2.13mm) of the humeral head on the glenoid fossa during flexion (Harryman et al., 1990). GIRD is often present in patients involved in regular overhead activity and subsequently affects scapular kinematics by increasing anterior scapular tilt during glenohumeral flexion and abduction, thereby reducing subacromial space distance (Borich et al., 2006; Hébert et al., 2002). Most importantly, GIRD also contributes to a reduction in the acromiohumeral distance (AHD), or subacromial space distance, in overhead athletes (Maenhout et al., 2012) ultimately predisposing the supraspinatus, long head of the biceps tendon, and subacromial bursa to pathologic compression and injury within the subacromial space. All of these factors considered, it is likely that posterior shoulder muscle stiffness and subsequent internal rotation deficits may contribute to a narrowing of the subacromial space distance.

Altered Muscle Recruitment

Normal glenohumeral abduction involves a complex synchronization of the forces elicited by the supraspinatus, infraspinatus, and deltoid as they work in opposition to one another during the first phase of abduction. As the deltoid creates a superiorly directed vector of force on the humerus, the supraspinatus and infraspinatus apply a medially directed line of pull on the humerus in order to center it on the glenoid and prevent excessive superior humeral migration. This force couple enables partial stabilization of the glenohumeral joint during the beginnings of overhead activities. However, alteration of this force couple through the degeneration, inhibition, or fatigue of the rotator cuff muscles results in a domination of the deltoid during abduction consequently generating a relative increase in the superior translation of the humeral head (Deutsch et al., 1996). Theoretically, facilitation or stiffness of the posterior deltoid, infraspinatus, and teres minor may also alter the functions of this force couple, creating pathological movement patterns and abnormal humeral head translations. As a result, this causes a functional narrowing of the subacromial space contributing to the development of SAIS.

Intervention Programs

SAIS in overhead athletes establishes a need to address predisposing factors such as GIRD, muscle imbalances, and abnormal scapular and glenohumeral kinematics. Fortunately, these are all modifiable physical characteristics, ultimately making it possible to formulate intervention programs to decrease the risk of developing SAIS. In order to reduce the amount of GIRD in athletic shoulders, research has studied the effects of stretching the posterior shoulder on increasing internal rotation. Both the cross-body and sleeper stretches increase

internal rotation ROM (McClure et al., 2007); additionally, the sleeper stretch also increases glenohumeral internal rotation, acromiohumeral distance (AHD), or subacromial space distance, in overhead athletes at 0°, 45°, and 60° of shoulder abduction (Maenhout et al., 2012). Other stretching interventions have examined the effect of stretching the pectoralis minor in order to correct the forward head and rounded shoulders posture observed in shoulders with adaptive pectoralis minor shortening (Thigpen et al., 2010). A self stretch procedure, where the patient places the affected arm in a position of 90° of abduction and 90° of elbow flexion on a planar surface and rotates the trunk away from the targeted side thereby increasing the amount of horizontal abduction, has been demonstrated as the most effective stretch for lengthening the pectoralis minor (Borstad & Ludewig, 2006). Evidence indicates that increasing the length of the pectoralis minor will assist in correcting the abnormal scapular kinematics, such as decreased posterior tipping and external rotation, that contribute to SAIS (Borstad & Ludewig, 2005; Ludewig & Cook, 2000).

Scapular stabilization exercises are also necessary to correct deviations in scapular posture that contribute to reductions in subacromial space distance and development of SAIS. Başkurt et al. (Başkurt, Başkurt, Gelecek, & Ozkan, 2011) determined the effectiveness of scapular stabilization exercises on pain, ROM, joint position sense, muscle strength, and quality of life in patients' with SAIS and found that each of these factors improved as a result of the 6 week intervention program. Wilk et al. (Wilk, Meister, & Andrews, 2002) outlined the following 5 step program for nonoperative treatment of SAIS: 1) Rest for 7-10 days from repetitive overhead athletic activity. 2) Restore normal glenohumeral and scapular kinematics by stretching the posterior shoulder. 3) Increase stability of glenohumeral joint as well as scapular strength and stability through pectoralis minor stretching and lower trapezius

strengthening. 4) Emphasize scapular retraction, and 5) gradually return to throwing. These are common therapeutic strategies used in athletic training rehabilitation programs for the athlete with SAIS; however, other recent research attempted to validate these common rehabilitation strategies and found little success with the interventions. Hibberd et al. (Hibberd, Oyama, Spang, Prentice, & Myers, 2012) analyzed the effects of a 6-week preventative intervention on scapular and shoulder girdle strengthening and scapular kinematics in competitive collegiate swimmers and found the intervention program was unsuccessful in correcting and/or preventing a rounded shoulder posture. Not many other studies have been conducted on the efficacy of certain rehabilitation exercises in the treatment of subacromial impingement and there is a lack of current evidence for anecdotal treatment strategies. Further research is necessary to ascertain which rehabilitation strategies are effective for both treatment and prevention of SAIS.

Instrumentation

Myotonometer

A myotonometer (Neurogenic Technologies Inc., Missoula, MT) will be used to collect measurements of active and passive muscle stiffness. The myotonometer is a patented and computerized meter-type device that effectively and efficiently measures tissue compliance and stiffness. The myotonometer measures the amount of resistance encountered by the probe when it is applied to the muscle and underlying tissue and subsequently quantifies the amount of tissue displacement which is then used to calculate stiffness ($k = \Delta force/\Delta length$) (Blackburn et al., 2011; Hung et al., 2010). Measurements of

muscle stiffness using a myotonometer have been proven valid and reliable (Leonard, Stephens, & Stroppel, 2001; Rydahl & Brouwer, 2004).

Diagnostic Ultrasound

A diagnostic ultrasound (US) (Model: Sonosite, Sonosite, Inc., Bothella, WA) unit will be used to collect measurements of the subacromial space distance via measurements of the AHD. The AHD is defined as the shortest distance between the humeral head and most inferior and lateral portion of the acromion process (Desmeules et al., 2004). Coronal axis views of the subacromial space with the transducer positioned according to the methods described by Desmeules et al. (Desmeules et al., 2004) and Azzoni et al. (Azzoni, Cabitza, & Parrini, 2004) will allow for visualization and accurate measure of the AHD. Previous studies have measured subacromial space distance and AHD with the arm positioned at 0°, 45°, and 60° of abduction, but have been unable to collect measurements in greater degrees of humeral abduction because of the limitations of the US unit created by beam reflection on bone interfering with visual clarity and inhibiting accurate measurements. However, a recent study by Timmons et al. (Timmons et al., 2013), measured AHD at 90° of abduction in positions of clinical full can (neutral humeral rotation) and empty can tests (humeral internal rotation). These methods for US measurement of the AHD and for quantifying the subacromial space distance have been found both valid and reliable (Azzoni et al., 2004; Desmeules et al., 2004; Maenhout et al., 2012).

Vernier Caliper

A vernier caliper will be used to measure PML (Westward Tools, Edmonton, AB, Canada). The bony landmarks used to locate the origin and insertion of the pectoralis minor

are the sternal aspect of the fourth rib and the coracoid process respectively. The vernier caliper will then be used to measure the distance between these and calculate the length of the pectoralis minor. These procedures are outlined by the validation and reliability study of PML measurement conducted by Borstad et al (Borstad, 2008) in which they used an electromagnetic motion capture system, a vernier caliper, and a cloth tape measure to measure the pectoralis minor and established relatively high intraclass correlation coefficients (ICC) between the electromagnetic motion capture system and caliper as well as between the electromagnetic motion capture system and tape measure. Therefore, the vernier caliper has been found to be a clinically valid assessment tool for the measurement of PML (Borstad, 2008).

Summary

Subacromial impingement syndrome is a common pathologic condition of the shoulder, particularly within the overhead athletic population (Diederichsen et al., 2009; McClure et al., 2004). Studies have identified modifiable physical characteristics of the superficial shoulder musculature that contribute to subacromial impingement. These contributors include posterior shoulder tightness and GIRD (Harryman et al., 1990; Hung et al., 2010; Myers et al., 2006; Tyler et al., 2000), altered glenohumeral and scapular kinematics such as greater superior humeral head translation (Deutsch et al., 1996; Ludewig & Cook, 2002), anterior tilting and upward rotation of the scapula (Burkhart et al., 2003; Ludewig & Cook, 2000), shortened PML (Borstad & Ludewig, 2005), and muscle imbalances (Page, 2011). However, very little research has identified direct effects of these modifiable characteristics on subacromial space distance. Muscle stiffness has primarily been assessed relative to pathologic stiff shoulder and glenohumeral instability (Huxel et al., 2008;

Myers & Lephart, 2000; Olds et al., 2011); however, evidence points towards the influence of muscle stiffness on subacromial impingement particularly through its affect on subacromial space distance (Laudner & Williams, 2013; Maenhout et al., 2012). As such, it is important to consider the effects of greater stiffness of the infraspinatus, teres minor, posterior deltoid, upper and lower trapezius, and latissimus dorsi on the functional narrowing of the subacromial space and the potential for predisposition to SAIS as a result. It is also apparent that intervention programs have little basis and the literature is lacking in rehabilitation protocols for SAIS. Therefore, the purpose of this study was to evaluate side-to-side differences in subacromial space distance, muscle stiffness, and PML, as well as determine the ability of these physical characteristics to predict subacromial space distance. Through a better understanding of these possible risk factors, clinicians could develop better intervention and prevention programs that could ultimately reduce the likelihood of instigating the subacromial impingement cascade of injury.

CHAPTER III

METHODOLOGY

Population and Recruitment

Fifty male and female participants, all of whom were overhead athletes at the division I level, were recruited to participate (Table 2). Individuals were recruited via flyers, word of mouth communication, and presentations by the primary investigator. Potential participants met with the primary investigator, received explanation regarding the study, and, once enrolled, provided Institutional Review Board (IRB) approved informed consent.

Subject Inclusion Criterion

Participants were included in this study if they met the following criteria:

- Varsity overhead athlete between the ages of 18-25 years
- Currently participating in one of the following varsity sports: baseball, softball, tennis, swimming, volleyball.
- No history of shoulder surgery, no current shoulder pain, and were not receiving rehabilitation for shoulder injury/pain.

Subject Exclusion Criterion

Participants were excluded from this study if they met the following criteria:

- History of shoulder surgery

TABLE 2: Participant Demographics

Participant Demographics	
Number of Participants (n)	50
Males/Females	19/31
Age (yrs)	19.4±1.2
Height (cm)	176.4±8.0
Weight (kg)	75.6±9.8
Arm Dominance	
Right/Left	44/6
Subjects per sport	
Baseball	10
Softball	10
Volleyball	10
Swimming	10
Tennis	10
Years of playing experience	11.8±2.7

Instrumentation

Myotonometer

A myotonometer (Neurogenic Technologies Inc., Missoula, MT) was used to collect measurements of active muscle stiffness. The myotonometer is a patented and computerized meter-type device that effectively and efficiently measures tissue compliance and stiffness. The myotonometer measures the amount of resistance encountered by the probe when it is applied to the muscle and underlying tissue and subsequently quantifies the targeted tissue's stiffness (Hung et al., 2010). Measurements of muscle stiffness using a myotonometer have been shown to be valid and reliable in lower extremity muscles (Leonard et al., 2001; Rydahl & Brouwer, 2004). We established the reliability and validity of the myotonometer measurements of muscle stiffness of the muscles we proposed to assess in the current study in 10 varsity collegiate athletes (Table 3).

TABLE 3: Intraclass Correlations of Myotonometric Measurements of Muscle Stiffness

Muscle Stiffness	Intrasession ICC	Intrasession SEM (mm)	Mean Detectable Difference
INFRA 1.50	.984	0.65	1.79
INFRA 1.75	.981	0.67	1.85
INFRA 2.0	.978	0.68	1.89
TM 1.50	.955	1.02	2.82
TM 1.75	.957	1.03	2.86
TM 2.0	.959	1.03	2.86
PD 1.50	.891	0.53	1.46
PD 1.75	.884	0.52	1.44
PD 2.0	.882	0.51	1.40
UT 1.50	.757	0.39	1.08
UT 1.75	.789	0.41	1.13
UT 2.0	.808	0.41	1.15
LT 1.50	.829	0.57	1.57
LT 1.75	.845	0.58	1.60
LT 2.0	.656	0.67	1.85
LD 1.50	.986	0.98	2.72
LD 1.75	.972	0.99	2.76
LD 2.0	.975	1.01	2.79

Diagnostic Ultrasound

A diagnostic US unit (Model: Sonosite, Sonosite, Inc., Bothella, WA) was used to collect measurements of the subacromial space distance via measurements of the AHD. The AHD is defined as the shortest distance between the humeral head and most inferior and lateral portion of the acromion process (Desmeules et al., 2004). Coronal axis views of the subacromial space with the probe positioned according to previously described methods (Azzoni et al., 2004; Desmeules et al., 2004) enabled us to visualize and accurately measure the AHD. Previous studies have measured subacromial space distance and AHD with the arm positioned at 0°, 45°, and 60° of abduction, but have been unable to collect measurements in greater degrees of humeral abduction because of the limitations of the ultrasound unit created by beam reflection on bone interfering with visual clarity and inhibiting accurate measurements. However, a recent study, measured AHD at 90° of abduction in positions of

clinical full can (neutral humeral rotation) and empty can tests (humeral internal rotation). Preliminary data from 9 subjects enabled calculations of intra-rater and test-retest reliability (ICC = 0.90, SEM = 0.07 mm) (Timmons et al., 2013). These methods for US measurement of the AHD and for quantifying the subacromial space distance have been found both valid and reliable (Azzoni et al., 2004; Desmeules et al., 2004; Maenhout et al., 2012).

Vernier Caliper

A vernier caliper was used to measure PML (Westward Tools, Edmonton, AB, Canada). The bony landmarks used to locate the origin and insertion of the pectoralis minor are the sternal aspect of the fourth rib and the coracoid process. The vernier caliper was used to measure the distance between these points and to represent PML. These procedures are outlined by the validation and reliability study of PML measurement conducted by Borstad et al (Borstad, 2008) in which they used an electromagnetic motion capture system, a vernier caliper, and a cloth tape measure to measure the pectoralis minor and established relatively high intraclass correlation coefficients each measurement. Therefore, the vernier caliper has been found to be a clinically valid assessment tool for the measurement of PML (Borstad, 2008).

Procedures

A cross-sectional research design was used in this study. Study participants reported to the Neuromuscular Research Laboratory (NMRL) for a single session. Participants were introduced to the experiment and then read and signed a consent form approved by the University of North Carolina Institutional Review Board. Prior to testing, each participant

completed a brief survey detailing demographics including sex, age, arm dominance, current or previous overhead sport activity experience, and his/her history of shoulder pain and/or injury. Each participant then had height (cm) and mass (kg) measurements taken by one of the researchers. Each participant then underwent the testing procedures that included measurements of the subacromial space distance, muscle stiffness, and PML. Testing order and conditions were randomized and counterbalanced. Details of each procedure are discussed below.

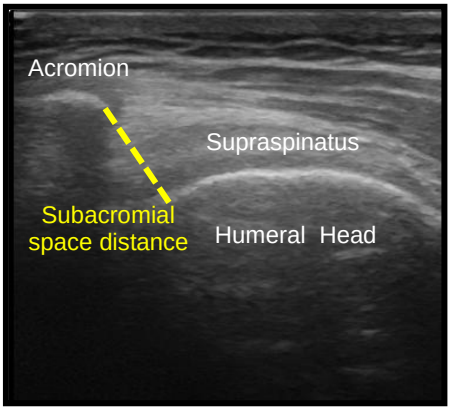
Subacromial Space Distance

The participant was instructed to take a seated position on a stool with the arms in a relaxed position hanging by his/her sides. Subacromial space distance was measured using US techniques as described by Maenhout et al. (Maenhout et al., 2012). Three US images were taken at 45° of abduction. For imaging at 45° of abduction, one loop of a belt was secured to the base of the stool upon which the participant sat, while the other end was looped around the participant's distal forearm. Arm position was verified by a digital inclinometer. The participant was instructed to apply tension to the belt in order to maintain arm position as well as to elicit activation of the shoulder musculature. The participant was also asked to hold a dumbbell in order to elicit activation of the muscles of interest. The weight of the dumbbell was determined relative to body mass, 1.4kg (3lbs) for those weighing less than 68.1kg (150lbs) and 2.3kg (5lbs) for those weighing more than 68.1kg (McClure, Tate, Kareha, Irwin, & Zlupko, 2009). The US transducer was placed on the superolateral aspect of the shoulder along the longitudinal axis of the humerus (Figure 2). Subacromial space distance was scanned from the coronal view and measured as the shortest

distance from the infero-lateral edge of the acromion to the humeral head (Leong, Tsui, Ying, Leung, & Fu, 2012; Maenhout et al., 2012) (Figure 3). The participant was instructed to rest between image trials with the arm at 0° abduction placing the hand and weight on his/her thigh in order to prevent muscle fatigue during the testing session. Subacromial space distance values were calculated as the average of three trials bilaterally. These values were normalized to each participant's height (subacromial space distance/height). We estabilished intrasession reliability (ICC: 0.840), standard error of the measurement (SEM: 0.87mm), and mean detectable difference (MDD: 2.41) through pilot testing.

FIGURE 2: Transducer Locations

FIGURE 3: Subacromial Space Distance



Muscle Stiffness

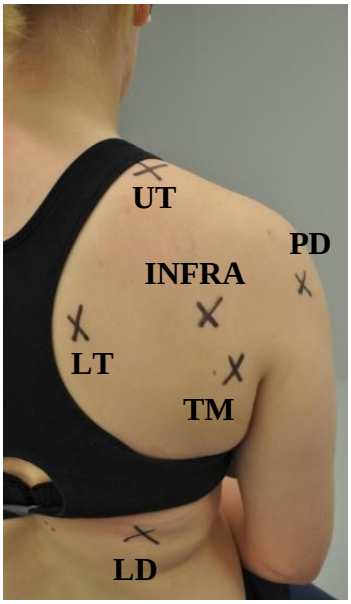
Muscle stiffness of the posterior deltoid, infraspinatus, teres minor, upper and lower trapezius, and latissimus dorsi was recorded using a handheld myotonometer. Testing order of the muscles was randomized for each participant. The participant was asked to remain sitting with his/her feet resting flat on the floor and with the arm raised into 45° of shoulder abduction. The same procedures previously outlined for subacromial space distance testing were used in order to maintain the arm position at 45° of abduction. The participant was also asked to hold a dumbbell, with the weight determined relative to body mass (1.4kg (3lbs) for those weighing less than 68.1kg (150lbs) and 2.3kg (5lbs) for those weighing more than 68.1kg), in order to elicit activation of the muscles of interest (McClure et al., 2009). The participant was instructed to rest the arm in 0° of shoulder abduction with the hand and weight resting on the thigh between trials at each muscle in order to prevent excessive muscle fatigue during the testing session. The following anatomical locations were used for the placement of the myotonometer probe (Figure 4):

- *Posterior deltoid* - 2 fingerbreadths inferior to the posterior margin of the acromion (Hung et al., 2010).
- *Infraspinatus* - 2 fingerbreadths below the medial portion of the spine of the scapula (Hung et al., 2010).
- *Teres minor* - one-third of the distance between the acromion and inferior angle of the scapula along the lateral border (Hung et al., 2010).
- *Upper trapezius* - midway between the spinous process of the seventh cervical vertebra and the posterior margin of the acromion process (based on electrode placement in electromyography) (Cools et al., 2007).

- *Lower trapezius* - obliquely upward and laterally along a linear pathway between the intersection of the spine of the scapula with the vertebral border of the scapula and seventh thoracic spinous process (based on electrode placement in electromyography) (Cools et al., 2007).
- *Latissimus dorsi* - 5cm inferior to the inferior portion of the scapular border (Laudner & Williams, 2013).

The mean of 5 trials at 8 different increments (0.25 – 2.00 kg) of 0.25kg of force pressure was calculated during probe application at each muscle to determine tissue displacement, which was used to calculate muscle stiffness. Procedures for tissue displacement were completed bilaterally.

FIGURE 4: Myotonometer Probe Locations



** UT=Upper Trapezius, LT=Lower Trapezius, INFRA=Infraspinatus, PD=Posterior Deltoid, TM=Teres Minor, and LD=Latissimus Dorsi

Pectoralis Minor Length

PML was measured at 45° shoulder abduction using a vernier caliper according to procedures described by Borstad (Borstad, 2008). For measurement at 45° abduction, the participant was asked to perform the previously described procedures used for elevating to and maintaining the arm at 45° abduction while the primary researcher palpated and located the origin of the 4th rib (muscle origin) and the coracoid process (muscle insertion). We estabilished intrasession reliability (ICC: 0.979), standard error of measurement (SEM: 0.53cm), and mean detectable difference (MDD: 1.46) were calculated in pilot testing. PML values were normalized to each subject's height through the division of PML values by height and calculated as an average of three trials using the distance values created between the arms of the vernier caliper (Figure 5).

FIGURE 5: Pectoralis Minor Length Measurement

Data Reduction

Still ultrasound images of the subacromial space were exported and subacromial space distance was measured as the shortest linear distance between the superolateral tip of

the acromion and the humeral head (Leong et al., 2012) using Image J software (National Institute of Health, Bethesda, MD). Subacromial space distance values were calculated as the average of three trials at 45° of shoulder abduction bilaterally.

The myotonometer generates force-displacement curves quantifying muscle stiffness (Hung et al., 2010). Muscle stiffness is measured as the change in force per change in length ($k = \Delta force/\Delta length$) (Blackburn et al., 2011). Three trial means were calculated for each muscle and each trial mean consisted of 5 applications of probe pressure at three different force increments (1.50, 1.75, 2.00 kg). Previous literature suggests that 1.50-2.00 kg of force are the primary increments of pressure that result in significant findings of muscle stiffness (Hung et al., 2010).

PML values were calculated as an average of three trials using the distance values created between the arms of the vernier caliper.

Data Analysis

Statistical analyses were performed using SPSS version 21.0 for Windows (SPSS Inc, Chicago, IL). A multiple linear regression analysis was performed to determine the ability of the predictor variables (PML and muscle stiffness of the posterior deltoid, infraspinatus, teres minor, upper trapezius, lower trapezius, and latissimus dorsi) to predict subacromial space distance values. Muscle stiffness values at 1.50kg of force were used for the regression model. All predictor variables were forced into the regression using the enter method. Paired samples t-tests were performed to compare muscle stiffness, AHD, and PML between dominant and non-dominant arms. An *a priori* alpha level was set at 0.05.

Summary of Research Questions

Question	Description	Data Source	Comparison	Method
1	What is the relative contribution of muscle stiffness of the superficial shoulder musculature and PML to subacromial space distance?	Average of three trials of 3 force increments of probe pressure (muscle stiffness), vernier caliper (PML), and diagnostic ultrasound (subacromial space distance)	Subacromial space distance in shoulders with greater muscle stiffness and a shorter PML to subacromial space distance in shoulders with less muscle stiffness and a longer PML	Linear multiple regression
2	Is there a difference in muscle stiffness values between the dominant and non-dominant arms?	Myotonometric measurements of muscle stiffness in dominant and non-dominant arms	Lesser muscle stiffness values in non-dominant arm and greater muscle stiffness values in dominant arm	Paired Samples t-test
3	Is there a difference in subacromial space distance between dominant and non-dominant arms?	Ultrasonographic measurements of subacromial space distance in dominant and non-dominant arms	Greater subacromial space distance in non-dominant arm and decreased subacromial space distance in dominant arm	Paired Samples t-test
4	Is there a difference in PML between dominant and non-dominant arms?	Vernier caliper measurements of PML in dominant and non-dominant arms	Greater PML in non-dominant arm and shorter PML in dominant arm	Paired Samples t-test

CHAPTER IV[1]

RELATIONSHIP BETWEEN MUSCLE STIFFNESS OF THE SUPERFICIAL SHOULDER MUSCULATURE AND ACROMIOHUMERAL DISTANCE

Background: Subacromial impingement syndrome (SAIS) of the dominant arm is a common pathology in overhead athletes that may be caused by a narrowing of the subacromial space due to modifiable physical characteristics such as decreased pectoralis minor length and increased muscle stiffness of superficial shoulder musculature. The purpose of this study was to evaluate side-to-side differences in acromiohumeral distance, muscle stiffness, and pectoralis minor length, and to determine the ability of these physical characteristics to predict acromiohumeral distance, in overhead athletes.

Hypothesis: The dominant arm will demonstrate decreased acromiohumeral distance, greater muscle stiffness, and shorter pectoralis minor length. The modifiable physical characteristics will significantly predict acromiohumeral distance.

Study Design: Cross-Sectional Study

Level of Evidence: 4

[1] Manuscript formatted for the Journal of Sports Health: A Multidisciplinary Approach

Methods: Fifty collegiate overhead athletes completed one testing session of bilateral measurements of the acromiohumeral distance, muscle stiffness, and pectoralis minor length.

Results: The dominant arm exhibited a shorter pectoralis minor ($p = 0.02$) and greater stiffness of the teres minor (1.50kg: $p < 0.005$; 1.75kg: $p < 0.005$; 2.0kg: $p < 0.005$), posterior deltoid (1.50kg: $p < 0.005$; 1.75kg: $p = 0.02$; 2.0kg: $p < 0.005$), and lower trapezius (1.50kg: $p = 0.04$; 1.75kg: $p = 0.03$; 2.0kg: $p = 0.03$) compared to the non-dominant arm. There were no significant differences in acromiohumeral distance ($p = 0.40$) at 45° abduction between limbs. Neither muscle stiffness nor pectoralis minor length predicted acromiohumeral distance in either limb of healthy overhead athletes.

Conclusions: These findings indicate differences in muscle stiffness and pectoralis minor length between limbs. Further research is needed to determine the relationship between muscle stiffness of the superficial shoulder musculature and acromiohumeral distance in overhead athletes with SAIS.

Clinical Relevance: Side-to-side differences in muscle stiffness and pectoralis minor length in collegiate overhead athletes may indicate alterations in glenohumeral and scapular kinematics which may predispose the athlete to develop SAIS. Clinicians could use this as a screening tool to identify individuals with side-to-side differences and then implement interventions to address these asymmetries.

Key Words: Muscle Stiffness, Overhead Athletes, Acromiohumeral Distance

Word Count: 326

INTRODUCTION

Shoulder pain is common among overhead athletes, particularly among competitive baseball, volleyball, tennis, and swimming athletes (Borich et al., 2006). The prevalence of shoulder pain among competitive overhead athletes is between 10 and 30% (Diederichsen et al., 2009). Subacromial impingement syndrome (SAIS) accounts for 44-65% of all shoulder pain related physician visits (de Witte et al., 2011; McClure et al., 2004; Michener et al., 2003; Umer et al., 2012) and is described as three progressive stages of rotator cuff tendinopathy (Neer, 1983). Collegiate overhead athletes primarily experience the early stages of SAIS (Cowderoy et al., 2009) such as inflammation of the subacromial bursa and rotator cuff muscles with minor tendon degeneration (Neer, 1983).

The overhead athlete more commonly experiences the effects of functional impingement on the dominant arm due to the repetitive nature of his/her sport (Cowderoy et al., 2009; Page, 2011). Functional, or secondary, impingement is the compression of the long head of the biceps tendon, the subacromial bursa, and/or the supraspinatus tendon between the humeral head and the acromion process as a result of superior migration of the humeral head during arm elevation (Cools et al., 2008; Desmeules et al., 2004; Diederichsen et al., 2009; Ludewig & Cook, 2000; Neer, 1983; Page, 2011). Secondary impingement manifests as a result of altered glenohumeral and scapular kinematics (Burkhart et al., 2003; Cools et al., 2008; Diederichsen et al., 2009; Ludewig & Cook, 2000; Maenhout et al., 2012; McClure et al., 2004; Page, 2011) and recent literature suggests that posterior capsule and muscle tightness (Myers et al., 2006; Tyler et al., 2000), are linked with SAIS (Maenhout et al., 2012). The overall effect of each of these etiologies is a narrowing of the acromiohumeral distance (AHD), ultimately increasing the likelihood of pathological compression of the long

head of the biceps tendon, the subacromial bursa, and the supraspinatus tendon located within this space (Burkhart et al., 2003; Maenhout et al., 2012).

Narrowing of the AHD has been partially attributed to abnormal glenohumeral and scapular kinematics, such as increased superior translation of the humeral head (Deutsch et al., 1996), decreased internal rotation (Borich et al., 2006; Maenhout et al., 2012), increased anterior scapular tilting (Borich et al., 2006; Hébert et al., 2002; Ludewig & Cook, 2000), increased scapular upward rotation (Karduna et al., 2005) and increased protraction of the scapula (Solem-Bertoft et al., 1993). Abnormal scapular kinematics that are related to subacromial impingement have also been linked with altered muscle activity (Graichen et al., 1998; Graichen, Stammberger, et al., 1999) and a shortened pectoralis minor length (PML) (Borstad & Ludewig, 2005). In addition to these known contributors, it is likely that stiffness of the superficial shoulder musculature may also play a role in reducing AHD (Hung et al., 2010).

Muscle stiffness quantifies a muscle's resistance to lengthening and is defined as the ratio of change in force per change in length (Blackburn et al., 2011; Hung et al., 2010; Huxel et al., 2008; Myers & Lephart, 2000; Oatis, 1993; Olds et al., 2011). Much of the current research in regards to stiffness and the shoulder concerns either the pathological "frozen," or stiff shoulder (Hung et al., 2010) or the benefits of muscle stiffness in relation to pathological instability of the glenohumeral joint (Huxel et al., 2008; Olds et al., 2011). Research clearly identifies the cascade of SAIS as a progression from posterior shoulder tightness to internal rotation deficits (Hung et al., 2010) to altered glenohumeral and scapular kinematics (i.e. increased scapular upward rotation (Karduna et al., 2005), anterior tilting (Borich et al., 2006), and internal rotation (Ludewig & Cook, 2000)) and finally to

subsequent reductions in subacromial space (Graichen, Bonel, et al., 1999; Maenhout et al., 2012). Because posterior shoulder tightness and alterations in glenohumeral and scapular kinematics are related to reductions in AHD, stiffness in the muscles contributing to abnormal glenohumeral and scapular kinematics may potentially be correlated to decreased AHD and a greater risk of SAIS. While there is a theoretical link between muscle stiffness and AHD, to date there are no previous studies that identify this relationship in either healthy or non-healthy shoulders; therefore, it is important to first determine if there is a relationship within the healthy overhead athlete's shoulder. The purpose of this study was to evaluate side-to-side differences in AHD, muscle stiffness, and PML, and to determine the ability of these physical characteristics to predict AHD.

Due to the nature of the unilateral demands of an overhead athlete's sport, with the exclusion of swimming, the dominant arm trains far more than the non-dominant arm, creating an increased load on the muscles surrounding the shoulder that is unique to that shoulder. It is likely then, that the dominant arm would develop greater levels of muscle stiffness of the superficial shoulder musculature and potentially exhibit a decrease in AHD as compared to the non-dominant arm. Understanding the contribution of each of these muscles to AHD may provide clinicians with valuable information regarding potential risk factors for decreasing AHD and developing SAIS. Through a better understanding of these possible risk factors, clinicians could develop better intervention and prevention programs that could ultimately reduce the likelihood of instigating the subacromial impingement cascade of injury.

MATERIALS AND METHODS

Participants

Fifty male and female participants, all of whom were collegiate overhead athletes at the division I level, were recruited to participate (Table 1). All participants were currently participating in one of the following varsity sports: baseball, softball, volleyball, swimming, or tennis, and had no history of shoulder surgery, no current shoulder pain, and were not currently receiving rehabilitation for shoulder injury/pain. All participants read and signed a consent form approved by the university's Institutional Review Board.

TABLE 1: Participant Demographics

Participant Demographics	
Number of Participants (n)	50
Males/Females	19/31
Age (yrs)	19.4±1.2
Height (cm)	176.4±8.0
Weight (kg)	75.6±9.8
Arm Dominance	
Right/Left	44/6
Subjects per sport	
Baseball	10
Softball	10
Volleyball	10
Swimming	10
Tennis	10
Years of playing experience	11.8±2.7

Procedures

A cross-sectional research design was used in this study. Participants reported to a university biomechanics laboratory for a single session. Prior to testing, each participant completed a brief survey detailing demographics including sex, age, arm dominance, current or previous overhead sport activity experience, and his/her history of shoulder pain and/or

injury. Each participant had height (cm) and mass (kg) measurements taken by the research team, and underwent the testing procedures that included measurements of the AHD, muscle stiffness, and PML. Testing procedure order was counterbalanced between participants.

Acromiohumeral Distance

AHD was measured using diagnostic ultrasound (Model: Sonosite, Sonosite, Inc., Bothella, WA) (US) as described by Maenhout et al. (Maenhout et al., 2012). AHD was defined as the shortest distance between the humeral head and most inferior and lateral portion of the acromion process (Figure 1) (Desmeules et al., 2004). Coronal axis views of the AHD were imaged by placing the transducer at the superolateral surface of the shoulder along the longitudinal axis of the humerus (Azzoni et al., 2004; Desmeules et al., 2004), enabling us to visualize and measure the AHD. Three AHD images were taken with the arm raised to 45° of abduction (Figure 2). Arm position was verified by a digital inclinometer. At 45°, the participant was instructed to apply tension to a belt in order to maintain arm position as well as to elicit activation of the shoulder musculature. The participant was also asked to hold a dumbbell in order to further activate the muscles of interest. The weight of the dumbbell was determined relative to body mass, 1.4kg (3lbs) for those weighing less than 68.1kg (150lbs) and 2.3kg (5lbs) for those weighing more than 68.1kg (McClure et al., 2009). US imaging of the AHD was completed bilaterally. Still US images of the subacromial space were imported into Image J software (National Institute of Health, Bethesda, MD) and AHD was measured as the shortest linear distance between the inferolateral tip of the acromion and the humeral head (Desmeules et al., 2004). AHD values were calculated as the average of three trials bilaterally. These values were normalized to

each subject's height (stiffness value/height). We estabilished intrasession reliability (ICC: 0.840), standard error of the measurement (SEM: 0.87mm), and mean detectable difference (MDD: 2.41) through pilot testing.

FIGURE 1: Ultrasonographic Measurements of Acromiohumeral Distance

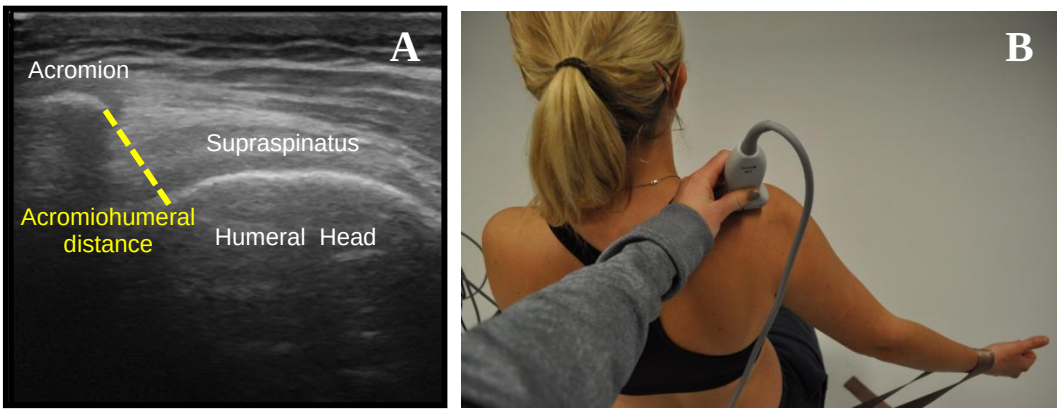
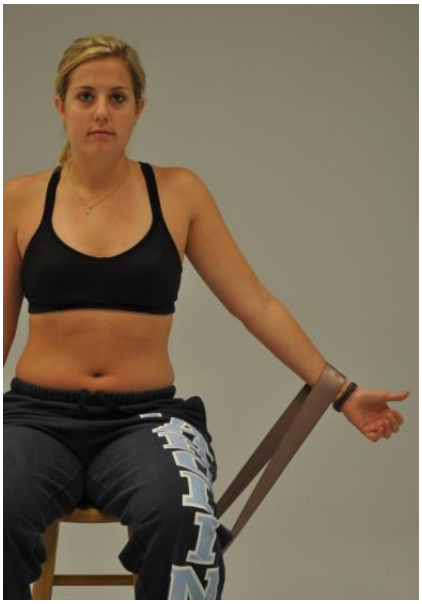


FIGURE 2: 45° Shoulder Abduction

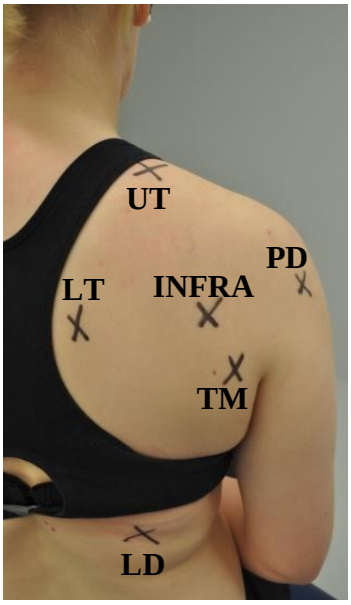


Muscle Stiffness

A myotonometer (Neurogenic Technologies Inc., Missoula, MT) was used to collect measurements of active muscle stiffness. We established the reliability and validity of the myotonometer measurements of muscle stiffness of the muscles we assessed in the current study in 10 varsity collegiate athletes (Table 2). Active muscle stiffness of the posterior deltoid, infraspinatus, teres minor, upper and lower trapezius, and latissimus dorsi was recorded with the participant's arm in 45° abduction and holding a dumbbell (as previously described) (Figure 2). The participant was instructed to rest the arm in 0° of shoulder abduction with the hand and weight resting on the thigh between trials at each muscle in order to prevent fatigue during the testing session. Anatomical locations (Figure 3) used for the placement of the myotonometer probe are described in Table 3. The mean of 5 trials at 8 different increments (0.25 – 2.00 kg) of 0.25kg of force pressure was calculated during probe application at each muscle to determine tissue displacement, which was used to calculate muscle stiffness. Procedures for tissue displacement were completed bilaterally and testing order was randomized.

Muscle stiffness was calculated for three different force increments (1.50, 1.75, 2.00 kg), as these force increments, both in our study and in a previous study (Hung et al., 2010), have been previously suggested as the most reliable in determining true tissue displacement. Muscle stiffness was calculated as the change in force per change in length, or tissue displacement, ($k = \Delta force/\Delta length$) (Blackburn et al., 2011).

FIGURE 3: Myotonometer Probe Locations



** UT=Upper Trapezius, LT=Lower Trapezius, INFRA=Infraspinatus, PD=Posterior Deltoid, TM=Teres Minor, and LD=Latissimus Dorsi

TABLE 2: Intraclass Correlations of Myotonometric Measurements of Muscle Stiffness

Muscle Stiffness	Intrasession ICC	Intrasession SEM (mm)	Mean Detectable Difference
INFRA 1.50	.984	0.65	1.79
INFRA 1.75	.981	0.67	1.85
INFRA 2.0	.978	0.68	1.89
TM 1.50	.955	1.02	2.82
TM 1.75	.957	1.03	2.86
TM 2.0	.959	1.03	2.86
PD 1.50	.891	0.53	1.46
PD 1.75	.884	0.52	1.44
PD 2.0	.882	0.51	1.40
UT 1.50	.757	0.39	1.08
UT 1.75	.789	0.41	1.13
UT 2.0	.808	0.41	1.15
LT 1.50	.829	0.57	1.57
LT 1.75	.845	0.58	1.60
LT 2.0	.656	0.67	1.85
LD 1.50	.986	0.98	2.72
LD 1.75	.972	0.99	2.76
LD 2.0	.975	1.01	2.79

TABLE 3: Myotonometer Probe Placements

Muscle	Anatomical Location
Posterior Deltoid	2 fingerbreadths inferior to the posterior margin of the acromion.(Hung et al., 2010)
Infraspinatus	2 fingerbreadths below the medial portion of the spine of the scapula.(Hung et al., 2010)
Teres Minor	One-third of the distance between the acromion and inferior angle of the scapula along the lateral border.(Hung et al., 2010)
Upper Trapezius	Midway between the spinous process of the seventh cervical vertebra and the posterior margin of the acromion process (based on electrode placement in electromyography).(Cools et al., 2007)
Lower Trapezius	Obliquely upward and laterally along a linear pathway between the intersection of the spine of the scapula with the vertebral border of the scapula and seventh thoracic spinous process (based on electrode placement in electromyography).(Cools et al., 2007)
Latissimus Dorsi	5cm inferior to the inferior portion of the scapular border.(Laudner & Williams, 2013)

Pectoralis Minor Length

PML was measured at 45° shoulder abduction using a vernier caliper (Westward Tools, Edmonton, AB, Canada) according to procedures described by Borstad et al. (Borstad, 2008). For measurement at 45° abduction, the participant was asked to elevate the arm until meeting the resistance of the belt (Figure 2) while the primary researcher palpated and located the sternocostal joint of the 4th rib (muscle origin) and the coracoid process (muscle insertion). The caliper arms were placed at these landmarks to measure the distance between them during 3 separate trials for which an average was calculated. We established intrasession reliability (ICC: 0.979), standard error of measurement (SEM: 0.53cm), and mean detectable difference (MDD: 1.46) in pilot testing. PML values were normalized to

each subject's height (PML/height) and calculated as an average of three trials using the distance values created between the arms of the vernier caliper.

Statistical Analysis

Statistical analyses were performed using SPSS version 21.0 for Windows (SPSS Inc, Chicago, IL). Paired samples t-tests were performed to compare muscle stiffness, AHD, and PML between dominant and non-dominant arms. A multiple linear regression analysis was performed to determine the ability of the predictor variables (PML and muscle stiffness of the posterior deltoid, infraspinatus, teres minor, upper trapezius, lower trapezius, and latissimus dorsi) to predict subacromial space distance values. Muscle stiffness values at 1.50kg of force were used for the regression model. All predictor variables were forced into the regression using the enter method. An *a priori* alpha level was set at 0.05.

RESULTS

PML was significantly shorter (t_{49} = -2.332, p = 0.02) in the dominant arms compared to the non-dominant arms. Additionally, active muscle stiffness was significantly greater in the teres minor (1.50kg: t_{49} = 6.078, p < 0.005; 1.75kg: t_{49} = 5.963, p < 0.005; 2.0kg: t_{49} = 5.556, p < 0.005), posterior deltoid (1.50kg: t_{49} = 4.301, p < 0.005; 1.75kg: t_{49} = 2.510, p = 0.02; 2.0kg: t_{49} = 4.486, p = 0.00), and lower trapezius (1.50kg: t_{49} = 2.115, p = 0.04; 1.75kg: t_{49} = 2.207, p = 0.03; 2.0kg: t_{49} = 2.217, p = 0.03) in the dominant arm compared to the non-dominant arm. However, there were no significant differences in AHD (t_{49} = .849, p = 0.40) between arms. The descriptive statistics for all variables are presented in Table 4.

PML and muscle stiffness values did not significantly predict AHD of the dominant arm ($F(7,42) = 1.332$, p = 0.26, R^2 of .182) or the non-dominant arm ($F(7,42) = 0.804$, p = 0.59, R^2 of .118) (Table 5). Additional analysis also revealed no significance in simple correlations.

TABLE 4: Paired Samples t-Tests Results for PML, AHD, and Muscle Stiffness

Dependent Variable	Dominant Arm	Non-dominant Arm	p-value*
	Mean±SD	*Mean±SD*	
Pectoralis Minor Length (cm)			
45° ABD	13.96±1.60	14.36±1.77	<0.02*
AHD (mm)			
45° ABD	9.86±2.91	9.56±2.52	<0.40
Muscle Stiffness (Δforce/Δlength)			
Infraspinatus			
1.50kg	0.166±0.040	0.158±0.039	<0.06
1.75kg	0.188±0.043	0.179±0.042	<0.06
2.00kg	0.209±0.047	0.201±0.047	<0.08
Teres Minor			
1.50kg	0.112±0.024	0.097±0.019	<0.00*
1.75kg	0.126±0.026	0.110±0.021	<0.00*
2.00kg	0.140±0.028	0.124±0.023	<0.00*
Posterior Deltoid			
1.50kg	0.232±0.038	0.208±0.024	<0.00*
1.75kg	0.251±0.053	0.230±0.027	<0.02*
2.00kg	0.280±0.041	0.253±0.029	<0.00*
Upper Trapezius			
1.50kg	0.173±0.022	0.171±0.022	<0.68
1.75kg	0.192±0.023	0.190±0.024	<0.70
2.00kg	0.213±0.025	0.211±0.027	<0.55
Lower Trapezius			
1.50kg	0.153±0.024	0.144±0.029	<0.04*
1.75kg	0.171±0.027	0.160±0.032	<0.03*
2.00kg	0.189±0.029	0.175±0.038	<0.03*
Latissimus Dorsi			
1.50kg	0.127±0.039	0.132±0.039	<0.35
1.75kg	0.147±0.044	0.152±0.042	<0.25
2.00kg	0.164±0.048	0.172±0.046	<0.18

**significant p-value <0.05*

TABLE 5: Regression Analysis Between AHD, PML, and Muscle Stiffness

Variable	Dominant Arm	*P* value*	Non-dominant Arm	*P* value*
	Standardized Beta Coefficient		*Standardized Beta Coefficient*	
Pectoralis Minor Length	.123	.41	-.026	0.86
Infraspinatus	-.008	.96	.172	0.29
Teres Minor	.298	.09	.204	0.25
Posterior Deltoid	.196	.23	.073	0.68
Upper Trapezius	-.126	.40	.078	0.62
Lower Trapezius	-.079	.60	-.003	0.99
Latissimus Dorsi	-.076	.61	-.075	0.63

**significant p-value <0.05*

DISCUSSION

The purpose of this study was to evaluate side-to-side differences in AHD, superficial shoulder muscle stiffness and PML, and to determine the ability of these physical characteristics to predict AHD. Our findings indicate that healthy overhead athletes presented with greater posterior deltoid, teres minor, and lower trapezius muscle stiffness and shorter PML on the dominant limb compared to the non-dominant limb. There were no other statistically significant differences related to limb dominance. Muscle stiffness of superficial shoulder musculature and PML did not predict AHD.

Current research clearly identifies the cascade of SAIS as a progression from posterior shoulder tightness to internal rotation deficits (Hung et al., 2010) to altered glenohumeral and scapular kinematics (increased upward rotation (Karduna et al., 2005), anterior tilting (Borich et al., 2006), and internal rotation (Ludewig & Cook, 2000)) and finally to subsequent reductions in subacromial space (Graichen, Bonel, et al., 1999; Maenhout et al., 2012). Because our findings indicate stiffness and PML differences in the dominant arm and previous research indicates subsequent changes in glenohumeral and scapular kinematics, it is crucial to consider that these alterations in stiffness and PML on the

dominant limb create asymmetries which may predispose the overhead athlete to a greater risk of injury.

Our results demonstrated significant differences in muscle stiffness and PML between dominant and non-dominant arms in healthy overhead athletes. The dominant arm exhibited greater stiffness values in the posterior deltoid and teres minor, two of the three primary muscles of the posterior shoulder. Previous research has demonstrated that increased stiffness of the posterior shoulder (posterior deltoid, teres minor, infraspinatus) is correlated with a decrease in glenohumeral internal rotation range of motion (Hung et al., 2010), ultimately contributing to glenohumeral internal rotation deficits (GIRD) (Myers et al., 2006) and a loss of total arc of motion. Tyler et al. (Tyler et al., 2000) showed participants (non-throwers) with subacromial impingement in their dominant arm demonstrated significant internal rotation deficits (mean -22.29°) as compared contralaterally, as well as greater posterior capsule tightness than the control group. GIRD is often present in individuals involved in regular overhead activity and subsequently affects scapular kinematics by increasing anterior scapular tilt (Borich et al., 2006) and increasing scapular upward rotation (Karduna et al., 2005) during glenohumeral flexion and abduction, thereby reducing AHD (Borich et al., 2006; Hébert et al., 2002). Most importantly, GIRD also contributes to a reduction in AHD in overhead athletes (Maenhout et al., 2012) ultimately predisposing the supraspinatus, long head of the biceps tendon, and subacromial bursa to pathologic compression and injury within the subacromial space. Our results support these findings of side-to-side differences in posterior shoulder stiffness in overhead athletes, particularly within a healthy population, and may indicate a potential risk for the development of injury.

Our results also indicate greater stiffness of the lower trapezius in the dominant arm. This is important when considering the effects of the lower trapezius on scapular orientation. Stiffness of the lower trapezius may increase scapular upward rotation and depression (Terry & Chopp, 2000) and previous research has identified increased lower trapezius activation and scapular upward rotation in shoulders with impingement (Ludewig & Cook, 2000). This is particularly important when considering the sport demands of an overhead athlete. For example, the pitching motion requires the greater tuberosity of the humeral head to pass inferiorly to the acromion process during external rotation of the cocking phase (Dillman et al., 1993; Flatow et al., 1994; Meister, 2000; Neagle & Bennett, 1994) and with scapular dyskinesis, such as abnormal scapular upward rotation (Karduna et al., 2005; Ludewig & Cook, 2000), the humeral head is limited in its ability to clear the subacromial space.

Finally, the dominant arm demonstrated a significantly shorter PML than the non-dominant arm. The pectoralis minor originates on the ribs and inserts at the coracoid process of the scapula and functions to protract and internally rotate the scapula (Terry & Chopp, 2000). A shortened PML orients the scapula in a more protracted position (Borstad & Ludewig, 2005) and protraction and internal rotation of the scapula diminishes the subacromial space thereby increasing the amount of contact pressure on the structures within (Solem-Bertoft et al., 1993). It is possible that each of these findings (posterior deltoid, teres minor, lower trapezius stiffness and a shortened PML) within the dominant arm may offer a potential screening tool for clinicians. Since previous research indicates development of abnormal scapular kinematics as a result of the changes in shoulder muscle stiffness and PML, it is likely that clinicians could track asymmetries between dominant and non-dominant arms of overhead athletes in order to ascertain the risk of injury.

Side-to-side differences in muscle stiffness and PML in collegiate overhead athletes may indicate alterations in glenohumeral and scapular kinematics which may predispose the athlete to develop SAIS. Clinicians could use this as a screening tool to identify individuals with side-to-side differences and then implement interventions to address these asymmetries.

Future prospective studies should consider determining if differences in PML and superficial shoulder muscle stiffness between dominant and non-dominant arms contribute to the development of injury and the predictive ability of this screening to identify those who develop SAIS. Determining the influence of these modifiable physical characteristics on AHD will potentially enable clinicians to identify predisposing risk factors for subacromial impingement, implement therapeutic intervention strategies, and subsequently reduce the risk of developing this cascade of injury.

Participants in this study did not demonstrate significant differences in AHD between dominant and non-dominant arms and muscle stiffness and PML were not able to predict AHD in the dominant or non-dominant arm. Our results did not support our hypotheses that there would be a significant difference in AHD between dominant and non-dominant arms or that PML and muscle stiffness would be significant predictors of AHD. However, it is possible that because we used a healthy population we did not see the originally expected differences in AHD. In a healthy shoulder, a normal subacromial space distance is between 6-14mm, but is affected by normal overhead movements. At 30° of abduction, the subacromial space is at its maximum width, whereas it narrows to its minimum at 120°, with the majority of spatial reductions occurring between 60 and 120° of abduction (Graichen, Stammberger, et al., 1999). The dominant limb in our study demonstrated an average AHD of approximately 9mm at 45° abduction falling within the normative range described by

previous studies (Graichen, Stammberger, et al., 1999). A significant decrease in AHD serves to increase the mechanical compression on the contents of the subacromial space and is a risk factor for the development of SAIS. A decrease in subacromial space, identified using diagnostic US, has been found on the affected shoulder of individuals with impingement syndrome when compared to healthy controls (Cholewinski, Kusz, Wojciechowski, Cielinski, & Zoladz, 2008). Because healthy overhead athletes were used in this study, significant decreases in AHD were not present; however, there may be significant differences in AHD, muscles stiffness, and PML in overhead athletes with SAIS. Future research should determine if these differences in PML and muscle stiffness between sides are evident in an injured population of overhead athletes as well as determining the predictive value of these variables on AHD within shoulders clinically diagnosed with SAIS.

Limitations of this study should be noted. Overhead athletes function within their respective sports at about 90° of shoulder abduction or greater. However, we assessed active muscle stiffness and AHD at 45° of shoulder abduction. It is likely that at greater degrees of abduction, we would have seen greater stiffness values. Yet, because myotonometer measurements can be compromised by proximity of other muscles, it is therefore possible that our stiffness measurements do not fully reflect the true stiffness values. It is also possible that we might have seen greater reductions in AHD at greater degrees of shoulder abduction; however, due to the limitations of measurement of the AHD with US at greater arm elevation, this study could not examine the relative contribution of muscle stiffness to AHD in the functional position common to overhead athletes. However, this study was designed with the intention to use clinically applicable tools for evaluation of PML, muscle stiffness, and AHD. Other research could potentially use 3D MRI to evaluate AHD at functional

positions of glenohumeral abduction normal to the athletic shoulder. Lastly, it is important to note that the majority of the participants in this study participate in a unilateral sport, with the exception of swimmers. Because of the bilateral demands of swimming, it is possible that this may have altered some of our results. However, as part of some additional analyses we excluded swimmers to determine if findings of our study differed. Our results remained consistent regardless of whether swimmers were included in the dataset.

CONCLUSION

These findings indicate differences in muscle stiffness and PML between arms that may be contributing to injury risk in the dominant arm of overhead athletes. Side-to-side differences in muscle stiffness and PML in collegiate overhead athletes may indicate alterations in glenohumeral and scapular kinematics which may predispose the athlete to develop SAIS. Clinicians could use this as a screening tool to identify individuals with side-to-side differences and then implement interventions to address these asymmetries. Further research is needed to determine the relationship between muscle stiffness of the superficial shoulder musculature and subacromial space distance in overhead athletes with SAIS.

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