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Virginia Commonwealth University

This is to certify that the thesis prepared by Nitin Kalra entitled 'Effect of posture on acromiohumeral distance with arm elevation in subjects with and without rotator cuff disease using ultrasonography', has been approved by his or her committee as satisfactory completion of the thesis requirement for the degree of Master of Science in Anatomy and Neurobiology

Lori A. Michener, Ph. D., Director of Thesis

Thomas P. Mayhew, Ph.D., School of Allied Health Professions

Stephen A. Gudas, Ph. D., School of Medicine

Dr. John T. Povlishock, Ph.D., Chair, Department of Anatomy

Jerome F. Strauss, III, M.D., Ph.D., Dean, School of Medicine

F. Douglas Boudinot, Ph.D., Dean, Graduate School

July 24, 2009

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EFFECT OF POSTURE ON ACROMIOHUMERAL DISTANCE WITH ARM
ELEVATION IN SUBJECTS WITH AND WITHOUT ROTATOR CUFF DISEASE
USING ULTRASONOGRAPHY

A Thesis submitted in partial fulfillment of the requirements for the degree of Master of
Science at Virginia Commonwealth University.

by

NITIN KALRA

Bachelor of Physical Therapy, Manipal University, India, 2004

Director: Lori A. Michener, PT, PhD, ATC, SCS
Associate Professor, Department of Physical Therapy

Virginia Commonwealth University
Richmond, Virginia

August, 2009

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Abstract

EFFECT OF POSTURE ON ACROMIOHUMERAL DISTANCE WITH ARM ELEVATION IN SUBJECTS WITH AND WITHOUT ROTATOR CUFF DISEASE USING ULTRASONOGRAPHY

By Nitin Kalra, BPT

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

Virginia Commonwealth University, 2009

Major Director: Lori A. Michener, PT, PhD, ATC, SCS
Associate Professor, Department of Physical Therapy

Objectives: To examine the effects of posture on subacromial (SA) space with arm elevation in patients with rotator cuff disease (RCD) and healthy subjects.

Background: Poor posture has been linked to altered scapular mechanics, theorized to lead to excessive SA space narrowing. However, no study has examined the direct effects of posture on SA space using ultrasonography. We hypothesize that when compared to a neutral posture, upright posture will increase SA space and slouched posture will decrease SA space.

Methods: Subjects with painful RCD (n=31) and healthy (n=29) shoulders participated. SA space was measured on ultrasound generated images using the acromiohumeral distance (AHD), which is the shortest distance between acromion and the humerus. Two trials each were collected in three postures of normal, slouched and upright posture with the arm at two angles of rest (0° elevation) and 45° abduction.

Results: Two RMANOVAs for each arm angle compared the AHD across postures and groups. There was no interaction between group and posture, and no significant main effect of group for AHD. Groups were collapsed for further analysis. There was a significant main effect of posture on AHD at the 45° abduction ($p = 0.0002$); AHD increased significantly from normal (AHD= 8.63mm) to upright (AHD= 9.76mm) posture.

Conclusion: The effect of posture on SA space is minimal; only upright posture increasing AHD over normal posture by 1.13mm, at 45° abduction position. Research is needed to determine the effects of posture on shoulder pain and posture interventions.

CHAPTER 1 Relationship between Upper Quadrant Posture and Shoulder Rotator Cuff Disease

Introduction

Shoulder pain is the second most common cause of musculoskeletal pain, accounting for at least 16 % of musculoskeletal pain (Picavet & Schouten, 2003). Rotator cuff (RC) disease is reported as the most common cause of shoulder pain when presenting to physician practices within or outside the United States (Chard, Hazleman, Hazleman, King, & Reiss, 1991; van der Windt, Koes, de Jong, & Bouter, 1995; Vecchio, Kavanagh, Hazleman, & King, 1995; Wofford, Mansfield, & Watkins, 2005). The prevalence of patients with shoulder pain was reported between 31 to 48 % during a survey of general physician practices (Pope, Croft, Pritchard, & Silman, 1997). A survey of physical therapy outpatient practices in the United States ranks shoulder pain as the second highest percentage of patients, following those reporting low back pain (Jette & Davis, 1991). The total cost for treatment of shoulder disorders in the United States in the year 2000 was estimated at \$7 billion (Meislin, Sperling, & Stitik, 2005). With such huge costs to the society, the mechanisms involved in the development of shoulder pain deserve attention.

RC disease involves a broad spectrum, to include RC tendinitis, impingement, partial thickness tear and full thickness tear. One of the many factors theorized to lead to

shoulder pain and RC disease is postural deviation (Borstad, 2006; Michener, McClure, & Karduna, 2003). Theoretically, postural deviations such as slouched posture, forward head, rounded shoulder, and thoracic hyperkyphosis produces soft tissue changes, altered muscle performance and scapular and glenohumeral kinematic changes leading to excessive reduction of the subacromial (SA) space and impingement of the RC with repetitive overuse. This repetitive impingement then may progress to a rotator cuff tear. The literature has reported contradictory findings of the relationships between upper quadrant posture, shoulder impairments and RC disease. Moreover, the effect of posture and the change in posture on shoulder pain due to RC disease is unclear. This review presents the literature exploring the soft tissue, muscle, and kinematic impairments involved with alterations in posture and observed in patients with RC disease, and how they may contribute to the impingement of the RC in the SA space. This paper reviews upper quadrant posture and the various clinical variables which are affected by postural deviations of the upper quadrant as well as by RC disease. Additionally this paper examines the relationships between upper quadrant postural deviation and RC disease, and explores the concurring points as well as disagreeing views.

Posture and Pain

Posture is one of the several etiologic factors linked to the pathogenesis of shoulder pain (Bullock, Foster, & Wright, 2005; Greenfield, Catlin, Coats, Green, McDonald, & North, 1995a; Michener et al., 2003). Sitting posture has been documented as a risk factor in the development of upper quarter musculoskeletal pain in adolescents

and children (Prins, Crous, & Louw, 2008). Niemi et al found that among girls, static sitting postures were significantly associated with neck and shoulder pain (Niemi, Levoska, Kemila, Rekola, & Keinanen-Kiukaanniemi, 1996). These results concur with another study that found significant association between neck and shoulder pain and time on task with static sitting (Harris & Straker, 2000). Cho, Hwang and Chen administered a questionnaire to high school students to determine the factors that they considered were most associated with neck and shoulder pain (Cho, Hwang, & Chen, 2003). The students reported posture to be the most important contributing factor for neck and shoulder pain. One to four hours of sitting and working in front of the computer has been equated to increase in 43 to 71 % discomfort in neck and shoulder (Ramos, James, & Bear-Lehman, 2005), and lesser trunk movement was associated with reports of upper back and neck pain (Murphy, Buckle, & Stubbs, 2004). It has been proposed that “slouched” posture, defined by forward head posture, rounded shoulders, and increased thoracic kyphosis, creates a muscular imbalance at the shoulder, and the scapula rotates forward and downward, depressing the acromion (Kibler, 2006). The supraspinatus tendon and the subdeltoid bursa may become impinged against the acromion during elevation of the humerus. However there are other studies which challenge the relationship between poor posture and pain (Bullock et al., 2005; Griegel-Morris, Larson, Mueller-Klaus, & Oatis, 1992). It still remains unclear whether correcting posture would improve patients’ shoulder symptoms in terms of pain. Griegel and Morris (1992) found that subjects with rounded shoulders, severe kyphosis, and forward head showed greater correlation with interscapular pain, but not with shoulder or upper-arm pain (Griegel-Morris et al., 1992).

However, since their studies were performed with healthy subjects, the description of pain by subjects may be inaccurate. Another study on shoulder impingement patients indicated that erect posture increased pain-free shoulder ROM; however there was no significant reduction of pain intensity after assuming erect position (Bullock et al., 2005). Therefore researchers have analyzed effect of posture and shoulder pain on various clinical variables like soft tissue changes, capsular tightness and shoulder ROM and strength of glenohumeral musculature in an attempt to identify relationship between posture and shoulder pain.

Posture is defined as the state of musculoskeletal balance that involves a minimal amount of stress or strain to the body (Kendall, McCreary, & Provance, 1993). There is a widespread acceptance of the theory that deviation from ideal posture will produce abnormal joint stress, and a subsequent imbalance of the surrounding musculature. With chronic postural changes, soft tissues on one side of the joint will lengthen, while soft tissues on the other side will shorten, creating muscular imbalance (Kendall et al., 1993). Basic science studies elicit the mechanisms at cellular level, which help to explain the resultant muscular stiffness and imbalance involved with chronic postural changes. Studies have described cellular changes when a muscle is lengthened or shortened (P. Williams, Kyberd, Simpson, Kenwright, & Goldspink, 1998; P. E. Williams & Goldspink, 1978; P. E. Williams, Catanese, Lucey, & Goldspink, 1988). When muscle fibers are shortened, their range of contraction is shortened (P. E. Williams et al., 1988). Coupled with lack of stretch, the contractile elements or sarcomeres are lost (P. E. Williams & Goldspink, 1978), and there is accumulation of excessive connective tissue

or collagen (P. E. Williams et al., 1988). This leads to the muscle stiffness. Lengthening or stretch of muscle tissues increase the number of serial sarcomeres, along with increase in the connective tissue elements in the endomyseal and perimyseal tissues (P. Williams et al., 1998). This increase in the ratio of connective tissues to contractile elements has been shown to damage the endomyseal and perimyseal connective fibers, adding to the stiffness of the joint. Adaptive changes in the soft tissues can likely lead to biomechanical alterations creating an imbalance in sequencing of the activation of muscles, and ultimately movement dysfunctions (Borstad, 2006; Sahrmann, 2002).

Upper quadrant posture

The upper quadrant posture is comprised of alignment of head, cervical and thoracic spine, shoulder and scapular position in relation to each other. Poor posture has been defined in terms of Forward Head Rounded Shoulder posture (FHRSP) by one author (Thigpen, 2006) or as increased thoracic angle by another (Kebaetse, McClure, & Pratt, 1999). Thigpen categorized subjects with FHRSP posture as those with average of 46 degrees or greater of Forward Head (FH) angle and 52 degrees or greater of Forward Shoulder (FS) angle, versus those without FHRSP having an average of 36 degrees or lesser of FH angle and 22 degrees or lesser of FS angle. FH angle reflects the amount of cervical flexion (Thigpen & Padua, 2006) while FS posture is defined as a position of abduction and elevation of scapula, which may appear as winging of scapula, and medial rotation of humerus (Kendall et al., 1993). Kebaetse et al described subjects with slouched posture as having a mean thoracic angle of 38.5 degrees as compared to mean

thoracic angle of 26.4 degrees in erect posture (Kebaetse et al., 1999). Hence 'Poor' or slouched posture can be described as the position of increased thoracic spine flexion, forward head (FH) posture, and forward shoulder (FS) posture. However, a postural study on 160 asymptomatic subjects revealed that all three components of slouched posture (increased thoracic spine flexion, FH posture, and FS posture) may not be present or occur concurrently (Raine & Twomey, 1997).

Scapular and Glenohumeral Kinematics, Upper Quadrant Posture and RC Disease

Normal Kinematics. During arm elevation, the scapula rotates upward, rotates externally, and tilts posteriorly on the thorax (McClure, Michener, Sennett, & Karduna, 2001). The largest amount of scapular motion is upward rotation, occurring approximately linearly with respect to humeral motion throughout humeral elevation above 20-30 degrees of humeral elevation (McClure et al., 2001). Scapular posterior tilt occurs in a non-linear fashion, occurring primarily when the arm is elevated above 90 degrees (McClure et al., 2001). Internal rotation has a similar magnitude as posterior tilting during humeral elevation. Internal rotation was found to increase in the early range of arm elevation up to approximately 100 degrees of elevation (Borstad & Ludewig, 2002), while at the end range of elevation scapular external rotation has also been demonstrated (McClure et al., 2001). During arm elevation, the glenohumeral joint acts as essentially as a ball and socket joint, fluctuating between inferior and superior humeral translations (Michener et al., 2003). Ludewig and Cook reported an average of 0.1 to 1.6 mm of overall superior translation in a healthy group of subjects across 3

phases of arm elevation – 30-60 degrees, 60-90 degrees, and 90-120 degrees (Ludewig & Cook, 2002). Other researchers have reported a superior translation of 0.7 mm to 1.1 mm for 0-120 degrees of arm elevation (Deutsch, Altchek, Schwartz, Otis, & Warren, 1996; Poppen & Walker, 1976).

Scapular Kinematics, Upper Quadrant Posture and RC Disease

Scapular Posterior Tilt. Reduced scapular posterior tilt with arm elevation with poor upper quadrant posture as compared to normal or resting posture in healthy subjects has been demonstrated in three studies (Finley & Lee, 2003; Kebaetse et al., 1999; Thigpen, 2006) . Poor posture was defined as slouched posture in two studies (Finley & Lee, 2003; Kebaetse et al., 1999) by asking patients to slouch ‘down’ thereby increasing thoracic flexion, and in one study (Thigpen, 2006) by recruiting patients with FHRSP. No studies have examined the effect of poor posture on scapular kinematics in patients with RC disease. Patients with RC disease have by in large demonstrated the same pattern of reduced scapular posterior tilting with arm elevation as compared to healthy individuals, but not controlling for the effect of thoracic spine posture in four prior studies (Endo, Ikata, Katoh, & Takeda, 2001; Lin et al., 2005; Ludewig & Cook, 2000; Lukasiewicz, McClure, Michener, Pratt, & Sennett, 1999). However two prior studies found increased scapular posterior tilt with arm elevation in patients with RC disease (Laudner, Myers, Pasquale, Bradley, & Lephart, 2006; McClure, Michener, & Karduna, 2006); and another found no significant difference in posterior tilt between subjects with shoulder disease and healthy (Hebert, Moffet, McFadyen, & Dionne, 2002). This body of

evidence indicates that decreased scapular posterior tilt may be a common phenomenon in healthy subjects with slouched or FHRSP posture and in patients with RC disease.

Scapular Upward Rotation. Increased upward rotation of the scapula was demonstrated in pain-free individuals with FHRSP as compared to subjects without FHRSP (Thigpen, 2006). Conversely, Finley and Lee found no difference in scapular upward rotation in pain-free individuals with slouched posture (Finley & Lee, 2003), inducing slouched posture by asking the subjects to slouch 'down'. Similarly, there is also discrepancy about upward rotation in studies examining patients with RC disease as compared to healthy subjects. Three studies reported decreased upward rotation of the scapula in patients with RC disease (Endo et al., 2001; Lin et al., 2005; Ludewig & Cook, 2000; Su, Johnson, Gracely, & Karduna, 2004); one found increased upward rotation (McClure et al., 2006); and four found no rotational differences in patients with RC disease when compared with healthy subjects (Graichen et al., 2001; Hebert et al., 2002; Laudner et al., 2006; Lukasiewicz et al., 1999). The effect of slouched posture on scapular upward rotation in healthy subjects is inconclusive. In patients with RC disease, the presence of decreased scapular upward rotation during arm elevation is likely. There is a need for further research to elucidate the relationship between scapular upward rotation and both RC disease and poor upper quadrant posture.

Scapular Internal Rotation. Increased scapular internal rotation in healthy subjects with FHRSP has been reported during arm elevation with loaded forward-reaching task (Thigpen, 2006). In patients with RC disease, two studies showed an increase in internal rotation of the scapula (Hebert et al., 2002; Ludewig & Cook, 2000),

while five other studies found no significant differences between patients with RC disease and healthy subjects (Endo et al., 2001; Laudner et al., 2006; Lin et al., 2005; Lukasiewicz et al., 1999; McClure et al., 2006). Increased scapular internal rotation appears to be associated with a posture of forward head and rounded shoulders; however in patients with RC disease it does not appear to be a critical variable associated with RC disease.

The studies (Endo et al., 2001; Finley & Lee, 2003; Graichen et al., 2001; Hebert et al., 2002; Kebaetse et al., 1999; Laudner et al., 2006; Lin et al., 2005; Ludewig & Cook, 2000; Lukasiewicz et al., 1999; McClure et al., 2006; Thigpen, 2006) investigating scapular kinematics with patients with poor upper quadrant posture and in patients with RC disease are discordant, which is likely due to limitations and techniques particular to the various studies. For example, some studies included a smaller sample size, which reduced the statistical significance and power (Ludewig & Cook, 2000; McClure et al., 2001). A few studies (Ludewig, Cook, & Nawoczenski, 1996; Thigpen, 2006) were conducted with subjects holding weighted objects in their hands to simulate practical situations, while others (Laudner et al., 2006; Lukasiewicz et al., 1999) included no such weight-holding. Based upon the totality of literature to date, there is evidence to suggest a relationship between poor posture and altered scapular kinematics. The findings of decreased posterior tilt, decreased upward rotation and increased internal rotation of the scapula are common factors observed in subjects with poor posture, and in patients with shoulder pain related to RC disease. The scapular orientation of excessive downward rotation, anterior tilt, and internal rotation may bring the acromion of the scapula closer to

the RC tendons and subacromial bursae, thus contributing to impingement of the bursae and tendons with repetitive overhead activity (Flatow et al., 1994; Fu, Harner, & Klein, 1991; Ludewig & Reynolds, 2009; Michener et al., 2003). The Figure 1.1 shows the schematic representation of the relationship between scapular kinematics, RC disease and poor upper quadrant posture.

Glenohumeral Kinematics, Upper Quadrant Posture and RC Disease

In patients with RC tears, the control of excessive superior translations is lost (Deutsch et al., 1996; Poppen & Walker, 1976; Yamaguchi et al., 2000). Abnormal superior translation of the humeral head of 1 mm–to-1.5 mm increase in subjects with full RC tears as compared to healthy subjects (Deutsch et al., 1996; Yamaguchi et al., 2000). Chen et al reported an excessive superior translation of the humeral head when the RC muscles were fatigued (Chen, Simonian, Wickiewicz, Otis, & Warren, 1999). Fatigue of RC muscles can be compared with RC tear as RC muscles do not function normally. It is theorized that with excessive humeral head anterior translation, the humeral head will tend to come closer to the acromion, compressing the soft tissue contents of the SA space against the acromion. There are no studies examining the effect of posture on humeral kinematics.

Soft Tissue Length, Upper Quadrant Posture and RC Disease

Posture Impairment Theory describes postural deviations such as FH posture, FS posture, and thoracic kyphosis can contribute to other impairments and shoulder pain

(Sahrmann, 2002). Studies of healthy subjects (Borstad, 2006; Finley & Lee, 2003; Griegel-Morris et al., 1992; Kebaetse et al., 1999), and one study of patients with internal impingement (Myers, Laudner, Pasquale, Bradley, & Lephart, 2006) examining soft tissue changes with posture and the relationship with shoulder pathology support the Posture Impairment theory, with shorter tissue length associated with postural deviations. Length of the pectoralis minor (Borstad, 2006) and length of the posterior capsule (Myers et al., 2006; Tyler, Nicholas, Roy, & Gleim, 2000) have both been implicated as a cause of altered posture and shoulder pain. Borstad (Borstad, 2006) found a positive correlation between increased thoracic flexion (kyphosis) to shorten pectoralis minor muscle length in healthy subjects. The study also found increased internal rotation of the scapula in patients with shortened pectoralis minor muscle length, thus confirming that an anatomical change (pectoralis minor's length) was correlated with a postural denominator (thoracic kyphosis) and a kinematic alteration (increased scapular internal rotation). These results concur with another study of healthy subjects which found that slouched posture, which induced an increase in thoracic flexion by asking subjects to slouch, resulted in decreased scapular posterior tipping and external rotation (Finley & Lee, 2003). It has been proposed that subjects with increased scapular internal rotation and shorter pectoralis minor muscle lengths, coupled with kinematic alterations, may be at risk for developing RC disease via impingement (Soslowsky et al., 2002). Healthy baseball pitchers but exposed to repetitive overhead activity (Tyler et al., 2000), and throwers with internal impingement (Myers et al., 2006) have demonstrated significant

posterior capsule tightness with limited shoulder ROM. These studies point out that soft tissue changes may be linked with RC disease and postural deviations.

However, some authors have presented evidence contradictory to the results reported above, posing challenges to this theory (Greenfield, Catlin, Coats, Green, McDonald, & North, 1995a; Lewis, Green, & Wright, 2005; Raine & Twomey, 1997; Raine & Twomey, 1997). Lewis et al (Lewis et al., 2005) analyzed the relationship between six postural variables (FH posture, FS posture, thoracic kyphosis, scapular protraction, and the ranges of glenohumeral flexion and abduction) in 60 symptomatic and 60 asymptomatic subjects. They found no patterns of correlation between FH posture, thoracic kyphosis, and scapular deviations. Similarly, other researchers have found no correlation between FH posture, FS posture and the thoracic kyphosis (Raine & Twomey, 1997)(Raine & Twomey, 1997); and no relationship between FH posture, thoracic kyphosis and scapular position in individuals with and without shoulder injuries (Greenfield, Catlin, Coats, Green, McDonald, & North, 1995b). Hence, the relationship between posture, abnormal joint kinematics, and RC disease remains ambiguous.

Shoulder Range of Motion, Upper Quadrant Posture and RC Disease

A strong correlation between slouched posture and decreased shoulder ROM has been documented (Bullock et al., 2005). Kebaetse et al noted significantly less ROM of shoulder elevation with slouched posture than with erect posture in 43 healthy subjects (Kebaetse et al., 1999). Limitation in the ROM of the shoulder has been directly linked with disability: Chakravarty and Webley examined ROM and functional limitations

(grooming, bathing, and dressing) among 100 elderly subjects (Chakravarty & Webley, 1993). They found that functional limitations were significantly correlated with limited shoulder ROM. It has been suggested that 17 degrees of increase in shoulder ROM by postural correction may lead to improvement in perceived difficulty level from “very difficult” to “somewhat difficult” in the performance of some tasks (Triffitt, 1998). These studies indicate that poor posture may contribute to restricted ROM, leading to disability. Postural correction with taping in subjects with sub acromial impingement syndrome (SAIS) has resulted in improved pain-free ROM (Lewis, Wright, & Green, 2005).

Patients with shoulder impingement also have been determined to have restricted abduction (Norwood, Barrack, & Jacobson, 1989), restricted flexion (Ardic et al., 2006), and restricted exterior rotation (ER) and interior rotation (IR) of the shoulder (Ardic et al., 2006). Patients with RC disease have reported a loss of function and disability in activities of daily living on tests such as the Simple Shoulder Test (SST) questionnaire and the Short Form-36 (SF -36) (Duckworth, Smith, Campbell, & Matsen, 1999; K. L. Smith et al., 2000). Hence, patients with shoulder overuse injuries also demonstrate restricted ROM like subjects with poor posture, which may be a common factor linked with loss of function and disability.

Muscle Performance, Upper Quadrant Posture and RC Disease

Kebaetse et al tested shoulder isometric strength at 90 degrees abduction in the scapular plane in healthy subjects with slouched posture. The study documented isometric strength reduced by 16% in subjects with slouched posture as compared to

those with erect posture, along with reduced active shoulder elevation, and reduced scapular posterior tilting (Kebaetse et al., 1999). It is hypothesized that protraction may reduce the mechanical efficiency of shoulder musculature, which may be the cause of reduced shoulder strength (Kibler, 1998; Kibler, Sciascia, & Dome, 2006). Smith and Kotajarvi (J. Smith, Kotajarvi, Padgett, & Eischen, 2002) also found reduced shoulder elevation strength with slouched posture, defined as shoulder protraction, of 23% in protraction as compared to neutral posture. They also found a 30% reduction in upright posture, defined by shoulder retraction as compared with strength at neutral posture (J. Smith et al., 2002). Kebaetse et al (Kebaetse et al., 1999) examined shoulder elevation strength only in a slouched posture but Smith and Kotajarvi (J. Smith et al., 2002) ensured at least 5 cm of scapular protraction, and 2 cm scapular retraction as compared to the neutral position, using a tape measure. Shoulder elevation strength is reduced with a slouched or shoulder protracted position as compared to a neutral posture. The unexpected results of reduced shoulder elevation strength in the retracted shoulder position is contrary to theory. The effect of posture shoulder strength requires further research.

Patients with RC disease also demonstrate altered musculature strength. This may be due to a combination of pain, limited ROM, altered kinematics and capsular tightness. Research has reported capsular tightness in patients with shoulder impingement in the anterior aspect (Flatow et al., 1994), posterior aspect (Warner, Micheli, Arslanian, Kennedy, & Kennedy, 1990), and inferior aspect (Hjelm, Draper, & Spencer, 1996) of the capsule. Posterior shoulder tightness may also alter the length–tension relationship

between scapular muscles, leading to a reduction in strength of the shoulder abductors (Kebaetse et al., 1999). In patients with shoulder overuse injuries, altered muscle strength ratios between external rotators (ER) and internal rotators (IR) have been shown to exist (Bak & Magnusson, 1997). The weaker ER may not be able to resist the superior translational force of the deltoid, causing the humerus to displace upwards (McCully, Suprak, Kosek, & Karduna, 2006). This process in turn may lead to excessive reduction of SA space in subjects with RC disease (Girometti et al., 2006). These studies offer evidence that patients with slouched posture as well as those with shoulder overuse injuries demonstrate altered patterns of muscle strength. Figure 1.2 illustrates the interplay of all clinical variables with scapular and glenohumeral kinematics.

Poor Upper Quadrant Posture and Rotator Cuff Disease: Effect on Subacromial Space

There are two main theories of RC disease, extrinsic and intrinsic. Extrinsic or outlet impingement is the predominantly held theory describing compression of the SA space structures due to repetitive compression (Neer CS II, 1983). Intrinsic impingement leading to RC disease describes intrinsic breakdown within the tendon due to repetitive tensile overload. Scapular alterations have been demonstrated to alter the SA space, a decrease in SA space with shoulder protraction, and an increase with retraction using MRI images to measure the linear distance between the anterior acromion and humeral head (Solem-Bertoft, Thuomas, & Westerberg, 1993). Subjects with thoracic spine hyperkyphosis also have been reported to have decreased SA space as compared to those

without hyperkyphosis, via linear measurements of SA space on radiographs (Gumina, Di Giorgio, Postacchini, & Postacchini, 2008). Patients with SAIS have demonstrated a reduction of 68% of the SA volume measured on MRI during arm elevation, with an average of 3 mm smaller linear acromiohumeral distance (Graichen et al., 1999). Using linear measurement from radiographs, the SA space has been shown to reduce up to 5 mm in cadavers with a deficient RC during arm elevation (Flatow et al., 1994). These studies indicate there may be a relationship between poor posture, reduced SA space and development of shoulder rotator cuff problems, illustrated in Figure 1.2.

Interplay of Poor Posture and Rotator Cuff Disease. The schematic in Figure 1.2 indicates the possible connections between poor posture and RC disease, relating mechanisms of RC disease. Poor posture may cause soft tissue tightness around the shoulder (Borstad, 2006), affecting the normal strength of muscular forces around the shoulder (J. Smith et al., 2002) and may make the shoulder vulnerable to overuse injury (Kibler, 1998; Sahrmann, 2002). An increase in thoracic kyphosis may lead to increased anterior tilt of the scapula, decreased upward rotation, and increased internal rotation (Kebaetse et al., 1999). This may narrow the anterior aspect of subacromial space (Solem-Bertoft et al., 1993), leading to excessive impingement. Excessive impingement may in turn lead to RC tear, causing pain, soft tissue tightness (Myers et al., 2006; Soslowsky et al., 2002; Tyler et al., 2000), decreased ROM (Ardic et al., 2006; Norwood et al., 1989), with reduced abductor and external rotator strength (Bak & Magnusson, 1997; Kebaetse et al., 1999), exacerbating the RC disease.

Conclusion

The current literature indicates that patients with poor upper quadrant posture and shoulder RC disease present similarly in terms of scapular and glenohumeral kinematics, soft tissue changes, muscular performance, and clinical manifestation of shoulder pain. There is limited evidence to indicate that both poor posture and RC disease are related to reduced SA space. However, the direct effect of poor upper quadrant posture on patients with shoulder RC disease is still largely unknown, and has not been adequately studied. The current literature reports contradictory results; more research is needed to quantify the effects and relationships of poor posture and RC disease to facilitate the development of better treatment programs.

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Figure 1.1. Theoretical Model of the relationships between impairments, upper quadrant posture and RC disease. Items in italics have evidence to support this relationship.

Figure 1.2. Relationship between poor upper quadrant posture and RC Disease and reduction in subacromial space. Items in italics have evidence to support this relationship.

CHAPTER 2 Effect of posture on acromiohumeral distance with arm elevation in subjects with and without rotator cuff disease using ultrasonography

INTRODUCTION

Shoulder pain is the third most common musculoskeletal problem, accounting for at least 16 % of all musculoskeletal problems (Urwin et al., 1998). In a survey of general physician practices the prevalence of patients with shoulder pain ranged from 31 to 48 % (Pope, Croft, Pritchard, & Silman, 1997). In the year 2000 overall treatment cost of shoulder pain was estimated at 7 billion in the United States (Meislin, Sperling, & Stitik, 2005). With such huge societal costs, shoulder pain warrants more research.

The most common cause of shoulder pain is rotator cuff disease (RCD) (van der Windt, Koes, de Jong, & Bouter, 1995). Upper quadrant postural deviations have been linked to RCD (Greenfield et al., 1995; Lewis, Green, & Wright, 2005; Lukasiewicz, McClure, Michener, Pratt, & Sennett, 1999; L. A. Michener, McClure, & Karduna, 2003) and general musculoskeletal pain (Griegel-Morris, Larson, Mueller-Klaus, & Oatis, 1992). Mechanistically, these upper quadrant postural deviations may lead to muscle length adaptations thus creating an imbalance that may alter normal shoulder joint motion or lead to aberrant scapular motion (Borstad, 2006). Slouched thoracic spine posture or increased thoracic kyphosis has been shown to decrease scapular upward rotation (Kebaetse, McClure, & Pratt, 1999), posterior tilting of the scapula (Culham & Peat, 1993; Finley & Lee, 2003; Kebaetse et al., 1999), and decrease external rotation of the

scapula (Culham & Peat, 1993; Kebaetse et al., 1999; Lewis et al., 2005). Theoretically, the abnormal scapular kinematics of decreased posterior tilt, upward rotation, and external rotation will decrease the subacromial (SA) space and decrease the clearance of the humeral head beneath the acromion during arm elevation (Borstad, 2006; Brossmann et al., 1996; Flatow et al., 1994; Hebert, Moffet, Dufour, & Moisan, 2003; Solem-Bertoft, Thuomas, & Westerberg, 1993). One study has directly measured the effect of scapular position on SA space, demonstrating a decrease in SA space via Moiré topography with artificially induced scapular protraction (Solem-Bertoft et al., 1993). Greenfield et al (Greenfield et al., 1995) examined forward head posture and scapular movements in healthy subjects and patients with shoulder pain. However, they found no relationship between scapular movements and posture, which may be due to the measurement of only one motion of the 3-dimensional scapular motion. The effect of postural abnormalities on scapular kinematics and SA space in patients with shoulder pain remains unclear.

Abnormal scapular patterns resulting from altered posture are similar to what has been observed in patients with RCD, specifically shoulder subacromial impingement syndrome (Ludewig & Cook, 2000; Lukasiewicz et al., 1999). Two studies have demonstrated a reduction of the space between the acromion and humerus (SA space) in patients with subacromial impingement syndrome as compared to healthy subjects (Desmeules, Minville, Riederer, Cote, & Fremont, 2004; Graichen et al., 1999). However, no studies have examined the effects of posture on SA space in patients with RCD. Studies on shoulder overuse has also linked poor posture (Greenfield et al., 1995)

and impingement syndrome (Hawkins & Kennedy, 1980; McCann & Bigliani, 1994), implying an association between poor posture, abnormal shoulder mechanics and RCD.

Several attempts have been made to examine the upper quadrant posture-impairment mechanism in patients with shoulder pathologies. Patients with shoulder overuse injuries have demonstrated increased forward head posture (Greenfield et al., 1995). Thoracic kyphosis or artificially induced 'slouched' posture has been associated with decreased glenohumeral abduction strength (Kebaetse et al., 1999), while upright or erect posture was associated with increased glenohumeral elevation (Bullock, Foster, & Wright, 2005; Lewis, Wright, & Green, 2005). This suggests that poor or 'slouched' posture may limit shoulder motion due to impingement beneath the acromion, creating a mechanical block to shoulder elevation coupled with tissue impingement (Donatelli R, 2004; Neer CS II, 1983). Taping applied to the posterior trunk along the thoracic spine and over the scapula to patients with shoulder pain increased thoracic extension, reduced their pain with shoulder elevation and improved the resting scapular position (Lewis, Wright et al., 2005). These studies provide evidence that increased thoracic flexion and slouched posture is linked to shoulder pain, decreased glenohumeral strength, and limited range of motion contributing to functional disability. However, what is not established is the mechanisms that explain the link between upper quadrant posture, shoulder pain and RCD.

Investigation of SA space with various posture positions in patients with RCD will expand our understanding of the mechanisms of posture on RCD. Moreover, it will provide mechanistic evidence for postural correction as a rehabilitation intervention. The

purpose of this study was to determine the effect of posture on SA space at various arm elevations in subjects with and without RCD. We hypothesized that slouched posture of the thoracic spine would lead to a reduction in SA space, and upright posture would lead to an increase in SA space.

METHODS

Subjects

Subjects with RCD (n=31) and subjects with healthy (HLTY) shoulders (n=29) were recruited through the physician offices at the University Health System. Healthy subjects were recruited through Internal Review Board-approved advertisement flyers posted across the University Health System and University campus. This study was approved by the Institutional Review Board at the University. An informed consent form was signed by each subject prior to the data collection procedure.

The inclusion criteria for the RCD group were: a) shoulder pain; b) MRI confirmation of RCD (tendinosis, bursitis, impingement, or partial thickness and full thickness rotator cuff tear); c) ability to lift the arm up to 90 degrees of elevation. If patients had two or more diagnoses, the diagnosis with the greatest RCD pathology was used to classify group membership. The descriptive statistics and demographics are shown in Tables 2.1 and 2.2. The exclusion criteria for the RCD were: a) range of motion of the neck that reproduced shoulder pain; b) presence of pain below the elbow, which might indicate cervical and nerve pathologies; c) past shoulder surgery; d) presence of glenohumeral joint arthritis as indicated in the MRI report. The inclusion criteria for the

HLTY shoulder group were that subjects had to be pain-free, with no previous or current shoulder pathology. Exclusion criteria for the HLTY group were: a) history of rotator cuff injury; b) previous shoulder surgery.

To determine the sample size, we used data derived from a study of test-retest reliability of AHD measures on 10 subjects with RCD and 10 subjects without shoulder pain. For the rest and 45 degree abduction position, respectively the ICC (2-way random) was 0.78 and 0.76, and the standard error of the measure (SEM) was 2.3 and 2.5mm (L. A. Michener, Kalra, Pinkstaff, Ericksen, & Boardman, 2007). Based on this data, we wanted to have a minimal difference of 2.5mm between normal posture and both slouched and upright postures, and the average standard deviation was conservatively estimated at 4.0mm. For a power of 90%, alpha set at 0.05 using repeated measures ANOVA, the sample size calculated indicated 10 subjects were required.

Procedure

This was a controlled laboratory study with repeated measures comparison of two groups. After signing the informed consent form, the subjects completed an intake form consisting of demographic information and information regarding their shoulder symptoms, and the American Shoulder and Elbow Surgeons patient self-report shoulder score to measure shoulder functional loss and disability (TABLE 2.2). Next, the SA space was measured in three postures at two arm angles.

Subacromial Space Measurement.

The SA space was measured by the acromio-humeral distance (AHD) from ultrasound generated images. The AHD is defined as the shortest distance between the

acromion and the humerus (FIGURE 2.1). An ultrasound unit (The Pyramid 764; Pyramid med Inc.; Los Alamitos, CA) with a 7.5 MHz linear ultrasound transducer was utilized for all ultrasound images. The ultrasound probe was placed on the posterior lateral shoulder, and adjusted until the image of the acromion and humerus (as shown in FIGURES 2.5 AND 2.6) was visualized. All ultrasound images were saved on a computer for blinded measurements of AHD performed later. Two images were taken at each of the two arm positions: 1) at rest with the arm by the trunk; and 2) at 45 degrees of active abduction. For the 45-degree arm position, the arm was suspended in an adjustable sling set up to the desired angle when the arm was held actively above the sling (FIGURE 2.7). This sling allowed the patient to rest the arm between measurements and during setup. The subject was then instructed to hold his or her arm up and off the sling for a few seconds, while the tester verified the desired humeral position with a bubble inclinometer. Then, the ultrasound image of the AHD was captured at 45 degrees of abduction with active arm elevation. Prior to initiating the study, a reliability study was performed on healthy and RCD subjects (n=20) at the normal resting posture, with results indicating good reliability [ICC (2,1)= 0.76-0.78] across the two arm positions at 0 and 45 degrees of elevation.

Posture

Subjects were asked to assume three postures in sitting: 1) normal resting posture; 2) slouched posture; and 3) upright posture with scapular retraction. For normal posture, the subject was asked to sit in a chair with the back supported, feet flat on the floor, hips and knees at 90 degrees of flexion, head and shoulder in their habitual posture, looking

straight ahead (FIGURE 2.2). Then, the subjects were instructed to roll their shoulders three times in a circle and come to a stop. This step ensured that subjects would assume their habitual posture. Slouched posture was achieved by having each subject move forward in the chair so that their back was six inches away from the back support, then slump forward and down to attain a flexed thoracic and lumbar spine, forward head, and rounded posture (FIGURE 2.3). For upright posture, subjects were asked to sit back against the back rest of the chair with a pillow between their back and the back support. Then they were instructed to “sit up straight,” pull their shoulders back, and look straight ahead, to extend the thoracic and lumbar spine and retract the shoulder (FIGURE 2.4). For each posture, ultrasound images were collected to measure the SA space in 2 arm positions: 0 degrees abduction (rest); and 45 degrees actively maintained abduction.

Data Measurement

The AHD was measured after data collection by randomly retrieving images on the computer to avoid bias. The AHD was measured using the software ‘Universal Desktop Ruler’ (AVP Inc., Voronezh, Russia) to measure distances on-screen. First, a mark on the ruler was placed at the most inferior aspect of the acromion; then, the second marker was placed at the humeral head measuring the shortest distance between acromion and humerus. The two measurements at each arm angle were averaged for data analysis.

RESULTS

The mean and standard deviation of the AHD measure for each group - RCD, and HLTY, at each arm angle in three postures are represented in Table 2.4 and Figure 2.8.

To examine the effect of posture on AHD in the RCD and HLTY groups, analyses at each arm position were performed using a mixed model repeated measures ANOVA that included effects for: group, posture, and group-posture interaction. There were no significant interactions regarding group-posture with the arm at rest and 45° of abduction. Therefore, the effect of posture was determined to be independent of group classification. There were no statistically significant main effects of group classification at either rest or 45 degrees of abduction. With the groups collapsed, there was no statistically significant main effect of posture with the arm at rest ($F_{(2,116)}=1.4$, $p=0.2557$); however, there was a statistically significant main effect of posture at 45° of abduction ($F_{(2,115)}=9.1$, $p=0.0002$). Post hoc testing using Tukeys HSD revealed AHD was greater in the upright posture (mean AHD=9.76mm, SE=0.27) compared to normal posture (mean AHD=8.63, SE=0.26), with a mean difference=1.13mm, SE=0.27. There were no significant differences between slumped (mean AHD mm=9.24, SE=0.26) and normal or upright postures. Post hoc power analysis revealed 99% power.

DISCUSSION

The Posture Impairment Theory links the presence of postural deviations with anatomical alterations about the shoulder and subsequent impairments and pain (Borstad, 2006). One of the anatomical alterations may be change in SA space. Forward shoulder posture has been linked to altered scapulohumeral kinematics, which is theorized to cause excessive reduction of the SA space (Brossmann et al., 1996; Flatow et al., 1994; Kebaetse et al., 1999; Solem-Bertoft et al., 1993). The SA space has also been shown to

be reduced in patients with subacromial impingement syndrome (Desmeules et al., 2004; Graichen et al., 1999). For this study we induced slouched posture by asking patients with RCD and asymptomatic subjects to slump forward and down to attain a flexed lumbar and thoracic spine and forward shoulder posture. This slouched posture did not change the SA space as compared to normal posture. For this study, upright posture was induced by asking the subjects to sit up straight, retracting their shoulders and extending their thoracic and lumbar spine. Upright posture increased the SA space when compared to the normal posture position.

We examined the direct effect of posture on SA space in patients with RCD and healthy subjects across two arm angles. At 45 degrees of abduction, SA space increased as posture changed from a "normal" to an "upright" position. This supports our hypothesis that upright posture causes an increase in SA space. This increase may have the effect of relieving the symptoms of compression of the SA space structures. A prior study revealed a decrease in SA space of 0.3mm to 1.5mm with scapular protraction as compared to a retracted scapular position in 4 healthy subjects, measured with arms at rest and in the coronal plane (Solem-Bertoft et al., 1993). In our study, the change in SA space (AHD) was greater, with a mean change of 1.13mm (SD: 0.5-1.75) with the upright posture as compared to the normal posture when the arm was at 45 degrees of elevation. This result was found to be statistically significant. The minimal detectable change (MDC) value for our AHD measure was calculated to be 2.21mm, as calculated from the test retest reliability pilot study. Only 18 out of 60 subjects (30%) experienced a change in SA space of equal to or more than 2.21mm from normal to upright posture. Hence,

even though the change is statistically significant, it was less than the MDC in the measure for 70% of the subjects. Further research is needed to determine if the increase in SA space of 1.13mm with upright posture is a meaningful magnitude, as it relates to patient symptoms and function.

Slouched posture was expected to induce a decrease in SA space measurement when compared to normal posture. However, we did not find a decrease in SA space as posture changed from normal to slouched posture. What may explain this is the report by some subjects of difficulty and pain in maintaining their arm at 45 degrees of abduction in the slouched posture; therefore, they may have elevated their shoulder and thus their scapula, to relieve pain. These substitution movements may have prevented a reduction in the SA space. During the slouched posture position, the scapula has demonstrated an increase in anterior tilt (Culham & Peat, 1993; Finley & Lee, 2003; Kebaetse et al., 1999; Kebaetse et al., 1999), which is theorized to decrease the SA space. Because we measured the posterior to middle aspect of the SA space with our measurement, we may have not captured the decrease in SA space that is theorized to occur anteriorly. This may explain why a significant reduction in SA space was not seen with slouched posture. There was no difference in SA space changes with posture with or without the presence of RCD. Previous research (Azzoni & Cabitza, 2004; Azzoni, Cabitza, & Parrini, 2004; Desmeules et al., 2004) offers conflicting evidence about SA space differences between subjects with or without RCD. Some authors have linked SA space size to the severity of RCD (Azzoni & Cabitza, 2004; Azzoni et al., 2004) while others have found no statistically significant difference between the SA space of RCD patients or healthy

subjects (Desmeules et al., 2004). This study as well did not find a statistically significant difference between the two groups.

There were limitations to this study. Postural positions were artificially induced in a laboratory setting, therefore these postures may not represent faulty postures seen in patients with shoulder pain. There was no intervention for correction of posture. The mechanisms of posture interventions are critical to examine, to determine their mechanisms on RCD and their effects on patient report of pain and function. The subjects with RCD were a combined group of diagnoses of impingement, partial thickness, and full thickness tears. This heterogeneity may have decreased our ability to detect differences between the RCD and healthy groups. Third, we did not monitor scapular movements; therefore, we could not account for substitute motions or relate the changes in SA space to scapular positions. Lastly, we examined only the posterior to middle aspect of the SA space. The anterior aspect of the SA space has been demonstrated to be the area to decrease the most with clinical impingement maneuvers (Roberts, Davila, Hushek, Tillett, & Corrigan, 2002), therefore measurement of the anterior aspect of the SA space is indicated in future studies. Future studies should monitor scapular motions and examine the effects of posture within subgroups of patients according to the severity of the RCD.

CONCLUSIONS

To our knowledge, this is the first study to examine the direct effects of posture on SA space using ultrasonography. We found that upright posture had a significant

increase in SA space in patients with RCD and healthy subjects at 45 degrees of arm abduction. However, the mean change in SA space of 1.13mm from upright to normal posture fell within the range of error for 70% of the subjects indicating the study implications are limited and need further exploration. The SA space did not differ with slouched posture as compared to normal posture, and between those with or without the presence of RCD across the two arm positions.

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TABLE 2.1. Descriptive Statistics

	Patients with RCD (n=31)		Healthy Subjects (n=29)	
	Means (SD)	Range	Means (SD)	Range
Age (years)	53.48 (13.7)	(20, 80)	31.89(10.7)	(23,62)
Height (Inches)	67.00 (3.76)	(60, 74)	66.87(3.48)	(59.5, 74)
Weight (lbs)	177.70 (29.5)	(133, 254)	159.20(30.2)	(125, 250)
History of Pain (months)	18.20 (17.32)	(2, 84)	0.0	(0, 0)

TABLE 2.2. Subject Demographics

	Patients with RCD	Healthy Subjects
Total subjects (n)	31	29
Gender (n)		
Male	11	14
Female	20	15
Dominant Shoulder (n)		
Right	22	27
Left	5	2
Ambidextrous	4	0
Tested shoulder (n)		
Right	17	26
Left	14	3
RCD Diagnosis (n)		
Impingement	15	
Partial Thickness Tear	9	
Full thickness Tear	7	
ASES Pain Score (M, SD, range)	31.30 (11.81)	
0-50 points;	Range: 10, 50	
(50 = No pain)		
Function Score	25.17 (10.26)	
0-50	Range: 5, 45	
(50 = No functional loss)		
Total Score	56.95 (17.82)	
0-100	Range: 28.3, 95	
(100= No Pain/ Functional Loss)		

ASES = American Shoulder and Elbow Surgeon's Self-Report Form

TABLE 2.3. AHD (Posterior lateral AHD) in millimeters for two postures across 3 arm angles, mean, standard deviation and 95% confidence intervals. Data represents collapsed results for healthy and RCD groups

Posture	Angle of Arm Elevation	
	0 degrees Mean (SD)	45 degrees abduction Means(SD)
Slouched	12.52 (3.11)	9.24 (1.98)
(95 % CI)	(11.72-13.33)	(8.71-9.74)
Normal	12.14 (2.57)	8.63 (1.99)
(95% CI)	(11.48-12.81)	(8.11-9.14)
Upright	12.58 (2.45)	9.76 (2.02) *
(95% CI)	(11.95-13.21)	(9.24-10.29)

* represents significantly different from normal posture

FIGURE 2.1. Acromio Humeral Distance (AHD)- The colored bar represents AHD

FIGURE 2.2. Normal Posture

FIGURE 2.3. Slouched Posture

FIGURE 2.4. Upright Posture

FIGURE 2.5. Probe positioning on cadaver model

FIGURE 2.6. Probe Positioning on subject

FIGURE 2.7. Arm at 45 degrees elevation

Figure 2.8. Graph showing Mean of AHD in millimeters (mm) at three postures across 2 arm angles.

Data represents collapsed results from healthy and patients. 0 and 45 represent arm angles.

CHAPTER 3 Discussion

Impingement syndrome of the shoulder is defined as a mechanical compression and repetitive overuse of the rotator cuff (RC) tendons and bursa within in the subacromial space (SA space), leading to bursitis, tendonopathy, or RC tears (Valadie, Jobe, Pink, Ekman, & Jobe, 2000). Repetitive mechanical compression of the supraspinatus tendon by the coracoacromial arch is cited as the primary mechanism responsible for tendonopathy and tears of the RC (Michener, McClure, & Karduna, 2003). The coracoacromial arch is formed by the coracoid process, the coracoacromial ligament, and the acromion. The supraspinatus and the RC tendons lie within the SA space, defined by the coracoacromial arch the humerus (Pratt, 1994). The SA space can be measured via the acromiohumeral distance, (AHD), that is defined as the linear shortest distance between the acromion and the humerus (Desmeules, Minville, Riederer, Cote, & Fremont, 2004). It has been demonstrated that as the arm is raised, the SA space narrows (Allmann et al., 1997; Bey et al., 2007; Desmeules et al., 2004; Flatow et al., 1994; Graichen, Bonel, Stammberger, Englmeier et al., 1999), which may lead to RC impingement or tears (Azzoni & Cabitza, 2004; Azzoni, Cabitza, & Parrini, 2004; Flatow et al., 1994; Graichen, Bonel, Stammberger, Haubner et al., 1999; Solem-Bertoft, Thuomas, & Westerberg, 1993). Classic shoulder impingement syndrome has been described as involving the anterior edge and anterior third part of the undersurface of the

acromion (Graichen, Bonel, Stammberger, Haubner et al., 1999; Pappas et al., 2006; Roberts, Davila, Hushek, Tillett, & Corrigan, 2002). However, various research studies utilizing different a range of techniques to measure SA space changes have reported contrasting results, challenging these conventional views (Cholewinski, Kusz, Wojciechowski, Cielinski, & Zoladz, 2008; Desmeules et al., 2004; Wang, Lin, Pan, & Wang, 2005). This discussion explores the various research reports on SA space changes with arm elevation, and examines the importance of studying SA space changes in the anterolateral aspect of the shoulder. Further, this discussion assesses the link between poor posture and pain, and stresses the importance of researching the effects of posture on shoulder pain.

Importance of studying Acromiohumeral Distance (AHD)

The diagnosis of RC disease and shoulder impingement syndrome (SAIS) is based on each patient's history and clinical assessment. The Neer (Neer CS II, 1983) and Hawkins tests (Hawkins & Kennedy, 1980) have been described for the clinical diagnosis of RC disease. Hawkins and Kennedy proposed a test for assessing impingement at 90 degrees elevation of the arm coupled with internal rotation (Hawkins & Kennedy, 1980), while Neer's test requires full elevation of arm to 180 degrees to elicit impingement symptoms (Neer CS II, 1983). Both tests postulate that the maximum reduction in SA space occurs at or beyond 90 degrees of shoulder elevation. The kinematic study on the humeral head translations during Neer's and Hawkin's test positions on patients with shoulder impingement syndrome and on healthy subjects found no significant differences

in humeral rotations and translations between patients and controls (Hallstrom & Karrholm, 2008). However in the Hawkin's sign position, the center of the humeral head was positioned more lateral and proximal in patients than in controls. The study indicates that Hawkin's sign may be more sensitive in detecting the impingement. In order to get more definitive answers about SA space narrowing and its effect on impingement, various other researchers have used a variety of techniques to study the patterns of SA space reduction with arm elevation (Allmann et al., 1997; Brossmann et al., 1996; Flatow et al., 1994; Graichen, Bonel, Stammberger, Haubner et al., 1999; Pappas et al., 2006; Roberts et al., 2002). Brossmann et al performed a cadaver study using radioactive markers to describe the shoulder positions that caused maximum impingement on the supraspinatus tendon by the coracoacromial arch (Brossmann et al., 1996). They found that with the arm in 0 degrees abduction and 0 degrees forward flexion, the supraspinatus tendon was positioned well beyond the structures of the coracoacromial ligament. With internal rotation at 90 degrees of forward flexion, a position simulating the Hawkins Test position, the greater tuberosity of the humerus abutted the acromion. However, maximum impingement was recorded at 60 degrees forward flexion, 60 degrees abduction, and internal rotation. This study was limited by the use cadavers, and use of just three specimens. In addition, the positioning was passive; results may have been different with active elevation. Also, the authors did not quantify their results, but rather presented a qualitative description of them. These results are in agreement with another cadaveric study, where researchers using stereophotogrammetry techniques, examined the relative position of the undersurface of the acromion and the proximal humerus (Flatow et al.,

1994). The researchers found that the humerus and the undersurface of the acromion were in maximum proximity of each other in the 60 to 120 degrees range, with close contact at the supraspinatus insertion. Using 3- D MRI, Pappas et al examined the subacromial contact of the supraspinatus and the infraspinatus tendon, and found that the subacromial contact was the greatest at the Hawkins Test position (90 degrees elevation, internal rotation) (Pappas et al., 2006). So, these studies support the theory about reduction of SA space with arm elevation to higher angles. However, contradictory results are also reported in the literature.

Roberts et al reported that AHD, visualized via MRI, was smallest with the arms at the side (mean 6.4 mm), and it increased as the arms was elevated from 90 to 120 degrees (mean 7.7 -14.2 mm) (Roberts et al., 2002). These authors also found that the classic positions of impingement (Neers and Hawkins Test positions) did not decrease the space between the acromion and the RC tendons. These results are consistent with other studies that found that Neers Test positions, with the arm in full elevation, did not induce compression of the RC under the acromion (Flatow et al., 1994; Pappas et al., 2006). Using the MRI, Graichen et al examined SA space measurements in subjects in the supine position, and also found an increase in AHD at points from 60 to 120 degrees abduction in patients with SIS as well as healthy subjects (Graichen, Bonel, Stammberger, Haubner et al., 1999). Wang et al reported that SA space dimensions were higher in athletes with shoulder injuries at 90 degree elevation than at 0 degrees (Wang et al., 2005). Thus, the SA space dimensions with arm elevation remains a subject for further research, especially in patients with RC disease.

When studying patients with RC disease, results generally indicate that the AHD is smaller than that of healthy shoulders (Allmann et al., 1997; Azzoni et al., 2004; Girometti et al., 2006; Graichen, Bonel, Stammberger, Haubner et al., 1999; Hebert, Moffet, Dufour, & Moisan, 2003). A series of MRI studies comparing AHD space in patients with SAIS to AHD space in healthy subjects reported that the AHD space in patients with SAIS was smaller than those of healthy participants at 90 degrees abduction (Graichen, Bonel, Stammberger, Haubner et al., 1999); at 110 degrees abduction (Hebert et al., 2003); and at 135 degrees abduction (Allmann et al., 1997). Recent ultrasonography studies corroborates with the findings stating that SA space was smaller in patients with RC pathologies (Girometti et al., 2006) and that SA space gets smaller as the severity of RC tear increases (Azzoni et al., 2004). However there are studies which indicate the contrary.

A recent study used ultrasonography to quantify the SA space measurements in healthy patients and those with RCD. The researchers examined the SA space in patients during elevation of the arm from 0 to 45 and 0 to 60 degrees. They found progressive reduction of SA space as the arm was elevated (Desmeules et al., 2004). However, contrary to their expectations, the SA space was observed as being greater in subjects with SAIS compared to healthy subjects. The authors attribute these results to their small sample size and low statistical power. These results are in agreement with studies by Wang et al who found using ultrasonography that SA space was greater in athletes with history of shoulder injuries as compared to the control subjects (Wang et al., 2005). Cholewinski et al examined 57 patients with unilateral symptoms of impingement and

compared their results with 72 healthy shoulders and reconfirmed the findings of higher SA space in symptomatic patients (Cholewinski et al., 2008). These results support the findings of Chen et al who determined that in a fatigued state, the humeral head migrated inferiorly in healthy subjects as compared to measurements taken during a non-fatigued state -- which may explain the increased SA space (Chen, Simonian, Wickiewicz, Otis, & Warren, 1999). These results may be applied to patients with RC disease, as the function of the RC in RC disease may be similar to the induced fatigued state. These contradictions in findings about SA space changes in symptomatic patients present grounds for additional study of the SA space with arm elevation in patients with RC disease.

Therefore, with the literature presenting contradictory results in healthy and symptomatic subjects, the behavior of the SA space remains unclear beyond 90 degrees elevation. Since most patients with RCD are symptomatic during arm elevation and overhead movement (Wilk et al., 2009), SA space measurements, and the position of maximum impingement, require further investigation to quantify impingement upon arm elevation -- especially beyond 90 degrees elevation.

Importance of studying the anterolateral aspect of the shoulder

The critical zone of the RC tendon is indicted as the most susceptible to rupture which is the region of the supraspinatus tendon located at 1 cm medial to the insertion of the tendon (Neviaser & Neviaser, 1990). This location is relatively avascular, and prone to degenerative changes preceding rupture (Rathbun & Macnab, 1970). Brossman et al

found that with 30 degrees of arm abduction, the critical zone, or the vulnerable portion of the RC, began to move under the anterior third of the acromion. With further abduction of the arm to 90 degrees in a neutral position, the critical zone was found to lie completely underneath the acromion and the coracoacromial ligament. At 60 degrees forward flexion, the anterior section of the critical zone of the RC was very close to the anterior acromion and coracoacromial ligament (Brossmann et al., 1996). The results of this study are in agreement with previous studies that state that the regions of contact between the critical zone and the acromion are on the antero-inferior portion of the acromion, and occur with internal rotation of the arm (Burns & Whipple, 1993; Flatow et al., 1994). However, a few other studies report a different aspect, or no aspect, of the acromion being in contact with the RC upon arm elevation (Roberts et al., 2002; Valadie et al., 2000). One study reported that Hawkins test as well as Neers test produced contact between the medial aspect of the acromion and the RC or biceps tendon (Valadie et al., 2000). Studies on healthy subjects reported no acromial contact between the acromion and the RC at the Hawkins or Neers positions (Roberts et al., 2002) or a decrease in contact between the acromion and the RC tendon in cadavers (Edelson & Teitz, 2000). Thus, the pattern of subacromial contact with the RC tendon is still open for research using advanced technologies.

Thoracic spine posture and its relation to shoulder pain

Abnormalities in thoracic spine posture or poor posture have been described as a risk factor for upper-quadrant musculoskeletal injuries such as shoulder impingement,

nerve entrapment, and thoracic outlet syndrome (Szeto, Straker, & Raine, 2002).

Researchers have described sitting posture as a risk factor in the development of upper quadrant musculoskeletal pain (Prins, Crous, & Louw, 2008). The association between static sitting postures and neck and shoulder pain has been reported among girls (Niemi, Levoska, Kemila, Rekola, & Keinanen-Kiukaanniemi, 1996). These results concur with another study where students reported posture to be the most important contributing factor for neck and shoulder pain (Cho, Hwang, & Chen, 2003). Sitting and working in front of the computer for 1 to 4 hours has been equated with a 43% to 71% increase in neck and shoulder discomfort (Ramos, James, & Bear-Lehman, 2005), and lesser trunk movement was associated with reports of upper back and neck pain (Murphy, Buckle, & Stubbs, 2004).

In the case of shoulder pain, poor posture of the thoracic spine has been linked with a presence of pain by a number of authors and researchers (Bullock, Foster, & Wright, 2005; Greenfield et al., 1995; Griegel-Morris, Larson, Mueller-Klaus, & Oatis, 1992; Kebaetse, McClure, & Pratt, 1999; Kendall, McCreary, & Provance, 1993; Sahrmann, 2002). Evidence for this theory states that the SA space decreases with scapular protraction of the shoulder (Solem-Bertoft et al., 1993) with scapular protraction indicated as one of the components of thoracic spine kyphosis. Scapular protraction, along with downward rotation and anterior tilting of the scapula as scapular positions is theorized to be associated with poor upper quadrant posture and resulting in a reduction in SA space and leading to increase compression on rotator cuff (Michener et al., 2003). Changes in the SA space have also been found to be directly correlated with the severity

of thoracic spine kyphosis (Gumina, Di Giorgio, Postacchini, & Postacchini, 2008). Gumina et al examined 47 patients with hyperkyphosis and 175 subjects with healthy shoulders. They found that the SA space was smaller in patients with hyperkyphosis compared to healthy subjects. Patients with more severe hyperkyphosis also had significantly smaller AHD than those with less-severe kyphosis. However hyperkyphosis in this study was a lot more pronounced. Patients with more than 50 degrees of kyphotic curve were classified to have severe hyperkyphosis while those with less than 50 degrees were categorized to have less severe hyperkyphosis. Slouched posture has been linked with reduced shoulder ROM (Crawford H. J. & Jull G. A., 1993) and less muscular force production (Kebaetse et al., 1999). Reduced ROM is reported to be significantly related to disability and RC disorders (Chakravarty & Webley, 1993). These reports indicate that poor thoracic spine posture may be related to pain and hence disability in the shoulder.

Conclusion

In light of the findings reported in the current body of literature addressing SAIS and RC disease, it is imperative that future research focus on the examination of the SA space at higher levels of arm elevation, employing advanced techniques such as ultrasonography, MRI, or CT Scan, to further our understanding of shoulder mechanics. There is a need for to investigate the effectiveness of postural correction interventions such as taping, stretching, biofeedback, and exercises, and their effect on the SA space, on RC tendons, and on shoulder impairments such as pain, muscle performance and shoulder kinematics.

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VITA

Nitin Kalra was born in Meerut City, India and is a permanent resident of the United States of America. He earned his Bachelor's Degree in Physical Therapy from College of Allied Health Sciences, Manipal University, India. In 2004, after his internship in Indian Spinal Injuries Center and Hospital, New Delhi, India, he moved to the United States to attend Graduate School at Virginia Commonwealth University, Richmond, VA. Nitin resides with his wife Geetam, in Gainesville, VA and is practicing Physical Therapy in Washington D.C. and Northern Virginia.