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Mechanisms of Rotator Cuff Disease: Alterations of Scapular Kinematics on Subacromial Space

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Mechanisms of Rotator Cuff Disease: Alterations of Scapular Kinematics on Subacromial Space

A dissertation submitted in partial fulfillment of the requirements for the degree of
Doctor of Philosophy at Virginia Commonwealth University

By
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# TABLE OF CONTENTS

TABLE OF CONTENTS ...........................................................................................................................................v

LIST OF TABLES ....................................................................................................................................................ix

LIST OF FIGURES ...................................................................................................................................................x

ABSTRACT ............................................................................................................................................................xi

CHAPTER ONE: INTRODUCTION ..........................................................................................................................1

Problem and Study Significance ..........................................................................................................................1

Theoretical Framework .........................................................................................................................................2

Purpose of Research ...............................................................................................................................................5

Specific Aims and Hypotheses .............................................................................................................................6

Remaining Chapters .............................................................................................................................................7

References .............................................................................................................................................................8

CHAPTER TWO: LITERATURE REVIEW ..................................................................................................................13

Abstract ...............................................................................................................................................................13

Introduction ..........................................................................................................................................................14

Extrinsic Mechanisms .........................................................................................................................................16

*Anatomical Factors* ........................................................................................................................................18

*Biomechanical Factors* .....................................................................................................................................21
Scapular Kinematics & Influence of Posture, Muscle Deficit and Soft Tissue Tightness .................................................................21

Humeral Kinematics & Influence of Posture, Muscle Deficit and Soft Tissue Tightness .................................................................25

Internal Impingement ........................................................................................................................................................................30

Intrinsic Mechanisms ..........................................................................................................................................................................31

Age Related Tendon Degenerative Changes ..........................................................................................32

Tendon Vascularity .................................................................................................................................................................33

Impact of Alterations of Tendon Matrix on Mechanical Properties ...............................35

Tensile Tissue Overload: Inhomogeneous Mechanical Properties .................................37

Subgroups of Patients with Tendinopathy Based on Mechanisms .................................40

Factors for Subgroups of RC Tendonopathy ........................................................................41

Conclusion ....................................................................................................................................................................................42

References ....................................................................................................................................................................................43

Tables and Figures ........................................................................................................................................................................66

CHAPTER THREE: SCAPULAR UPWARD ROTATION AND POSTERIOR TILT ALTERS SUBACROMIAL SPACE, BUT NOT STRENGTH IN SUBACROMIAL IMPINGEMENT ............................................................................................................70

Abstract ......................................................................................................................................................................................70

Introduction ...............................................................................................................................................................................72

Methods .....................................................................................................................................................................................75

Subjects ....................................................................................................................................................................................75
CHAPTER FOUR: DOES SCAPULAR DYSKINESIS ALTER SCAPULAR KINEMATICS AND SUBACROMIAL SPACE WITH AND WITHOUT THE SCAPULAR ASSISTANCE TEST IN ASYMPTOMATIC INDIVIDUALS?

Abstract ......................................................................................................................114

Introduction ..............................................................................................................116

Methods ....................................................................................................................119

Participants ..............................................................................................................119
### Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Instrumentation</td>
<td>121</td>
</tr>
<tr>
<td>Testing Procedure</td>
<td>123</td>
</tr>
<tr>
<td>Data Analysis</td>
<td>124</td>
</tr>
<tr>
<td>Results</td>
<td>125</td>
</tr>
<tr>
<td>Effects of Scapular Dyskinesis</td>
<td>125</td>
</tr>
<tr>
<td>Effects of Scapular Assistance Test</td>
<td>126</td>
</tr>
<tr>
<td>Discussion</td>
<td>128</td>
</tr>
<tr>
<td>Limitations</td>
<td>131</td>
</tr>
<tr>
<td>Conclusion</td>
<td>133</td>
</tr>
<tr>
<td>Key Points</td>
<td>133</td>
</tr>
<tr>
<td>References</td>
<td>135</td>
</tr>
<tr>
<td>Tables and Figures</td>
<td>144</td>
</tr>
<tr>
<td><strong>CHAPTER FIVE: CONCLUSION OF DISSERTATION</strong></td>
<td>152</td>
</tr>
<tr>
<td>Clinical Implications</td>
<td>154</td>
</tr>
<tr>
<td>Future Research</td>
<td>155</td>
</tr>
<tr>
<td>References</td>
<td>157</td>
</tr>
</tbody>
</table>
LIST OF TABLES

1. Rotator Cuff Pathological Mechanisms ................................................................. 66

2. History and Examination Factors to Subcategorize RC Tendinopathy Based on Mechanism ........................................................................................................ 67

3. Chapter 3. Table 1. Subject Characteristics ....................................................... 103

4. Chapter 3. Table 2. Intraclass Correlation Coefficients and Minimal Detectable Change with 90% Confidence of Measures ........................................ 104

5. Chapter 3. Table 3. Scapular Upward Rotation, Posterior Tilt and Acromiohumeral Distance with and Without Scapular Assistance Test ............... 105

6. Chapter 3. Table 4. Normalized Peak Torque in External Rotation and Elevation with and without Scapular Assistance Test ........................................ 106

7. Chapter 3. Table 5. Changes with Scapular Assistance Test and Proportion of Subjects Exceeding the Minimal Detectable Change ......................... 107

8. Chapter 4. Table 1. Characteristics of Participants Classified with Obvious Dyskinesis and Normal Scapulothoracic Motion using the Scapular Dyskinesis Test ................................................................. 144

9. Chapter 4. Table 2. Three Dimensional Scapular Kinematics and Acromio-Humeral Distance in Participants with and Without Obvious Scapular Dyskinesis .. 145

10. Chapter 4. Table 3. Summary of Results of 3-factor Mixed-model ANOVAs for Scapular kinematics and Acromiohumeral distance ........................................ 146
LIST OF FIGURES

1. Chapter 2, Figure 1. Intrinsic and Extrinsic Mechanisms of Rotator Cuff Tendinopathy ..............................................................................................................................................68

2. Chapter 2, Figure 2. Superior Migration of the Humerus and Diminished Subacromial Space in Patient with a Chronic Large RC tear. ..............................................69

3. Chapter 3, Figure 1. Modified Scapular Assistance Test ........................................108

4. Chapter 3, Figure 2. A Linear Measure of the Subacromial Space at the Anterior Outlet, the Acromiohumeral Distance, on Ultrasound Image .........................109

5. Chapter 3, Figure 3. Mean Scapular Upward Rotation with and Without Scapular Assistance in Subjects with Subacromial Impingement Syndrome and Control Subjects. ................................................110

6. Chapter 3, Figure 4. Mean Scapular Posterior Tilt with and Without Scapular Assistance in Subjects with Subacromial Impingement and Control Subjects. ........................................................................111

7. Chapter 3, Figure 5. Mean Acromiohumeral Distance with and Without Scapular Assistance Test in Subjects with Subacromial Impingement Syndrome and Control Subjects .........................................................112

8. Chapter 3, Figure 6. External Rotation and Elevation Strength in Subjects with Subacromial Impingement and Control subjects ........................................113

9. Chapter 4, Figure 1. Participant with Obvious Scapular Dyskinesis ........................147

10. Chapter 4, Figure 2. Mean Scapular kinematics and Acromiohumeral Distance in Individuals with Normal Scapular Motion and Obvious Scapular Dyskinesis ........................................................................................................148

11. Chapter 4, Figure 3. Mean Scapular Kinematics with and without Passive Alteration with the Scapular Assistance Test in Individuals with and without Obvious Scapular Dyskinesis ................................................................................149

12. Chapter 4, Figure 4. Acromiohumeral Distance with and without Passive Manual Correction with the Scapular Assistance Test ........................................151
ABSTRACT

MECHANISMS OF ROTATOR CUFF DISEASE: SCAPULAR ALTERATIONS ON SUBACROMIAL SPACE

By Amee L. Seitz, Ph.D.

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University.

Virginia Commonwealth University, 2010.

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

Rotator cuff disease is multi-factored and has been attributed to both intrinsic and extrinsic factors. Extrinsic factors contribute to compression of the rotator cuff tendons. Intrinsic factors that contribute to rotator cuff tendon degradation with tensile/shear overload include alterations in biology, mechanical properties, morphology, and vascularity. Subacromial impingement is related to factors that encroach upon the subacromial space, while internal impingement affects the articular side of the tendons adjacent to glenoid. While the mechanisms of impingement are varied, further research is necessary to improve treatment and patient outcomes. Chapter 2 is a thorough review of literature on the mechanisms of rotator cuff disease.

Alterations in scapular kinematics may influence subacromial space and either contribute to the etiology of subacromial impingement with rotator cuff tendon
compression or serve as a compensation to alleviate compression. Furthermore alterations in scapular position may directly influence rotator cuff muscle strength. Chapter 3 compares the influence of the scapular assistance test on scapular upward rotation, posterior tilt, subacromial space, and shoulder strength between healthy individuals and subjects with subacromial impingement syndrome. Scapular upward rotation and posterior tilt induced with scapular assistance test appears to influence subacromial space, but not shoulder muscle strength; however, the influence of these scapular rotations do not differ between asymptomatic individuals and those with subacromial impingement. Furthermore scapular posterior tilt appears to have a greater influence on increasing subacromial space and should be emphasized in the treatment of individuals with subacromial impingement.

In chapter 4, we examine the influence that obvious scapular dyskinesis and passive scapular correction with the scapular assistance test have on 3D scapular kinematics and subacromial space. Scapular dyskinesis did not alter scapular kinematics or acromiohumeral distance during active elevation in static positions, in the scapular plane, and without a load when compared to those without scapular dyskinesis. This suggests other contributing factors, such as pain, increased load, or fatigue is requisite to alterations in scapular kinematics or AHD. Passive correction with the scapular assistance test increased scapular upward rotation, posterior tilt, and subacromial space in individuals with and without dyskinesis. In patients with obvious dyskinesis, there was a greater increase in scapular upward rotation with passive scapular assistance. This increased scapular upward rotation had a negative relationship with change in the
acromiohumeral distance. The scapular dyskinesis test increased acromiohumeral distance and therefore may be helpful identifying individuals where subacromial compression is producing symptoms, regardless of dyskinesis.

The results of this research suggest scapular kinematics and subacromial space are altered with the passive maneuver of the scapular assistance test in all individuals, regardless of subacromial impingement syndrome or scapular dyskinesis. Scapular dyskinesis alone may not be detrimental to scapular position and subacromial space when evaluated in static positions of active arm elevation. Other potential factors may be required to alter scapular kinematics to reduce subacromial space including pain, dynamic movement, load or fatigue. Further study is necessary to determine the influence of the combination of these factors in individuals with scapular dyskinesis.
CHAPTER 1: INTRODUCTION

The rotator cuff (RC) is comprised of four muscles that originate on the scapula and attach to the humerus. The RC tendons which attach the muscles to the head of the humerus, traverse beneath the bony arch of the acromion process of the scapula to insert on the humerus. The subacromial space is the anatomical region between the acromion of the scapula and the head of the humerus that contains the RC tendons. The RC functions to center the humeral head on the glenoid of the scapula and opposes the superior force of the larger deltoid muscle. (Halder, Zhao, Odriscoll, Morrey, & An, 2001)

Problem and Study Significance

RC disease is a continuum of three stages of pathology beginning with subacromial impingement, progressing to tendinosis with partial thickness tears, and eventually resulting in complete tendon rupture. (Neer, 1983) The prevalence of RC disease increases with age, primarily affecting those over the age of 40 years with over 50% of individuals over the age of 60 years. (Milgrom, Schaffler, Gilbert, & van Holsbeeck, 1995; Sher, Uribe, Posada, Murphy, & Zlatkin, 1995) RC disease accounts for the majority, 29-70%, of all shoulder disorders. (Chard, Hazleman, Hazleman, King, & Reiss, 1991; van der Windt, Koes, de Jong, & Bouter, 1995; Vecchio, Kavanagh, Hazleman, & King, 1995) RC disease causes pain and disability (Bartolozzi, Andreychik, & Ahmad, 1994; Duckworth, Smith, Campbell, & Matsen, 1999; MacDermid, Ramos, Drosdowech, Faber, & Patterson, 2004; Smith et al., 2000), and has a significant impact
on health-related quality of life. (MacDermid et al., 2004) The economic impact of caring for shoulder injuries in the United States is sizable, with estimates of over $7 billion dollars in direct US healthcare expenditures in the year 2000. (Johnson, Crossley, O'Neil, & Al-Zakwani, 2004) These costs are expected to escalate as the overall number of individuals with RC disease is expected to grow coincident with an aging population. (Gomoll, Katz, Warner, & Millett, 2004)

Randomized trials suggest the short and long-term outcomes of patients with RC disease treated with surgery are comparable to conservative treatment. (Brox et al., 1999; Brox, Staff, Ljunggren, & Brevik, 1993; Haahr & Andersen, 2006; Haahr et al., 2005) Unfortunately regardless of treatment, more than a third of patients (38%) do not have a successful outcome with persistent pain and disability. (Brox et al., 1999; Brox et al., 1993) Given the high prevalence and progressive nature, research is needed to better understand the mechanisms of RC disease to improve effectiveness of treatment, guide specific treatment choices, and better prognosticate the outcomes of various treatments for patients with RC disease.

Theoretical Framework

Studies have shown a decrease in subacromial space with active arm elevation in patients with RC disease, specifically subacromial impingement, when compared to healthy/uninvolved shoulders. (Graichen et al., 1999; Hebert, Moffet, Dufour, & Moisan, 2003) However, it is unknown whether subacromial space narrowing is due to altered kinematics of the scapula that move the acromion inferiorly contributing to extrinsic compression of the tendons or due to superior translation of the humerus attributed to
deficits intrinsic to the rotator cuff. Altered 3-dimensional scapular kinematics have been identified in patients with RC disease, with the majority of evidence demonstrating patients with subacromial impingement have decreased scapular upward rotation (Endo, Ikata, Katoh, & Takeda, 2001; Ludewig & Cook, 2000; Su, Johnson, Gracely, & Karduna, 2004) and posterior tilt (Endo et al., 2001; Ludewig & Cook, 2000; Lukasiewicz, McClure, Michener, Pratt, & Sennett, 1999) compared to healthy subjects. Decreased scapular posterior tilt and upward rotation has been theorized to contribute to extrinsic impingement of the RC tendons by decreasing the subacromial space (Ludewig & Cook, 2000) However, no study has directly or concurrently examined this theory.

Alternatively, increased scapular upward rotation and posterior tilt has been theorized to increase the subacromial space (P. W. McClure, Michener, & Karduna, 2006) Strategies to promote scapular posterior tilt with thoracic postural retraining (Kendall, McCreary, & Provance, 1993), scapular taping (Lewis, Wright, & Green, 2005; Selkowitz, Chaney, Stuckey, & Vlad, 2007), stretching and strengthening (Ludewig & Borstad, 2003) have been employed for the treatment of patients with subacromial impingement. There is evidence that limiting upward rotation and posterior tilt decreases subacromial space (Atalar et al., 2009); however, there is no evidence that inducing a position of increased scapular upward rotation and posterior tilt alters subacromial space in either healthy subjects or patients with subacromial impingement. Further study is necessary to determine if alterations of the scapula into scapular upward rotation and posterior tilt can induce a change in subacromial space in either patients with subacromial impingement or healthy subjects.
Despite substantial evidence that identifies alterations in scapular kinematics in patients with subacromial impingement compared to healthy subjects (Endo et al., 2001; Graichen et al., 2001; Hebert, Moffet, McFadyen, & Dionne, 2002; Laudner, Myers, Pasquale, Bradley, & Lephart, 2006; Lin et al., 2005; Ludewig & Cook, 2000; Lukasiewicz et al., 1999; P. W. McClure et al., 2006; Mell et al., 2005; Warner, Micheli, Arslanian, Kennedy, & Kennedy, 1992), the magnitude of group differences in these studies have been small. Graichen et al. identified a subset of patients with subacromial impingement who demonstrate significant scapular kinematic alterations, defined as those that exceed 2 standard deviations from the mean.(Graichen et al., 2001) This suggests that not all patients with subacromial impingement have altered scapular kinematics, but likely only a subset has significant abnormal scapular kinematics that may benefit from a distinct intervention.

A clinical observation method of the scapular dyskinesis test has been validated as a useful and reliable method to identify obvious aberrant movement of the scapula, known as scapular dyskinesis.(P. McClure, Tate, Kareha, Irwin, & Zlupko, 2009; Tate, McClure, Kareha, Irwin, & Barbe, 2009) Using this scapular dyskinesis test, subjects identified as having obvious scapular dyskinesis were found to have less scapular upward rotation and posterior tilt with active arm elevation compared to subjects without dyskinesis. It is unknown if active alterations in scapular motion, or clinically identified obvious scapular dyskinesis, contribute to decreased subacromial space which is theorized to be the underlying etiology of subacromial impingement. Further study is
warranted to determine if active alterations, specifically obvious dyskinesis as defined by the scapular dyskinesis test, can affect the subacromial space.

**Purpose of Research**

This research proposes to examine the mechanisms of RC disease, by determining the influence that both passive and active alterations in scapular kinematics have on subacromial space, and the potential explanatory factor of RC strength. Specifically, to determine the effect of specific 3-dimensional (3D) passive alterations in scapular position on subacromial space as measured by acromiohumeral distance (AHD) and RC muscle strength in 20 subjects with subacromial impingement and 20 healthy subjects, matched by age, gender and hand dominance. The scapula will be passively altered with the manual technique of the Scapular Assistance Test (SAT) (Kibler, 1998; Rabin, Irrgang, Fitzgerald, & Eubanks, 2006). We will determine if the influence that passive alteration with the SAT has on AHD and 3D scapular position differs in subjects with subacromial impingement compared to healthy subjects. We will also determine if there are associations among change in 3D position of the scapula, AHD, and rotator cuff strength with the SAT within each group and the entire cohort. Second, the purpose is to examine the effect of active alterations of the scapula on AHD, by comparing AHD and 3D scapular kinematics between 20 healthy subjects with obvious scapular dyskinesis and 20 healthy subjects without obvious dyskinesis as determined by the Scapular Dyskinesis Test.
Specific Aims

**Specific Aim 1:** evaluate within and between group changes in AHD and 3D scapular position with artificially induced passive alteration using the SAT at 0°, 45° and 90° degrees of scapular elevation in 20 patients with subacromial impingement and 20 healthy subjects.

**Hypotheses:**
1.a. Within both subject cohorts, the SAT will increase AHD with active humeral elevation at 45° and 90° degrees when compared to AHD during arm elevation without the SAT. 1.b. There will be no differences between healthy subjects and subjects with subacromial impingement in the amount of change in AHD that is induced with the SAT with active arm elevation at 45° and 90°. 1.c At 45 and 90 degrees of arm elevation, the SAT will increase upward rotation or posterior tilt in both subject cohorts.

**Specific Aim 2:** examine the association between: a) the change in 3D position of the scapula and AHD with passive alteration, and b) change in AHD and shoulder isometric strength with the SAT in healthy subjects and subjects with subacromial impingement.

**Hypotheses:**
2.a There will be a positive linear relationship between change in AHD and change in scapular upward rotation and posterior tilt with passive alteration of the scapula induced with the SAT. 2.b. There will be a positive linear relationship between change in AHD with SAT and change in strength of the rotator cuff.

**Specific Aim 3:** To examine the effect of active alterations of the scapula, we will compare AHD and 3D scapular kinematics with 0°, 45° and 90° active arm elevation
between 20 healthy subjects with obvious scapular dyskinesis as determined by the Scapular Dyskinesis Test and 20 healthy subjects without dyskinesis (normal).

**Hypothesis:** Subjects with obvious dyskinesis will demonstrate decreased AHD concurrent with decreased upward rotation/posterior tilt 3D scapular kinematics with active arm elevation compared to subjects without dyskinesis.

Results of this study will further elucidate the mechanisms of RC disease, specifically subacromial impingement, by examining the effect of scapular passive and active alterations on subacromial space and RC strength. This will begin to identify subgroups of patients based on mechanisms of passive and active altered scapular kinematics and their influence on subacromial space. This information can be used to improve effectiveness of treatment, guide specific treatment choices, and better prognosticate the outcomes of various treatments for patients with subacromial impingement.

**Remaining Chapters**

The remaining chapters contain a review of literature, two chapters with the results of the experimental research and a concluding chapter. Chapter two is a thorough review of literature on the extrinsic and intrinsic mechanisms of RC tendinopathy. Chapter three is the experimental study which addresses the first two specific aims of the dissertation. Chapter four is comprised of the manuscript which addresses the last specific aim. Chapter five is a concluding summary chapter which summarizes the results of the experimental studies, clinical implications, and future study.
References


CHAPTER 2: LITERATURE REVIEW

Mechanisms of rotator cuff tendinopathy: Intrinsc, extrinsic, or both?

Accepted for Publication in Clinical Biomechanics

Abstract

The etiology of rotator cuff tendinopathy is multi-factorial, and has been attributed to both extrinsic and intrinsic mechanisms. Extrinsic factors that encroach upon the subacromial space and contribute to bursal side compression of the rotator cuff tendons include anatomical variants of the acromion, alterations in scapular or humeral kinematics, postural abnormalities, rotator cuff and scapular muscle performance deficits, and decreased extensibility of pectoralis minor or posterior shoulder. A unique extrinsic mechanism, internal impingement, is attributed to compression of the posterior articular surface of the tendons between the humeral head and glenoid and is not related to subacromial space narrowing. Intrinsic factors that contribute to rotator cuff tendon degradation with tensile/shear overload include alterations in biology, mechanical properties, morphology, and vascularity. The varied nature of these mechanisms indicates that rotator cuff tendinopathy is not a homogenous entity, and thus may require different treatment interventions. Treatment aimed at addressing mechanistic factors appears to be beneficial for patients with rotator cuff tendinopathy, however, not for all patients.
Classification of rotator cuff tendinopathy into subgroups based on underlying mechanism may improve treatment outcomes.

**Introduction**

Disorders of the rotator cuff (RC) or associated tissues are the most common problem of the shoulder (Chard et al., 1991, Van Der Windt et al., 1995, Vecchio et al., 1995). The prevalence of RC disease, specifically partial and full thickness RC tendon tears, has been shown to increase as a function of age starting at 40 years (Iannotti et al., 1991, Milgrom et al., 1995, Sher et al., 1995, Tempelhof et al., 1999), and to exceed as much as 50% by the age of 60 years (Milgrom et al., 1995, Sher et al., 1995). Furthermore, RC disease contributes to pain and disability (Macdermid et al., 2004, Bartolozzi et al., 1994, Duckworth et al., 1999, Smith et al., 2000), and has an impact on health-related quality of life. (Macdermid et al., 2004) Randomized trials suggest the short and long-term outcomes of patients with RC tendinopathy treated with surgery are comparable to conservative treatment that includes exercise or exercise combined with a multimodal rehabilitation program. (Brox et al., 1999, Brox et al., 1993, Haahr and Andersen, 2006, Haahr et al., 2005) However, regardless of treatment, more than a third of patients do not have a successful outcome with continued persistent pain and disability. (Brox et al., 1999, Brox et al., 1993) Given the high prevalence and continued pain despite treatment in patients with RC disease, research to better understand the mechanisms to improve effectiveness of treatment, guide specific treatment choices, and better prognosticate treatment outcomes is necessary.
RC disease has been classically described as a progressive disorder of the RC tendons which begins with an acute tendinitis, progresses to tendinosis with degeneration and partial thickness tears, and results in full thickness rupture. (Neer, 1983) The diagnostic terms of RC tendinitis and tendinosis represent tendon pathology subsets of RC tendinopathy. RC tendinitis is often used to define both acute and chronic pain associated with, by definition, inflammation. However, histological studies of patients with RC disease have found minimal to no inflammatory cells in the RC tendons (Fukuda et al., 1990) and subacromial bursa (Sarkar and Uhthoff, 1983). Tendinosis is the diagnostic label for tendon pathology that is degenerative with or without inflammation. In contrast, RC tendinopathy is used to signify a combination of pain and impaired performance associated with RC tendons. (Alfredson, 2003, Alfredson, 2005) Tendinopathy is preferred term, to indicate a clinical diagnosis without knowing the specific underlying mechanism or tendon pathology. (Fredberg and Stengaard-Pedersen, 2008, Almekinders, 1998) The focus of this review is RC tendinopathy which includes external or internal impingement, tendinitis, tendinosis with degeneration and partial thickness tendon tears. Full thickness tendon tears are unique and beyond the scope of this review.

Mechanisms of RC tendinopathy have been classically described as extrinsic, intrinsic or a combination of both. Extrinsic factors are defined as those causing compression of the RC tendons, while intrinsic mechanisms are those associated with degeneration of the RC tendon. Neer proposed an extrinsic mechanism to the etiology RC tendinopathy with compression of the RC tendons and associated tissues within the
subacromial space under the anterior aspect of the acromion or surrounding structures (Neer, 1972) and coined this subacromial impingement syndrome (Neer, 1983). The diagnosis of “subacromial impingement” inherently implies an extrinsic compression mechanism due to narrowing of the subacromial space, which may not accurately represent all RC tendon pathology. A unique extrinsic mechanism, internal impingement, has been described particularly in overhead athletes (Burkhart et al., 2003, Jobe, 1995, Kibler, 1998, Kvitne and Jobe, 1993). Internal impingement occurs due to compression of the articular-side rather than the bursal side of the RC tendons, between the posterior superior glenoid rim and humerus when the arm is in full external rotation, abduction, and extension (Davidson et al., 1995, Edelson and Teitz, 2000). Although internal impingement can be considered an extrinsic mechanism, narrowing of the subacromial space is not a hallmark finding. In contrast to extrinsic mechanisms of RC tendinopathy, Codman postulated an intrinsic mechanism due to degeneration within the tendon (Codman and Akerson, 1931), confounded by aging (Iannotti et al., 1991, Milgrom et al., 1995, Sher et al., 1995, Tempelhof et al., 1999). Figure 1 illustrates the mechanisms and the relationships of the varied mechanisms of RC tendinopathy. Despite the debate over the pathogenesis, evidence indicates the etiology of RC tendinopathy is multi-factorial and likely both intrinsic and extrinsic mechanisms play a role. (Table 1.)

**EXTRINSIC MECHANISMS OF ROTATOR CUFF TENDINOPATHY**

Extrinsic mechanisms of RC tendinopathy that result in bursal sided RC tendon compression due to narrowing of the subacromial space include anatomical factors, biomechanical factors, or a combination. The subacromial space is the interval between
the coracoacromial arch, anterior acromion and the humeral head (Neer and Poppen, 1987). The acromiohumeral distance (AHD), a linear measure between the acromion and the humeral head used to quantify the subacromial space, has been studied in patients with RC disease using magnetic resonance imaging (MRI) (Graichen et al., 1999, Hebert et al., 2003, Saupe et al., 2006), ultrasonography (Azzoni and Cabitza, 2004, Azzoni et al., 2004, Cholewinski et al., 2007, Desmeules et al., 2004) and radiographs (Norwood et al., 1989, Nove-Josserand et al., 2005, Petersson and Redlund-Johnell, 1984, Saupe et al., 2006, Weiner and Macnab, 1970). AHD is normally between 7 and 14 mm in healthy shoulders, but is reduced in those with RC tendon tears (Azzoni and Cabitza, 2004, Azzoni et al., 2004, Ellman et al., 1986, Golding, 1962, Weiner and Macnab, 1970). Furthermore, AHD less than 7 mm with the arm at rest is a predictor indicator of less favorable surgical outcome. (Walch et al., 1992, Weiner and Macnab, 1970, Ellman et al., 1986, Norwood et al., 1989). However, patients with RC tendinopathy do not consistently present with significant deficits in subacromial space narrowing with the arm at rest. (Azzoni and Cabitza, 2004, Desmeules et al., 2004) Only measures of subacromial space taken with muscle activation are useful to detect deficits related to biomechanical factors that “functionally” narrow the subacromial space. (Graichen et al., 1999) In a series of MRI studies, AHD during active arm elevation was smaller in subjects with RC tendinopathy compared to healthy shoulders. (Allmann et al., 1997, Graichen et al., 1999, Hebert et al., 2003) Limited evidence suggests changes in the subacromial space linear distance or extent of narrowing with arm elevation is a sensitive marker of RC tendinopathy (Cholewinski et al., 2007), and may predict the outcome of
rehabilitation (Desmeules et al., 2004). Further research that examines changes in
subacromial space with active arm elevation in patients with RC tendinopathy is
advocated and may be useful to identify the presence of an extrinsic mechanism
influencing the articular side of the RC tendons.

Anatomical Factors

Anatomical factors that may excessively narrow the subacromial space and outlet
to the RC tendons include variations in shape of the acromion (Bigliani et al., 1991,
Epstein et al., 1993, Gill et al., 2002, Ogawa et al., 2005), orientation of the slope/angle
of the acromion (Toivonen et al., 1995, Aoki et al., 1986, Edelson, 1995, Vaz et al., 2000)
or prominent osseous changes to the inferior aspect of the acromio-clavicular (AC) joint
or coracoacromial ligament (Ogawa et al., 2005, Nicholson et al., 1996, Farley et al.,
1994). Bigliani et al described the role of the shape of the acromion as an extrinsic
mechanism of RC tendinopathy by describing the morphologic condition of the acromion
as a Type I (flat), Type II (curved), or Type III (hooked). (Bigliani et al., 1986) An
association between acromion shape and severity of RC pathology has been well
documented (Bigliani et al., 1991, Epstein et al., 1993, Gill et al., 2002, Ogawa et al.,
2005) with trends of a greater prevalence of Type III, or hooked acromion in patients
with impingement (Epstein et al., 1993) and full thickness RC tears (Bigliani et al., 1991,
Gill et al., 2002, Toivonen et al., 1995, Epstein et al., 1993). Success of conservative
treatment for patients with RC tendinopathy has been related to shape/type of acromion;
Morrison et al found better outcomes in patients with type I acromions. (Morrison et al.,
1997) These findings were similar to those of Wang et al, who found 89% of patients
with type I acromion had a successful response, 73% with type II, and 58.3% of type III. (Wang et al., 2000) Whether acromial shape is congenital (Nicholson et al., 1996) or acquired with age (Wang and Shapiro, 1997, Bonsell et al., 2000, Edelson, 1995, Speer et al., 2001) remains controversial. Moreover, the acromial shape classification has been questioned because of poor interobserver reliability. (Jacobson et al., 1995, Zuckerman et al., 1997)

Measurement of the slope or angle of the acromion is another method to capture the acromial shape, and both have been proposed to cause RC tendon compression (Toivonen et al., 1995, Aoki et al., 1986, Edelson, 1995, Vaz et al., 2000). A flatter slope or more horizontal position of the acromion is associated with subacromial impingement (Edelson, 1995), degenerative changes of the RC (Toivonen et al., 1995, Aoki et al., 1986), subacromial spur formation (Toivonen et al., 1995, Aoki et al., 1986), and a greater loss of function in patients with tendinopathy (Vaz et al., 2000). Similarly, other anatomical factors like large subacromial spurs, thickening or ossification of the attachment of the coracoacromial ligament (CAL) are associated with RC pathology with bursal-sided partial thickness tears (Ogawa et al., 2005) and progression to full thickness RC tears (Ogawa et al., 2005, Nicholson et al., 1996, Farley et al., 1994); however, these same osseous changes in CAL have also been documented with age (Edelson, 1995).

Arthritic changes of the AC joint have also been theorized to contribute to external mechanical impingement of the RC tendons. (Neer, 1972, Neer, 1983, Petersson and Gentz, 1983) The AC joint undergoes radiographic degeneration with age including narrowing of the joint space and development of osteophytes at the distal clavicle and

There is substantial evidence that anatomical variants such as subacromial spurs, AC joint spurs, and acromial shape may contribute biomechanically to an extrinsic mechanism of RC tendinopathy and progressive RC disease; however, the presence of these alone may be insufficient to result in RC tendinopathy. Soslowsky et al(Soslowsky et al., 2002) found external mechanical compression of RC tendons in rats exposed to normal cage activity did not cause pathological changes, but combined with overuse activity had a significant effect on tendon injury. Therefore, bony anatomy such as a hooked acromion may not necessarily cause, but predispose an individual to RC tendinopathy. Supporting this theory of a requisite overuse exposure, symptomatic RC disease is more often present in dominant than non-dominant shoulders (Yamaguchi et al., 2006).

**Biomechanical Factors**

Biomechanical factors that can lead to extrinsic mechanical RC tendon compression include abnormal scapular and humeral kinematics, postural abnormalities, rotator cuff and scapular muscle performance deficits, and decreased extensibility of pectoralis minor or posterior shoulder tissues. Scapular and humeral kinematic abnormalities can cause dynamic narrowing of the subacromial space leading to RC tendon compression secondary to superior translation of the humeral head(Deutsch et al.,
or aberrant scapular motion that causes the acromion to move inferiorly (Ludewig and Cook, 2000). Postural abnormalities, muscle deficits, and soft tissue tightness factors as external mechanisms can directly influence scapular and humeral kinematics.

**Scapular Kinematics & Influence of Posture, Muscle Deficit and Soft Tissue Tightness**

Scapular kinematic abnormalities have been identified in patients with RC tendinopathy compared to healthy individuals. (Endo et al., 2001, Graichen et al., 2001, Hebert et al., 2002, Ludewig and Cook, 2000, Lukasiewicz et al., 1999, Mcclure et al., 2006, Warner et al., 1992) Subjects with subacromial impingement generally have decreased scapular posterior tilting (Endo et al., 2001, Ludewig and Cook, 2000, Lukasiewicz et al., 1999), decreased upward rotation (Endo et al., 2001, Ludewig and Cook, 2000, Su et al., 2004), and increased internal rotation (Endo et al., 2001, Hebert et al., 2002, Ludewig and Cook, 2000, Warner et al., 1992) compared to healthy subjects. As a result, the anterior aspect of the acromion may fail to move away from the humeral head during arm elevation and in theory contribute to a reduction of subacromial space and external RC compression. (Ludewig and Cook, 2000) The anterior aspect of the acromion has been identified as the predominant site of RC compression or impingement. (Flatow et al., 1994, Neer, 1972, Neer, 1983, Lee et al., 2001, Yamamoto et al., 2009, Brossmann et al., 1996) In contrast, increased scapular posterior tilting, upward rotation, and superior translation of the scapula have also been identified in patients with RC tendinopathy compared to asymptomatic subjects. (Lukasiewicz et al.,
These aberrant patterns are theorized to be a favorable compensatory response to relieve compression of the RC tendons by increasing subacromial space. While variable patterns of abnormal scapular kinematics in patients with RC tendinopathy have emerged, the differences between groups are small in magnitude which casts doubt upon the significance of these findings related to changes in subacromial space and role of abnormal scapular kinematics as an extrinsic mechanism for all patients with RC tendinopathy.

Interestingly, Graichen et al suggest that not all patients with RC tendinopathy have altered scapular kinematics, but a subset exists with significant alterations that are greater than 2 standard deviations from the mean of healthy individuals. Moreover, patients with scapular alterations classified with obvious scapular dyskinesis compared to less obvious, or subtle, alternations may have meaningful abnormal scapular kinematics that impacts the subacromial space and contribute to an extrinsic mechanism of RC tendinopathy. Silva et al found a greater reduction in subacromial space in elite tennis players with scapular dyskinesis compared to players without dyskinesis; however, the clinical method used to identify scapular dyskinesis and associated reliability was not reported.

While there is evidence of abnormal scapular kinematics in a subset of patients with RC tendinopathy, the influence of these specific biomechanical alterations on subacromial space remains speculative. Alternatively, passive alterations in scapular position may influence subacromial space. In a study by Atalar et al, limiting scapular motion by externally binding the scapular
down to the thorax while the arm is positioned at 90° compared to unrestricted scapula caused a reduction in subacromial space in healthy individuals.(Atalar et al., 2009) In study by Solem-Bertoft, positioning the scapula of 4 healthy individuals in protraction compared to retraction with sandbags reduced subacromial space.(Solem-Bertoft et al., 1993) In contrast to these findings, cadaveric study by Karduna et al found inducing scapular upward rotation from a neutral position reduced subacromial clearance(Karduna et al., 2005). Further research is necessary to determine which scapular kinematic alterations are most related to changes in subacromial space and the magnitude of change in scapular kinematics needed to affect the subacromial space.

The mechanisms responsible for scapular alterations found in subjects with RC tendinopathy have not been clearly defined, but have been theorized to include adaptive shortening of the pectoralis minor muscle(Borstad, 2006, Hebert et al., 2002, Kendall et al., 1993, Ludewig and Cook, 2000), posterior shoulder tightness(Borich et al., 2006), aberrant scapular and rotator cuff muscle performance(Ludewig and Cook, 2000), and an increase in thoracic spine flexion or kyphosis(Kebaetse et al., 1999, Wang et al., 1999, Ludewig and Cook, 2000). Subjects with a relatively shorter pectoralis minor muscle length at rest demonstrate increased scapular internal rotation during arm elevation and decreased scapular posterior tilting at higher arm elevation angles (90° and 120°) when compared with subjects with a relatively longer pectoralis minor muscle length at rest.(Borstad and Ludewig, 2005) Similarly, overhead athletes with a loss of glenohumeral internal rotation of 20% or more as compared to their opposite shoulder demonstrate increased scapular anterior tilt at end range glenohumeral internal rotation
with the arm abducted or flexed to 90 degrees. (Borich et al., 2006) Scapular alterations associated with shortened pectoralis minor length and glenohumeral internal rotation deficit are consistent with previous studies of subjects with RC tendinopathy (Endo et al., 2001, Hebert et al., 2002, Ludewig and Cook, 2000, Warner et al., 1992). The relationship between pectoralis minor muscle length at rest has been indirectly linked to pain and functional limitations attributed to RC tendinopathy via alterations in scapular kinematics (Borstad and Ludewig, 2005). The extent of pectoralis minor shortening needed to decrease the subacromial space and contribute to an extrinsic mechanism has yet to be determined.

Aberrant scapular muscle activity has been identified in patients with RC tendinopathy (Cools et al., 2003, Cools et al., 2004, Cools et al., 2005, Ruwe et al., 1994, Wadsworth and Bullock-Saxton, 1997, Moraes et al., 2008, Cools et al., 2007, Ludewig and Cook, 2000, Diederichsen et al., 2008) and been directly linked to abnormal scapular kinematics in patients with RC tendinopathy (Ludewig and Cook, 2000). Of particular interest are the relative contributions of the upper and lower serratus anterior muscles and trapezius muscles, found to stabilize the scapula and induce scapular upward rotation, external rotation, and/or posterior tilt (Johnson and Pandyan, 2005, Kronberg et al., 1990, Bagg and Forrest, 1988) to potentially allow the humeral head to clear the acromion with elevation (Mcquade et al., 1998). Individuals with RC tendinopathy have decreased muscle performance of the serratus anterior in terms of force output (Cools et al., 2004), muscle balance/ratios (Cools et al., 2004), electromyographical (EMG) activity (Ludewig and Cook, 2000, Diederichsen et al., 2008), and latencies in activation (Moraes et al.,
2008, Wadsworth and Bullock-Saxton, 1997). Similar deficits have been found in the lower trapezius muscle including increased latencies of muscle onset (Cools et al., 2003) and alterations in maximal EMG activity (Cools et al., 2007, Cools et al., 2004, Ludewig and Cook, 2000, Diederichsen et al., 2008). Relatively small changes in the muscle performance of the scapulothoracic muscles can alter the position of the scapula at a fixed angle of humeral elevation and, in theory, affect the length-tension relationship (point on the length-tension curve) of the RC muscles and the subacromial space.

Thoracic spine kyphosis posture has been directly linked to alterations in subacromial space (Gumina et al., 2008), alterations in scapular kinematics (Finley and Lee, 2003), and thus theorized to contribute to an extrinsic mechanism of RC tendinopathy. An increase in thoracic spine kyphosis/ flexion is associated with a decrease in subacromial space (Gumina et al., 2008) and a decrease in scapular posterior tilt (Kebaetse et al., 1999, Finley and Lee, 2003). These alterations in scapular kinematics are consistent with those found in patients with RC tendinopathy (Endo et al., 2001, Ludewig and Cook, 2000, Lukasiewicz et al., 1999)

**Humeral Kinematics & Influence of Posture Muscle Deficits, and Soft Tissue Tightness**

Excessive humeral head migration proximally on the glenoid is theorized to reduce subacromial space and contribute to RC tendon compression. Proximal, or superior, humeral migration and reduction of subacromial space have been used synonymously at times; however, the amount of superior displacement of the humeral head has not been correlated with linear measures or the 3D volume of the subacromial
space and may not occur at a 1:1 ratio. (Refer to Figure 2) This distinction may be futile in patients with a large RC tendon tears who present dramatic excessive proximal humeral migration with the arm at rest (Keener et al., 2009); however, in patients with RC tendinopathy the changes in subacromial space may only be apparent with active movement. (Graichen et al., 2001) The extent of subacromial space narrowing that occurs with superior humeral head translation on the glenoid may be counteracted with scapular rotation that moves the acromion superiority or posterior which may increase the subacromial space. Furthermore, a combination of aberrant humeral and scapular kinematics could cause a clinically meaningful reduction of the subacromial space. This relationship requires further study.

Proximal migration of the humerus on the glenoid while the arm is at rest is regarded as a sign of advanced RC disease (Bezer et al., 2005, Keener et al., 2009, Norwood et al., 1989, Yamaguchi et al., 2000), and attributed to chronically diminished RC performance to counteract the superior pull of the deltoid (Deutsch et al., 1996). Similar to subacromial space, patients with RC tendinopathy do not exhibit proximal humeral migration on the glenoid with the arm at rest; but rather demonstrate excessive superior- anterior translations of the humeral head with active arm elevation (Deutsch et al., 1996, Hallstrom and Karrholm, 2006, Keener et al., 2009, Ludewig and Cook, 2002, Paletta et al., 1997, Royer et al., 2009). Patients with RC tendinopathy have presented with a 1.0-1.5 mm greater superior translation (Deutsch et al., 1996, Hallstrom and Karrholm, 2006, Yamaguchi et al., 2000) and 3 mm of greater anterior translation (Ludewig and Cook, 2002) with active arm elevation compared to
asymptomatic subjects. Biomechanical mechanisms for excessive proximal humeral migration in patients with RC tendinopathy include shortening of the posterior-inferior glenohumeral joint capsule and decreased RC muscle performance.

Decreased posterior capsule length has been directly linked to excessive anterior-superior humeral translation in cadaveric study. (Harryman et al., 1990) Glenohumeral internal rotation range of motion (IR ROM) and horizontal adduction at 90° of elevation are reliable clinical measures (Tyler et al., 1999, Laudner et al., 2006b, Myers et al., 2007, Warner et al., 1990) that potentially assess posterior capsule length. Content validity for IR ROM has been demonstrated in cadaveric study with a reduction of motion after the posterior-inferior capsule was artificially shortened. (Gagey and Boisrenoult, 2004, Gerber et al., 2003) Construct validity has been demonstrated for the measure of horizontal adduction range of motion by its ability to identify deficits unique to overhead athletes. (Myers et al., 2007) Clinical measures of glenohumeral internal rotation and horizontal adduction range of motion may also be influenced by potential adaptations of the infraspinatus, teres minor, and/or posterior deltoid musculature (Reinold et al., 2008), or osseous changes of humeral and/or glenoid retroversion (Schwab and Blanch, 2009, Crockett et al., 2002, Osbahr et al., 2002, Reagan et al., 2002).

A relationship between the two measures of posterior shoulder tightness, horizontal adduction and IR ROM, have been found in patients with RC tendinopathy (Tyler et al., 2000) and asymptomatic professional baseball pitchers (Laudner et al., 2006b). Posterior shoulder tightness has been demonstrated in patients with RC tendinopathy. (Warner et al., 1990, Myers et al., 2006, Tyler et al., 2000)
Furthermore, stretching to address impairments of posterior shoulder tightness has been identified as an important component to rehabilitation for patients with RC tendinopathy (Kuhn, 2009), and change in IR ROM was significantly correlated (r=0.54) with functional improvement in patients undergoing rehabilitation (Mcclure et al., 2004). While this mechanism for RC tendinopathy may be prevalent, this is likely not a contributing mechanism of all patients with RC tendinopathy.

Deficits in RC muscle performance contribute to RC tendinopathy, by leading to proximal migration and subsequent intrinsic breakdown or extrinsic impingement. (Chen et al., 1999, Royer et al., 2009, Deutsch et al., 1996) In biomechanical studies, decreased RC muscles force, in particular the infraspinatus has resulted in increased superior humeral head translation and decreased abduction torque. (Hurschler et al., 2000, Mura et al., 2003, Sharkey and Marder, 1995) Although more recently, the concept that decreased RC muscle performance alone can result in proximal humeral migration has been challenged with an in vivo study. Artificially induced paralysis of the supraspinatus and infraspinatus muscles in 10 healthy individuals resulted in no immediate effects on proximal humeral head translation. (Werner et al., 2006) Results of this study suggest time, or duration of the muscle impairment may also be a factor. Significant decreases in RC muscle peak isometric, concentric, and eccentric torque have been demonstrated in patients with RC tendinopathy compared to asymptomatic subjects. (Leroux et al., 1994, Warner et al., 1990, Macdermid et al., 2004, Tyler et al., 2005) Reddy et al found a decrease in electromyographic (EMG) activity of the infraspinatus, and subscapularis from 30° to 60° of active elevation and in the infraspinatus muscle alone from 60° to 90°
of active elevation in subjects with tendinopathy compared to healthy subjects. (Reddy et al., 2000) Diederichsen et al found decreased infraspinatus EMG muscle activity with resisted external rotation in patients with RC tendinopathy compared to healthy subjects. (Diederichsen et al., 2008) However, alterations in muscle activity were also found in the asymptomatic side leading the authors to propose alterations in muscle activity are a factor in the pathogenesis not a result of RC tendinopathy. Lastly, Myers et al found a decrease in co-activation ratios of the subscapularis- infraspinatus and supraspinatus-infraspinatus muscles with arm elevation from 0-30°, and an increase at elevation above 90° in patients with impingement compared to control participants. (Myers et al., 2008) Decreased RC muscle co-activation levels may occur as a result of pain (Myers et al., 2008) or altered scapular or humeral head position or movement, changing the muscle length tension relationship and therefore muscle force (Michener et al., 2003). Biomechanical consequences of altered RC muscle activity may be an extrinsic mechanism of RC tendinopathy as superior migration may narrow the subacromial space or result in altered stress and intrinsic tendon degradation. Diminished RC muscle performance correlates with patient-rated function and health-related quality of life in patients with RC tendinopathy. (Macdermid et al., 2004)

No study has concurrently examined the influence of scapular position on RC muscle activity in patients with RC tendinopathy; however, there is evidence to suggest that a change in scapular position can alter muscle performance. (Kibler et al., 2006, Tate et al., 2008, Kebaetse et al., 1999) Kebaetse et al found a decrease in isometric abduction muscle force with the arm at 90° concurrent with an increase in scapular anterior tilt in
healthy subjects actively assuming a slouched trunk posture compared to an erect posture.(Kebaetse et al., 1999) Other research has found that passively altering scapular position, as with scapular retraction and scapular reposition tests, influences isometric arm elevation isometric force(Kibler et al., 2006, Tate et al., 2008, Smith et al., 2002), with an increase noted with reposition and conflicting results with scapular retraction. Changes in muscle force may be due to improved proximal stability or alterations in RC muscle length at the same humeral elevation angle.

EXTRINSIC MECHANISMS FOR THE SUBGROUP OF INTERNAL IMPINGEMENT

A unique subset of RC tendinopathy with an extrinsic mechanism is internal impingement. Patients with internal impingement tend to present with pain located in the posterior and superior aspect of the shoulder typically while the arm is in abduction and external rotation of the late cocking phase of throwing.(Jobe, 1995, Kvitne and Jobe, 1993) In this position, the articular aspect of the RC tendons become mechanically impinged between the posterior superior glenoid rim and the humeral head. This is accentuated with further hyperangulation of the humerus to the glenoid with anterior glenohumeral joint instability(Davidson et al., 1995) or in theory, with a reduction in scapular retraction (Burkhart et al., 2003, Kibler, 1998) and posterior tilt.(Laudner et al., 2006a) Alterations in scapular kinematics were found in a cohort of baseball players with internal impingement, confirmed with arthroscopy, of greater scapular posterior tilt compared to age matched healthy baseball players.(Laudner et al., 2006a) In contrast, a decrease in scapular posterior tilt has been frequently found in patients with RC

Conflicting findings in scapular kinematics may not be due to causative or compensation patterns of RC tendinopathy as previously theorized (Ludewig and Cook, 2000, McClure et al., 2006), but may be a result of differences in underlying mechanism. Further study should examine potential differences in mechanisms of this unique subgroup of RC tendinopathy.

**INTRINSIC MECHANISMS OF ROTATOR CUFF TENDINOPATHY**

There is a growing body of evidence to support an intrinsic mechanism. Intrinsic mechanisms of RC tendinopathy influence tendon morphology and performance. Intrinsic factors of RC tendinopathy result in tendon degradation due to the natural process of aging (Iannotti et al., 1991, Milgrom et al., 1995, Sher et al., 1995, Tempelhof et al., 1999), poor vascularity (Biberthaler et al., 2003, Brooks et al., 1992, Fukuda et al., 1990, Goodmurphy et al., 2003, Rathbun and Macnab, 1970, Rudzki et al., 2008), altered biology (Kumagai et al., 1994, Riley et al., 1994a, Riley et al., 1994b), and inferior mechanical properties resulting in damage with tensile or shear loads (Huang et al., 2005, Reilly et al., 2003a, Lake et al., 2009, Bey et al., 2002). A genetic component for the development of RC disease has also been identified (Harvie et al., 2004) and theorized to be related to polymorphism of collagen genes such as found with Achilles tendinopathy (Mokone et al., 2005); however, no specific genotype has yet to be identified as a risk factor for the development of RC disease (September et al., 2007).
Furthermore, RC tendinopathy with an intrinsic mechanism may lead to a reduction in subacromial space creating an interaction of intrinsic and extrinsic mechanisms.

The morphology of the RC tendons has been studied in detail. The RC tendon near their insertions have been shown to interdigitate; specifically, the supraspinatus tendon consists of five axial plane layers from the bursal to articular side (Clark and Harryman, 1992) in the critical zone where pathology is most prevalent (Codman, 1934). RC tendinopathy can include symptomatic tendon pathology with degeneration and partial thickness tears that extend through several, but not all layers. It is commonly described as occurring in 3 regions: bursal-sided, mid-substance, and articular-sided. Furthermore, pathology that occurs within the mid-substance and articular sided layers without bursal side involvement is further support for the intrinsic mechanisms of RC tendinopathy (Fukuda et al., 1990, Hashimoto et al., 2003).

**Age-Related Degenerative Changes**

Codman first proposed an underlying degenerative process within the tendon which precedes suprapsinatus tendinopathy and tears. (Codman and Akerson, 1931) Neer described RC disease as a continuum of pathology with 3 stages characterized by age: less than 25 years for stage I, between 25 to 40 years for stage II, and greater than 40 years of age for stage III respectively. (Neer, 1983) Although Neer’s theory is biased by an extrinsic mechanism, age was included as an important factor for RC disease. The prevalence of tendon degeneration including partial and full thickness tears increases as a function of age starting at 40 years (Iannotti et al., 1991, Milgrom et al., 1995, Sher et al., 1995, Tempelhof et al., 1999). Additionally, a prospective study has shown RC disease is
progressive and leads to pain and disability in more than 50% of previously asymptomatic individuals in less than 4 years. (Yamaguchi et al., 2001)

Age has been shown to have a negative impact on tendon properties. Evidence from biomechanical studies suggest there is a reduced toe region of a stress-strain curve, decreased elasticity, and decreased overall tensile strength of tendons with age. (Woo et al., 2000) Histological study of RC tendons have shown calcification and fibrovascular proliferation degenerative changes in elderly subjects without history of shoulder ailments that were not present in younger subjects, both without a history of shoulder ailments. (Kumagai et al., 1994) Also, with age, there is a decrease in total glycosaminoglycan (GAGs) and proteoglycans (PGs) content in the supraspinatus tendon (Riley et al., 1994a). An overall reduction of collagen content and an increased proportion of weaker, more irregularly arranged type III collagen has been found with aging (Kumagai et al., 1994); however, there is conflicting evidence that these changes in the supraspinatus are not age related but attributed to inferior healing response from microtrauma to the tendon. (Bank et al., 1999, Riley et al., 1994a, Riley et al., 1994b) There is no consensus whether changes in the tendon are primarily due to aging or a secondary consequence of reduced mechanical properties that make the tendon more susceptible to injury with repetitive motion. Regardless, age related changes to the tendon appear to be a significant factor in the intrinsic pathoetiology of RC tendinopathy.

**Tendon Vascularity**

A deficient vascular supply of the human RC tendons has been implicated in the pathogenesis and mechanism of RC tendinopathy. Codman first described the ‘critical
zone’, an area within the supraspinatus tendon approximately one centimeter from the insertion on the greater tubercle with decreased vascularity and the most common site for RC tendon injury (Codman, 1934). Furthermore, this hypovascular zone and resultant diminished healing capacity predisposes one to RC tendinopathy (Biberthaler et al., 2003, Brooks et al., 1992, Fukuda et al., 1990, Goodmurphy et al., 2003, Rathbun and Macnab, 1970, Rudzki et al., 2008) and tends to worsen with age. (Rudzki et al., 2008) However, this notion has been challenged with in vivo studies that found no apparent region of avascularity in the critical zone (Longo et al., 2008, Matthews et al., 2006, Levy et al., 2008) or evidence that hypovascularity is limited to the articular side and not the bursal side of the tendon. (Lohr and Ulthoff, 1990, Rudzki et al., 2008)

Research suggests an increased vascular response, or neovascularization, in regions of degenerative changes and smaller tendon tears such as with chronic RC tendinopathy (Fukuda et al., 1990, Hashimoto et al., 2003, Kumagai et al., 1994, Rathbun and Macnab, 1970, Goodmurphy et al., 2003, Levy et al., 2008), that is theorized to be a healing response to tissue microtrauma (Rathbun and Macnab, 1970, Levy et al., 2008). In contrast, tendinopathy that progresses to complete tendon tears have been shown to be avascular. (Matthews et al., 2006, Biberthaler et al., 2003, Fukuda et al., 1990, Rathbun and Macnab, 1970) It is unclear whether this avascular condition is a cause of progressive tendinopathy or a consequence of a complete tear. In subjects with RC tendinopathy, imaging with laser or ultrasound color Doppler has been used to detect the presence of neovascularization in vivo. (Alfredson et al., 2003, Levy et al., 2008) Levy et al found subjects with acute RC tendinopathy (impingement without tear) had hypovascularity in
the supraspinatus tendon compared to subjects without RC disease, while those with chronic RC tears had hypervascularity near the degenerative changes. (Levy et al., 2008) The role of vascularity in the intrinsic mechanism of symptomatic RC tendinopathy has not been fully elucidated, however it does appear to be a factor that is influenced by and or influences the extent and duration of tendon pathology.

Impact of Alterations in Tendon Matrix on Mechanical Properties

The composition and organization of the tendon matrix dictate the morphology and mechanical properties of tendons. Tendons are composed of proteins, collagen, and cells referred to as tenocytes. Collagen fibers in tendons are composed predominately of type I molecules in tight and parallel fiber bundles and a small proportion (<5%) of type III collagen fibers that are thinner, weaker, and more irregularly arranged. (Riley et al., 1994b, Kumagai et al., 1994) The collagens within the RC tendon matrix are stabilized by formations of cross-links, specifically hydroxylyslypyridinoline and lysylpyridinoline. (Bank et al., 1999) Within the RC tendons of elderly samples, the distribution of collagen types has been shown to vary with greater proportion of type II and III collagen near the insertional fibrocartilagenous region compared to more proximal tendon. (Kumagai et al., 1994) Since type III collagen fibrils are considered more extensible than type I fibers and tend to be more irregularly arranged, authors theorized that the insertional region of the supraspinatus may be subjected to greater non-linear stresses than other RC tendons. In agreement with this theory, Lake et al quantified the degree of collagen fiber alignment in different longitudinal sections of the supraspinatus tendon and demonstrated a highly inhomogeneous tissue with a relatively low degree of
fiber alignment in the region near the tendon to bone insertion (Lake et al., 2009). These changes correlated with diminished mechanical properties in this region. Furthermore, histological evidence of inferior tissue organization, greater disorganization in the mid-substance and/or the articular side compared to more regularly arranged collagen on the bursal-side layers of the RC tendons has been proposed to weaken the tendon and precede complete tendon tear (Fukuda et al., 1990, Hashimoto et al., 2003).

The intrinsic mechanism of RC tendinopathy assumes the demands placed on the tendon cells at some point exceeds the ability to effectively repair structural deficits (Riley, 2004) resulting in breakdown and eventually pain. Studies that have examined alterations in RC tendon matrix have found no differences in total GAG concentration and PG content (Riley et al., 1994a), but a reduction in total collagen content and an increased proportion of type III collagen fibers (Riley et al., 1994b) in patients with chronic RC tendinopathy compared to cadaveric samples of normal tendon. Additionally, greater tenocyte apoptosis (cell death) has been found in tendons of patients with chronic RC tendinopathy as compared to normal tendons (Yuan et al., 2002, Tuoheti et al., 2005). These matrix alterations are concurrent with morphology characterized by an irregular tendon contour and reduced tendon thickness (Selkowitz et al., 2007, Teefey et al., 2000, Teefey et al., 2004, Wiener and Seitz, 1993). Cholewinski et al found thinning of the RC tendons in patients with chronic unilateral (> 6 months) subacromial impingement compared to an asymptomatic individuals without a history of shoulder injury (Cholewinski et al., 2007).
In contrast, an accumulation of GAGs and disorganization of the collagen fibers, which is theorized to cause tendon thickening in RC tendinopathy, has been demonstrated within 12 weeks of the onset of injury.(Scott et al., 2007) The supraspinatus appears to have higher rates of collagen matrix turnover compared to other tendons and accelerates in the presence of RC pathology(Bank et al., 1999). In an animal model, tendon cells in the supraspinatus become more chondroid and increase proliferation in an acute injury(Scott et al., 2007). Joensen et al(Joensen et al., 2009) found that increased RC tendon thickness of greater than or equal to 0.80mm compared to the asymptomatic shoulder was associated with RC tendinopathy. The conflicting findings of tendon thickness in this study compared to those of tendon thinning by Cholewinski et al(Cholewinski et al., 2007) may be attributed to the duration of symptoms. Inclusion criteria for Joensen et al(Joensen et al., 2009) were greater than 1 month with 30% of the subjects having pain less than 3 months in duration compared to an inclusion criteria of pain greater than 6 months in duration in study by Cholewinski et al (mean duration was 7 months, range 6-48 months). Overall, tendon morphology has been suggested to vary based on the duration of tendon injury. An acute injury exhibits increased diffuse tendon thickness associated with matrix changes of a healing response(Malliaras et al., 2009) while a more chronic tendinopathy demonstrates focal defects and tendon thinning associated with degeneration.

**Tensile Tissue Overload: Inhomogeneous Mechanical Properties**

Another proposed intrinsic mechanism of RC tendinopathy is related to the response of the tendons to tensile load, or mechanical properties of the supraspinatus
tendon. (Bey et al., 2002, Hashimoto et al., 2003, Huang et al., 2005, Reilly et al., 2003a)

Lower ultimate strain values (Bey et al., 2002, Huang et al., 2005) and greater tissue
stiffness (Nakajima et al., 1994) to longitudinal loading have been found on the articular
side of the supraspinatus tendon near the insertion as compared to the bursal side;
although, this was in conflict with results of studies by other investigators who found no
differences in mechanical properties between articular (deep) and bursal sided
(superficial), but lack of homogenity between the anterior and posterior supraspinatus to
longitudinal loads. (Itoi et al., 1995, Lake et al., 2009) Moreover, loading the tendon at
various arm positions may result in strain differentials between the articular and bursal
side of the supraspinatus. Greater strain has been shown on the supraspinatus tendon
articular side with the arm positioned at the beginning of elevation (angles <30°
abduction (Bey et al., 2002, Huang et al., 2005) and 62° abduction (Huang et al., 2005))
while Reilly et al found a progressive increase in articular sided strain with elevation
(angles 0 to 120° abduction) (Reilly et al., 2003a). Greater bursal sided strain was found
when the glenohumeral joint is at mid-ranges (90°) (Huang et al., 2005). While there is
inconsistency with the specific results and methods used among these studies,
intratendinous degradation is theorized to result from shearing between various portions
of RC, specifically the supraspinatus tendon (Fukuda et al., 1990, Lee et al., 2000)
potentially due to the distinct mechanical characteristics and force differentials incurred
with various loads (Bey et al., 2002, Huang et al., 2005, Nakajima et al., 1994, Reilly et
al., 2003a, Lake et al., 2009). Intrasubstance degeneration in the supraspinatus initiates
mid-substance tendon tears and propagates with continued loading to an articular side.
tendon tear before complete tendon failure. (Reilly et al., 2003b) Biomechanical consequences of complex longitudinal and transverse inhomogeneous tendon properties would be exacerbated in combination with extrinsic factors such as repetitive tensile loading induced with daily activities such as lifting or pulling or the strain incurred with the follow through phase of overhead sports.

Other factors than collagen fiber alignment, such as tendon geometry can influence the mechanical properties. Alterations in tendon geometry including tendon irregularity and thinning has been demonstrated in patients with degenerative RC pathology (Cholewinski et al., 2007) which could influence its mechanical properties. Thickening of the tendon associated with an acute healing response to injury may create greater area to distribute forces; however, tendon thinning associated with degenerative or chronic tendinopathy would reduce the surface area for the same load conditions thus may perpetuate injury. A weak correlation has been shown between supraspinatus tendon thickness and in vivo mechanical properties. (Bey et al., 2002) There also is a strong correlation between the extent of RC tendon degenerative changes and tensile strength; as tendon degeneration increases the tensile strength decreases. (Sano et al., 1997).

However, both of these studies examined the mechanical properties of cadaveric tissue samples. However in patients with RC tendinopathy, a decrease in tendon thickness has been shown to be associated with a decrease in muscle performance. (Joensen et al., 2009)

SUBGROUPS OF PATIENTS WITH TENDINOPATHY BASED ON MECHANISM
Subgroups of RC tendinopathy may exist, based on intrinsic and extrinsic mechanism that may serve to facilitate treatment decision-making for patients with RC tendinopathy. In a cadaver study, bursal-sided tendon degeneration with partial thickness tears were always associated with attritional lesions on the coracoacromial ligament and anterior third of the acromion (Ozaki et al., 1988); however, this was not true of articular-sided RC pathology in which the undersurface of the acromion was almost always normal. Similarly in patients, milder pathological changes of the undersurface of the acromion and less severe RC degenerative changes were found in patients with articular sided RC pathology compared to bursal sided (Ko et al., 2006). It appears there is a link between pathoanatomy and mechanism of RC tendinopathy; articular sided degenerative changes of the tendons are primarily associated with an intrinsic mechanism, and bursal sided pathologies of the tendons are more associated with an extrinsic mechanism. As each of these distinct mechanisms progress, they may increasingly overlap. A patient with primary extrinsic compression mechanism of RC tendinopathy may progress with degenerative changes to the RC tendons over time. Alternatively, a patient with primary intrinsic degenerative mechanism of RC tendinopathy may progressively lose stabilizing function of the RC resulting in excessive superior humeral migration and extrinsic compression.

The literature suggests using the link between pathology and mechanism to drive treatment choices. For surgical treatment of RC tendinopathy attributable to an intrinsic mechanism, debridement of the RC without acromioplasty has been advocated (Budoff et al., 1998, Goldberg et al., 2001). In contrast, if the RC lesion is attributable to extrinsic
mechanism, decompression in the form of an acromioplasty has been proposed as a key component of the surgical procedure. (Neer, 1972, Neer, 1983, Rockwood and Lyons, 1993) Rehabilitation treatment decision-making for RC tendinopathy is not driven primarily by mechanism but also on impairments. Substantial evidence indicates exercise programs that include strengthening of the scapular stabilizers and RC muscles, flexibility exercises for the posterior shoulder, thoracic spine and pectoralis minor muscle, postural education and activity modification are beneficial in reducing pain and disability in patients with RC tendinopathy. (Brox et al., 1993, Haahr et al., 2005, Kuhn, 2009, Lombardi et al., 2008) Manual therapy of the spine and shoulder in addition to exercise programs have been shown to be beneficial, and superior to exercise alone (Bang and Deyle, 2000, Conroy and Hayes, 1998). Exercise programs for patients with RC tendinopathy appear to be biased towards the treatment of outlet impingement, an extrinsic mechanism which is likely not present in all patients with RC tendinopathy. The varied nature of contributing mechanisms indicates RC tendinopathy is not a homogenous entity, and thus may deserve different treatment interventions.

**Factors for Specific Subgroups of RC Tendinopathy**

Mechanism of RC tendinopathy as internal impingement, extrinsic, intrinsic, or a combination can be used to create patient subgroups. Outcomes of current rehabilitation approaches are not effective for all patients, with >30% of all patients treated with rehabilitation or surgery considered unsuccessful with persistent pain and disability (Brox et al., 1999, Brox et al., 1993). Improving specificity of treatment may improve treatment outcomes. Despite distinct differences in biomechanical mechanisms that appear to be
associated with internal impingement and intrinsic versus extrinsic mechanisms, there is a paucity of research that indicates which clinical examination findings identify the mechanism, and are thus useful to develop specific rehabilitation components. Factors from the patient history and examination that potentially can be used to indicate the underlying mechanism are shown in Table 2 which includes the location of symptoms, pain response with special tests based on altering symptoms, and precipitating event or activity. These factors may provide evidence of a particular contributing biomechanical mechanism and useful for guiding treatment decision-making.

CONCLUSION

RC tendinopathy is a common disorder that poses challenges for effective treatment. Evidence suggests extrinsic, intrinsic, and combinations of biomechanical mechanisms play a role. Intrinsic mechanisms, such as RC tendon mechanical properties, composition, and vascularity, and extrinsic mechanisms, such as alterations in scapular and glenohumeral kinematics that contribute to either internal and external impingement, appear to be particularly significant factors of RC tendinopathy. Research focused on prognosticating treatment outcome including the presence of a particular mechanism or combinations of mechanisms is needed. There are distinguishing characteristics from the history and physical exam that can be used to identify specific biomechanical factors to subcategorize RC tendinopathy as primarily an extrinsic mechanism, intrinsic mechanism, a combination, or internal impingement. Future research is needed to determine whether treatment distinct to these subgroups improves treatment outcomes.
References


Codman, E. A. (1934) *The shoulder: rupture of the supraspinatus tendon and other lesion in or about the subacromial bursa*, Boston, Thomas Todd.


530-6.

deficit in the prediction of tendon hypertrophy in symptomatic unilateral shoulder 

Johnson, G. R., Pandyan, A. D., 2005. The activity in the three regions of the trapezius under 

Karduna, A. R., Kerner, P. J., Lazarus, M. D., 2005. Contact forces in the subacromial space: 

Kebaetse, M., McClure, P., Pratt, N. A., 1999. Thoracic position effect on shoulder range of 
80, 945-950.

Keener, J. D., Wei, A. S., Kim, H. M., Steger-May, K., Yamaguchi, K., 2009. Proximal humeral 
migration in shoulders with symptomatic and asymptomatic rotator cuff tears. *J Bone 

(MD), Williams and Wilkins.

325-337.


Table 1. Rotator Cuff Pathological Mechanisms

<table>
<thead>
<tr>
<th>Distinguishing Features</th>
<th>Intrinsic</th>
<th>Extrinsic</th>
<th>Extrinsic- Internal Impingement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conceptual Mechanism</td>
<td>Degeneration of the tendon where tensile loading exceeds the tendon’s intrinsic healing and adaptive responses</td>
<td>Compression of the tendon within the subacromial space from anatomical or biomechanical abnormalities</td>
<td>Compression of the tendon posteriorly between the humerus and glenoid rim with abduction and external rotation</td>
</tr>
<tr>
<td>Pathology</td>
<td>Intratendinous and articular sided tendon pathology without coracoacromial abnormalities</td>
<td>Bursal sided tendon pathology with coracoacromial abnormalities more common</td>
<td>Articular sided pathology without coracoacromial abnormalities. May be related to glenohumeral joint instability.</td>
</tr>
</tbody>
</table>
Table 2. History and examination factors to subcategorize RC tendinopathy based on mechanism

<table>
<thead>
<tr>
<th>HISTORY</th>
<th>INTRINSIC</th>
<th>EXTRINSIC</th>
<th>COMBINATION EXTRINSIC &amp; INTRINSIC</th>
<th>EXTRINSIC-INTERNAL IMPINGEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event</td>
<td>Excessive tensile load (volume, intensity, or frequency) with overuse or traumatic onset</td>
<td>Overhead shoulder use</td>
<td>Overhead abduction &amp; external rotation with activity typically sport/recreation</td>
<td></td>
</tr>
<tr>
<td>Pain Location</td>
<td>Anterior shoulder, CS distribution</td>
<td>Anterior shoulder, CS distribution</td>
<td>Posterior shoulder, CS distribution</td>
<td></td>
</tr>
</tbody>
</table>

EXAMINATION FINDINGS

- **Anatomical**
  - Bony abnormalities: AC joint/acromion

- **Thoracic Posture & Mobility**
  - Mobile thoracic spine
  - Hypo/normal flexed posture

- **Scapulothoracic Motion**
  - If dyskinesia present → posterior tilt, and/or upward rotation
  - If dyskinesia present → posterior tilt, excessive rising/drug pattern

- **Symptom & Alteration Tests**
  - Negative (pain unchanged/t)
  - Positive (pain reduces ≥ 2/10 points)
  - Posterior shoulder pain
  - with apprehension test; reduced with relocation test

- **Posterior shoulder Tightness/GH Internal Rotation Deficit**
  - No or mild if present
  - Yes present

- **Pectoralis minor Muscle Length**
  - Decreased pectoralis minor index

- **RC Muscle Performance**
  - Decreased

- **Scapulothoracic Muscle Performance**
  - Impaired timing, weakness

- **Other**
  - Possible impaired timing/mobility
Figure 1. Extrinsic and intrinsic mechanisms of rotator cuff tendinopathy. Lines indicate non-directional evidence of these relationships.
Figure 2. Superior migration of the humerus (dotted line) and diminished subacromial space (solid line) in patient with a chronic large RC tear.
CHAPTER 3:
SCAPULAR UPWARD ROTATION AND POSTERIOR TILT ALTERS
SUBACROMIAL SPACE, BUT NOT STRENGTH IN SUBACROMIAL
IMPINGEMENT

ABSTRACT

STUDY DESIGN: Two-group repeated-measures.

PURPOSE: To determine the effect of the modified scapular assistance test (SAT) on 3-
dimensional shoulder kinematics, strength, and linear measures of subacromial space in
patients with subacromial impingement syndrome (SAIS).

BACKGROUND: Abnormal scapular kinematics have been identified in patients with
SAIS. Increased scapular upward rotation (UR) and posterior tilt (PT), as proposed with
manual assistance using the SAT, has been theorized to increase subacromial space and
may alter shoulder strength.

METHODS AND MEASURES: Forty-two subjects (21=with SAIS; 21=controls)
participated. The anterior outlet of the subacromial space, measured via the
acromiohumeral distance (AHD) on ultrasound images, and 3D scapular kinematics,
measured using electromagnetic motion analysis, were determined with the arm at rest,
45°, and 90° of elevation with and without SAT. A dynamometer was used to measure
shoulder isometric joint torque. Full factorial mixed-model ANOVAs evaluated the
effects of SAT on variables between groups. Associations between change in scapular
position, AHD, and torque with the SAT were explored with linear correlations.
RESULTS: The SAT increased scapular PT at all angles and UR at rest and 45° elevation. The SAT did not alter peak torque. AHD increased with the SAT at 45° and at 90°. SAIS did not have an effect on these changes. Greater UR was associated with smaller increase in AHD reduction at 90°.

CONCLUSIONS: The SAT increases scapular PT and subacromial space during active arm elevation. SAIS did not influence changes with the SAT. Scapular PT is likely more beneficial than UR.

KEY WORDS: acromiohumeral distance, scapular kinematics, strength, rotator cuff, scapular assistance test
INTRODUCTION

Adequate scapular motion during arm elevation is necessary for normal shoulder mechanics. During humeral elevation the scapula rotates in 3 directions, upward rotation, posterior tilt, and external rotation, to position the glenoid and allow clearance of the humeral head beneath the acromial arch.23, 38 Subacromial impingement syndrome (SAIS) is characterized by pain during an arc of elevation41, 42, where the minimal distance between the acromion and humerus is reduced in part by the greater tuberosity passing beneath and approximating the anterior aspect of the acromion15 resulting in compression41.

Substantial evidence indicates patients with SAIS demonstrate abnormal scapular motion.12, 18, 22, 30, 32, 34, 35, 37, 39, 51 The majority of evidence suggests patients with SAIS present with decreased scapular posterior tilt12, 34, 35, upward rotation12, 34, 48, and external rotation12, 22, 34, 51 when compared to control subjects. This pattern of motion has been theorized to contribute to compression of the rotator cuff tendons by failing to elevate the anterior-lateral margin of the acromion34, the predominant site of SAIS.14, 41, 42 In contrast, increased scapular posterior tilt30, 37 and upward rotation37 have also been identified in patients with SAIS. Increased posterior tilt and upward rotation are theorized to be favorable compensatory responses to relieve compression on the rotator cuff tendons by increasing the subacromial space37.

To date, there is little evidence to support the proposed mechanistic theories that changes in scapular kinematics alter subacromial space in SAIS. In healthy subjects,
passively altering scapular position from protraction to retraction increases subacromial space. Another study found limiting scapular motion by artificially binding the scapula to thorax reduces subacromial space in healthy subjects compared to elevation without limiting scapular motion. However, it is unknown what specific scapular rotations and how much of each are necessary to affect the anterior aspect of the subacromial space during active arm elevation in healthy individuals, as well as patients with SAIS. This holds important ramifications for treatment of patients with SAIS because specific scapular stretching and strengthening exercises are commonly prescribed, but vary from shrugs to tactile cuing strategies to prevent shrugging during active humeral elevation.

Several clinical examination methods presume to passively alter the position of the scapula to assess change in shoulder muscle strength, pain, or both compared to the natural unassisted condition during arm elevation. The scapular assistance test (SAT) is performed with the examiner manually assisting the scapula into upward rotation by pushing the inferior medial border of the scapula as the patient elevates the arm and has since been modified to include manual assistance into upward rotation and posterior tilt. In theory the SAT increases subacromial space and may alter rotator cuff muscle performance. It has been suggested that a reduction in pain with the SAT may be an indicator for the need to address scapular motion abnormalities in the rehabilitation of individuals with shoulder pain. The SAT has demonstrated acceptable inter-tester
reliability (76-84% agreement; kappa coefficients 0.51-0.66) for clinical use in patients with shoulder pain.43

While manual assistance with the SAT is proposed to directly alter the position of the scapula to increase subacromial space and alter rotator cuff strength, it is unknown whether, and if so, how much these factors are altered with manual assistance of the SAT. Contrary to theoretical mechanism for the SAT, recent cadaveric research suggests that increasing scapular upward rotation decreases subacromial clearance, while posterior tilt has no significant effect.24 In patients with SAIS, the effect of passive scapular upward rotation and/or posterior tilt on subacromial space is yet to be clearly determined.

While other methods of passively altering the scapula (ie. scapular retraction28 and reposition tests49) have been shown to influence muscle performance, the effect of the SAT on muscle performance has not been elucidated. Therefore, the purpose of this study is to determine the effect that manual assistance using the SAT has on 3D scapular position in upward rotation and posterior tilt, subacromial space, and shoulder strength within and between subjects with SAIS and control subjects. Additionally, we explored relationships between: 1) changes in 3D scapular position and subacromial space with the SAT, and 2) changes in subacromial space and rotator cuff strength with the SAT. Alterations in scapular motion have been theorized to impact SAIS by directly influencing the subacromial space and muscle performance of the shoulder. Examining these relationships will enhance our understanding of the influence of scapula motion on
mechanisms of SAIS. These results will also provide rationale to select and deliver
treatment interventions for patients with SAIS.

METHODS

Subjects

All subjects (N=45) recruited for this study were between 18 and 70 years of age,
without known systemic connective tissue disease, pain with cervical spine range of
motion, a history of shoulder fracture or surgery, or presence of a full-thickness rotator
cuff tear as diagnosed by a radiologist with ultrasound (US) imaging or magnetic
resonance imaging. Subjects seeking care for shoulder pain (n=23) and diagnosed by a
health care provider with SAIS were recruited from an academic orthopedic surgery
practice and local physical therapy clinics. Subjects diagnosed with SAIS were screened
by a physical therapist to confirm eligibility with the following inclusion criteria: 1) the
presence of shoulder pain, 2) a positive painful arc during arm elevation, 3) positive
impingement sign (Neer or Hawkins), and 4) pain with resisted isometric elevation and
internal rotation (Empty Can test) or resisted external rotation. Additionally, subjects
with SAIS were excluded if they had signs of adhesive capsulitis, defined as at least a
50% loss of passive range of motion in two planes of motion, or a positive apprehension
sign. As part of the screening examination, the SAT was performed on all subjects with
SAIS. Subjects were asked to rate their pain on a 11-point numeric rating scale (0-10,
with 0 defined as no pain) during arm elevation with and without the SAT. Subjects
without shoulder or cervical spine pain, known shoulder pathology (n=22) served as
control subjects, and were recruited from the community and matched with regard to age, gender, and arm dominance of the shoulder tested. with SAIS during the clinical examination to determine eligibility.

Two subjects with SAIS were excluded, one due to a full-thickness rotator cuff tear on US imaging and another due to a greater tuberosity fracture. One control subject was excluded due to kinematic data retrieval errors. Subsequently, data from 42 of the 45 adults recruited were retained for analysis; final sample included SAIS subjects (n=21) and control subjects (n=21). All subjects completed an intake questionnaire which included the Penn Shoulder Score. Subject characteristics, including descriptive self-report measures of pain, satisfaction, and function using the Penn Shoulder Score\textsuperscript{31}, are shown in TABLE 1. There were 11 females and 10 males in each cohort. The dominant shoulder, defined as the ipsilateral shoulder of the hand used to write, was tested in 15 subjects in each cohort. All subjects completed and signed the Virginia Commonwealth University Institutional Review Board-approved informed consent for this study protocol prior to participation.

Prior to initiating the study, a sample size of 20 subjects per group was calculated to be necessary to provide 90% power, with significance set at $\alpha=0.05$, to detect clinically meaningful differences of 2.0mm change in AHD with the SAT, and a standard deviation of 2.0mm based on pilot testing\textsuperscript{45}. Clinically meaningful changes in AHD were defined as the minimal detectable change (MDC) with 90% confidence bounds.

**Instrumentation**
Three types of data were collected: three-dimensional (3D) scapular kinematics, US images of the anterior outlet of the subacromial space, and isometric force during shoulder elevation and external rotation (ER). The Polhemus 3Space Fastrak electromagnetic-based motion capture system (Polhemus, Colchester, VT), sampling rate of 30 Hz, was used with Motion Monitor software (Innovative Sports Training, Inc, Chicago, IL) to collect 3D kinematic data of the scapula, humerus, and trunk. The electromagnetic tracking device consists of a transmitter affixed to a level rigid base, 4 receivers, one attached to a stylus used to digitize anatomical landmarks, and a systems unit. The transmitter emits a magnetic field with a measurement range of 0.45 - 2.4 m detected by the receivers. The reported accuracy of the electromagnetic tracking device is 0.8 mm and 0.15°. This surface-based electromagnetic tracking method to track scapular motion has been validated using bone pins. Average errors for skin mounted sensors is 1.06 mm linear displacement and 3.56° rotation for scapular motions during humeral elevation with the greatest error occurring above 120° of humeral elevation.

A diagnostic US unit, LogiQe (GE Healthcare, Wisconsin, USA) with a 4-12 mHz adjustable linear array transducer set at a frequency of 8.0 mHz was used to capture images in B-mode for subacromial space measurement. Subacromial space was operationally defined as the acromio-humeral distance (AHD), the shortest linear distance between the humeral head and the anterior inferior tip of the acromion. AHD was measured on US images using onscreen calipers (FIGURE 2.). The AHD measurement represents a 2-dimensional linear measure of the anterior outlet of the subacromial space.
US-generated AHD measures have demonstrated satisfactory reliability with intraclass correlation coefficients from 0.86-0.91 with the arm at rest (0°), 45°, 60° active arm elevation\textsuperscript{11} and 90° passive elevation\textsuperscript{50} and concurrent validity with radiographs (r= 0.77 - 0.85)\textsuperscript{5,6}.

Maximum isometric force production was measured with a dynamometer (Microfet; Hoggan Industries, Draper, UT) that was fixed to a rigid metal bar and clamped to a door frame to reduce measurement variation attributed to differences in examiner force and stabilization\textsuperscript{9,49}. Excellent inter- and intra-rater reliability has been reported using a handheld dynamometer for assessment of shoulder strength in both symptomatic\textsuperscript{20} and healthy subjects\textsuperscript{1}.

**Procedures for Data Collection**

Following informed consent and eligibility screening, subjects completed an intake questionnaire including a Penn Shoulder Score\textsuperscript{31} with subscales for pain, satisfaction and function. Subjects’ height (m), body mass (kg), and arm length (m) were measured and recorded. Collection of three dimensional scapular kinematic data and US imaging of subacromial space were conducted sequentially on all subjects during the active elevation procedure. Maximum isometric force was collected on a subset of control subjects (n=17) and subjects with SAIS (n=18). The three dependent variables of kinematics, AHD, and isometric force were collected with and without scapular assistance. The SAT was performed on all subjects as described by Rabin et al\textsuperscript{43} by a single experienced examiner. The SAT was used in this study as a method to passively
alter the position of the scapula in the direction of upward rotation and posterior tilt. The average of two trials for all dependent variables of scapular kinematics, AHD, and muscle performance measure was used for data analysis.

3D Scapular Kinematic Data Collection

Three electromagnetic sensors were attached to study participants using double-sided tape (3M Health Care, St Paul, MN) and further secured with CoverRoll (Beiersdorf, Norwalk, CT). One sensor was placed on the thorax over the spinous process of T3. The scapula sensor was placed over the broad flat surface of the posterior-lateral acromion and the humerus sensor was placed over the posterior aspect of the humerus distal to the triceps muscle belly, further secured with a Velcro strap. Anatomical landmarks on the subject’s thorax, scapula, and humerus were then palpated and digitized using the hand-held stylus following recommendations from the International Society of Biomechanics (ISB).52 Digitization allowed transformation of the sensor position and orientation into anatomically based position and orientation data of the humerus and scapula with respect to the thorax. Euler angle sequences were used for the humeral (Y-X’-Y’’) and scapular (Y-X’-Z’’) rotations following recommendations by ISB.52 Scapular rotations (ER, upward rotation, and posterior tilt) occur around orthogonal axes as previously illustrated and defined.36, 37

US Imaging

While the subject was seated, one examiner placed the US probe in a standardized position on the shoulder for US imaging of the subacromial space. The probe was placed
at the most anterior aspect of the anterior acromial margin confirmed with palpation, with
the long axis of the US transducer in the plane of the scapula, and parallel to the flat
surface of the acromion. Images were captured with the humeral head and acromion
consistently identified. We wanted to capture the anterior aspect of the subacromial space
because the anterior acromion has been identified as the predominant site of SAIS\textsuperscript{14, 41, 42}.
Images were saved on the US scanner hard drive for later AHD measurement. AHD
measures were made by a single examiner blinded to SAT condition and arm angle.
Examiners were not blinded to group allocation.

Active Arm Elevation Procedure

Subjects were seated in an armless chair positioned in neutral posture obtained with cues
to place feet flat on the floor shoulder-width apart, sit back in chair as far as possible with
low back against support, and look straight ahead. Shoulder kinematic data and US
images of the anterior outlet of the subacromial space were obtained with the subject’s
arm in neutral humeral rotation positioned at 0° of elevation (rest) and during active arm
elevation held statically at 45° and 90° in the scapular plane. Scapular plane was defined
as 30° anterior to the frontal plane. Positioning in the scapular plane was verified with a
goniometer and arm elevation angle was verified with an inclinometer placed parallel to
the humerus. For the trials with SAT, one examiner performed the SAT while another
examiner captured an US image or manually triggered an event indicating the SAT event
during continuous scapular kinematic data collection. Two trials were performed at each
arm angle (rest, 45°, and 90°) under each condition (with and without SAT) with both
randomized by drawing. The subject rested the arm in a suspension harness between trials. These specific arm angles were tested for several reasons. SAIS is characterized with pain during elevation\textsuperscript{41, 42}, typically in an arc of motion from where the greater tuberosity passes beneath and approximates the acromion\textsuperscript{15}. The rotator cuff tendons have been shown to pass beyond the acromion so that they are no longer susceptible to impingement within the subacromial space with elevation greater than 90°.\textsuperscript{8, 14, 15} Moreover, reduced co-activation ratios of the rotator cuff musculature have been detected in patients with SAIS during the initiation of elevation below 30°, but not at higher ranges.\textsuperscript{40} Lastly, imaging of AHD greater than 90° was challenged by poor reliability in pilot testing. Thus, scapular plane elevation at or below 90° was selected for this study as a critical range where mechanical compression of the RC tendons occurs at the anterior outlet in SAIS.

**Muscle Performance Testing**

Peak isometric contraction force of scapular plane elevation and humeral ER was measured with the subjects seated in neutral posture. To test scapular plane elevation force, subjects were positioned with the arm at 90° of elevation and 30° anterior to the frontal plane with the thumb up. The position of the dynamometer and subject was adjusted so the transducer pad contacted the distal dorsal aspect of the forearm proximal to the radial styloid for scapular plane elevation. To test humeral ER force, the subject’s arm was positioned at the side, elbow flexed to 90°, and forearm in neutral rotation. The dynamometer and subject were adjusted so the transducer pad was on the dorsal forearm
between the radial and ulnar styloid processes. All angular positions were confirmed with a goniometer. The positions used for scapular plane elevation and shoulder ER have been validated with electromyography (EMG) to demonstrate maximum activity of the supraspinatus and infraspinatus-teres minor muscles respectively. Subjects received standardized instructions on the strength testing procedures. Before testing began in each position, a sub-maximum (50%) effort trial was provided to minimize a learning effect. Maximum force output (N) of two separate 5-second maximal isometric contractions was recorded under each condition with a 30-second rest period between trials. The order of the strength tests and SAT condition (with or without SAT) was determined by random drawing. The mean peak force (N) of the two trials was converted to joint torque (Nm) by multiplying the peak force by the subject’s arm length (acromion to ulnar styloid) for elevation and forearm length (olecranon to ulnar styloid) for ER. Torque values were normalized by dividing by the subject’s mass (kg).

**Data Analysis**

Descriptive statistics were computed for all variables. Independent \( t \)-tests and chi-square tests compared subject characteristics between control subjects and subjects with SAIS. Intra-session intra-rater reliability of two trials for scapular position and orientation, AHD, and muscle performance was calculated using an intraclass correlation coefficient (ICC\(_{3,2}\)) analysis at each arm position. In addition, we examined the reproducibility of passive alteration of the scapula into upward rotation and posterior tilt using the SAT with an ICC\(_{3,2}\). The influence of manual assistance with the SAT on 3D scapular
kinematics and AHD in SAIS and control groups was determined using mixed-model
2×2×3-factorial analyses of variance (ANOVAs) with factors of SAT (with SAT; without
SAT), group (control; SAIS), arm angle (rest, 45°, 90°), and interactions. Comparisons of
interest are the main effects or interactions of group and SAT condition. Mixed-model 2-
way ANOVAs were used to assess differences in peak torque in elevation and external
rotation with 2 factors: a within subject factor of scapular assistance (with and without
SAT), between subject factor of group (control; SAIS) and interactions. Comparisons of
interest are the main effects or interactions of group. With statistical significance (α=.05),
post hoc comparisons were made with linear contrasts with a Bonferroni adjusted alpha.
The frequency of a meaningful increase in scapular upward rotation and posterior tilt,
AHD, and muscle performance with the SAT was assessed. A meaningful change was
defined as change greater than error in the measure using the upper limit of the 90%
confidence bounds of the minimal detectable change score (MDC90). The MDC90
indicates that a change of this magnitude has a 90% probability to be greater than
measurement error associated with repeated measures. MDC90 is calculated by
multiplying the average standard error of the measure (SEM) by the square root of 2 and
the z-score of 1.64. The SEM was defined as the standard deviation multiplied by the
square root of 1–ICC. To determine whether changes in scapular position with the SAT
are associated with changes in AHD, or changes in AHD are associated with changes in
strength with the SAT, separate tests of linear correlations were performed with all
subjects, subjects with SAIS, and control subject cohorts. All analyses were performed using SAS Software (JMP 8.1; SAS Institute Inc, Cary, NC).

RESULTS

As presented in TABLE 1, there were no differences in subject characteristics \((p>0.05)\) between SAIS and control groups, except as expected subjects with SAIS had lower mean Penn Shoulder pain, satisfaction, and function sub-scores and total scores \((p<0.001)\) than those without SAIS. Lower Penn Shoulder total and sub-scores are indicative of greater pain, loss of function, and less satisfaction. Intraclass correlation coefficients and MDC\(_{90}\) for scapular position and orientation, AHD, and are shown in TABLE 2.

**Scapular Kinematics**

The reliability to passively alter the orientation and position of the scapula with the SAT in upward rotation and posterior tilt was excellent with ICC\(_{3,2}\)= 0.98-0.99 (absolute angles produced with the SAT). We examined the influence of the SAT on mean 3D scapular kinematics in upward rotation (FIGURE 3) and posterior tilt (FIGURE 4) in SAIS and control groups. With both upward rotation and posterior tilt, there were no significant group by SAT condition (upward rotation, \(p=0.052\); posterior tilt, \(p=0.723\)) or group×arm angle×SAT condition (upward rotation, \(p=0.797\); posterior tilt, \(p=0.985\)) interactions. Thus, the influence of the SAT on scapular upward rotation and posterior tilt did not differ between subjects with SAIS and control subjects averaged across all angles or at individual angles. However, there was a significant interaction between arm angle and SAT condition \((F_{2,200}=8.9, p<0.001)\) with scapular upward rotation. As such, the
magnitude of upward rotation induced with SAT decreased with higher arm elevation angles. As shown in TABLE 3, the SAT increased scapular upward rotation with the arm at rest (8.9° ± 4.7°; p < 0.001) and at 45° of active scapular plane elevation (6.3° ± 3.6°; p < 0.001), but not at 90° (2.3° ± 3.8°; p=0.040) after adjusting for multiple comparisons (α=0.017). There was a significant arm angle by group interaction (F2, 200=8.0, p<0.001), with decreased scapular upward rotation during elevation in subjects with SAIS, regardless of SAT condition; however these differences were not statistically significant (p>0.1) with post hoc testing at any angle. There was no significant main effect of group with scapular upward rotation (p=0.572). With scapular posterior tilt, there was no significant interaction between arm angle and SAT (p = 0.086). There was a significant main effect of group with scapular PT (F1,40 =3.7, p=0.003); thus regardless of arm angle or SAT condition, subjects with SAIS demonstrate less scapular posterior tilt (mean difference 2.8°, 95% CI=1.0-4.6°). Additionally, there was a significant main effect of SAT condition (F1,200=164, p< 0.001). Therefore, the SAT increased posterior tilt by a mean of 4.5° ± 2.2° (95% CI= 3.7, 5.1; p < 0.001), regardless of arm angle or group.

**Acromiohumeral Distance**

The reliability of AHD measures were excellent with ICC3,2= 0.95-0.98. Mean AHD with and without the SAT in subjects with SAIS and control subjects are presented in FIGURE 5. There was a significant group by arm angle interaction (F2, 200=3.9, p=0.022). Subjects with SAIS had trends of smaller AHD at rest and 45° compared to control subjects, regardless of SAT condition, but these were not statistically different
with post hoc linear contrasts ($p>0.05$). There were no significant differences in response to the SAT between groups averaged across all arm angles (group×SAT condition; $p=0.739$) or within an individual arm angle (group×SAT condition×arm angle; $p=0.497$). There was a statistically significant interaction between arm angle and SAT condition ($F_{2,200}=5.9, p=0.005$). Thus, the influence of scapular assistance on AHD is dependent upon arm angle, regardless of SAIS. As shown in TABLE 3, the largest increase in AHD (2.1mm) with the SAT was at 45° active scapular plane elevation followed by 90° (1.7mm). There was no significant change in AHD with the arm at rest (0.8mm).

**Association between Scapular Position and AHD Changes with Scapular Assistance**

At 90° of active arm elevation, there was a significant negative linear relationship ($r = -0.48, p=0.001$) between changes that did occur in scapular upward rotation and changes in AHD with scapular assistance; a greater the increase in upward rotation was associated with less of an increase in AHD. This finding was consistent within control subjects ($r = -0.55, p=0.011$), but did not reach statistical significance in subjects with SAIS ($r = -0.36, p=0.106$). There were no significant linear relationships between change in scapular posterior tilt and AHD with scapular assistance at 90° in all subjects ($r = 0.08, p =0.588$), subjects with SAIS ($r= 0.02, p=0.908$), or in control subjects ($r = 0.16, p =0.476$). At rest and 45° of scapular plane elevation, there were no significant relationships ($p>0.05$) between change in scapular upward rotation or posterior tilt and AHD with scapular assistance in all subjects or within each group.
Muscle Performance and Association between Change in Muscle Performance and AHD

Normalized isometric mean peak torque (Nm•kg⁻¹) for ER and elevation, with and without SAT, are shown in TABLE 4. There were no significant main effects of SAT condition (ER, \(p = 0.516\); elevation, \(p = 0.811\)) or interactions between SAT condition and group (ER \(p = 0.408\); elevation, \(p = 0.913\)); altering scapular position with the SAT did not change peak torque in ER or elevation. However, there was a significant main effect for group in both ER (\(F_{1, 33}=9.0; p=0.005\)) and elevation (\(F_{1, 33}=7.5; p=0.010\)). Control subjects had greater normalized mean peak torque in ER (difference = 0.10 Nm/kg; 95% CI: 0.03, 0.20) and elevation (difference = 0.16 Nm/kg; 95% CI: 0.04, 0.27) than subjects with SAIS, regardless of scapular position (FIGURE 6).

There was no significant \((r =0.26, p=0.098)\) relationship between change in normalized ER mean peak torque and change in AHD with scapular assistance in all subjects or within either the control subjects \((r =0.42, p= 0.091)\) or SAIS subjects \((r =0.09, p=0.708)\). There was also no significant relationship between change in normalized elevation torque and change in AHD in all subjects \((r =0.14, p= 0.39)\), control subjects \((r =0.28, p= 0.222)\) and subjects with SAIS \((r =0.06, p= 0.778)\).

DISCUSSION

This study suggests manual positioning of the scapula using the modified SAT increases scapular upward rotation, posterior tilt and AHD, but does not change in ER or elevation muscle performance. Despite, decreased scapular posterior tilt, trends of decreased AHD,
and decreased ER and elevation strength in subjects with SAIS, there was no difference in change in scapular position or AHD with the SAT between SAIS and control groups. Manual assistance with the SAT appears to increase scapular posterior tilt by an average 4.5° across all arm angles (rest, 45° and 90°), regardless of SAIS. The SAT also increased scapular upward rotation at rest (8.9° ± 4.7) at 45° of arm elevation (6.3° ± 3.6), but not at 90°. Not only were changes in scapular upward rotation and posterior tilt statistically significant, the majority of subjects (79%-95%) had a meaningful change in scapular position. The proportion of subjects with changes in scapular upward rotation, posterior tilt and AHD that exceeded MDC90 are shown in TABLE 5. While scapular upward rotation, posterior tilt, and AHD increased with the SAT, the linear relationships between changes in scapular position and AHD were limited. An increase in scapular upward rotation was associated with a decrease in AHD at 90° of arm elevation, while increases in scapular posterior tilt were not significantly associated with changes in AHD at any angle of elevation.

The results of this study also show that subjects with SAIS had smaller AHD at rest and 45° compared to control subjects. This is consistent with prior studies suggesting SAIS is related to a reduction in subacromial space. Furthermore the SAT increases AHD at 45° (+2.1mm) and 90° (+1.7mm), but not with the arm at rest in both subject cohorts. As shown in TABLE 5, the majority of subjects had a meaningful change in AHD with SAT during active arm elevation that exceeded the MDC90. Altering scapular position with the SAT increases AHD during active arm elevation held
statically, but not differently in subjects with SAIS compared to control subjects. Scapular assistance with the SAT increased AHD by an average of 20.3% at 45° and 16.3% at 90°. However, the SAT did not affect the position of the scapula in upward rotation similarly across all arm angles.

Scapular posterior tilt is theorized to elevate the anterior acromion and, thus, more effectively increase the subacromial space at the anterior aspect as compared to scapular upward rotation which is theorized to elevate the lateral acromion. Results of this study support this theory. An increase in posterior tilt with the SAT was concurrent with an increase in AHD at 45° and 90° of scapular plane elevation. In spite of a lack of a linear relationship between change in AHD and change in posterior tilt with the SAT, results of this study suggest inducing posterior tilt is potentially a key component to increasing subacromial space at the anterior outlet. Given, the magnitude of posterior tilt necessary to increase AHD is not proportional; it is likely other factors not examined in this study such as posterior shoulder tightness may contribute to this finding. Perhaps a combination of rotator cuff muscle activity and increased posterior tilt is important. Additionally, decreased posterior tilt with active arm elevation has been a fairly consistent finding in individuals with SAIS. In contrast, the greatest increase in scapular upward rotation (8.9°± 4.7) with the SAT occurred at an arm position (rest) with no change in AHD. Despite there were no significant changes in scapular upward rotation at 90°, there was an increase in AHD with the SAT. Furthermore, changes that did occur in scapular upward rotation with the SAT were negatively associated (r = -0.48) with change in AHD.
at 90° of elevation. Thus, contrary to expectations, a greater increase in scapular upward rotation at 90° was associated with a smaller increase in AHD. Karduna et al\textsuperscript{24} found increasing scapular upward rotation reduced subacromial clearance with the humerus at 90° of abduction in cadaveric shoulder resections. Although we did not find with a reduction in AHD with upward rotation, our results were contradictory to our hypothesis; thus, greater change in upward rotation induced less AHD at 90°. Thus increased scapular upward rotation may not contribute to an increase in the linear dimension of the subacromial space at the anterior outlet.

Only two previous studies have examined the influence of passive alterations of the scapula on subacromial space in healthy subjects. In one study, AHD was examined while investigators passively restricted the scapula, by binding it to the thorax, in 10 healthy subjects with the arm at 60°, 90° and 120°.\textsuperscript{4} The AHD was decreased by 2.8mm at 90° compared to the unrestricted condition.\textsuperscript{4} However, this differs from results of the current study because AHD measurement was not isolated to the anterior aspect of the acromion where outlet impingement occurs\textsuperscript{14,41,42} and the arm was passively positioned which does not reflect changes in AHD with active arm elevation\textsuperscript{16} as performed in the current study. Furthermore, the magnitude or direction of change in scapular rotation(s) that occurred with scapular binding was not reported. Thus, direct comparisons of changes in AHD between studies are difficult.

Solem-Bertroft et al\textsuperscript{47} measured changes in AHD from extreme scapular retraction (adduction) to protraction (abduction) in 4 healthy subjects. Investigators
presumed to alter the position of the scapula using sandbags in subjects positioned supine, arms at their sides. A reduction of subacromial space was found in the protracted scapular position compared to retracted scapular position. Direct comparison of this study cannot be made to the current study because retraction (adduction) can occur with either scapular upward or downward rotation, as well as varying magnitudes of posterior-anterior tilt. However, results of the current study suggests therapists should encourage scapular posterior tilt perhaps, but not necessarily upward rotation, in patients with SAIS when performing manual therapy or passive scapular stretching exercises particularly when the arm is elevated to 90°. Furthermore, the technique with scapular exercises, such as shrugs and scapular retraction, should be carefully monitored to focus on increased scapular posterior tilt.

Despite a change in scapular position and AHD, the SAT did not alter isometric strength in shoulder elevation or ER. These results differ from prior studies that found increases in elevation strength with passive alterations of the scapula using the scapular retraction or reposition tests. With the scapular retraction test, the scapula is manually assisted into adduction by an examiner as compared to the reposition test in which the examiner attempts to manually induce scapular posterior tilt and ER without retraction. The scapular reposition test has been shown to increase peak shoulder elevation torque, however only 26% of subjects with impingement and 29% without had an increase that exceeded the MDC₉₀, casting doubt on the meaningfulness of this change. There have been mixed results of the influence of the scapular retraction on shoulder strength. One
investigation found decreased shoulder elevation strength with active scapular retraction compared to neutral position in healthy subjects.\textsuperscript{46} Another study found the scapular retraction test increased elevation force in patients with shoulder pain.\textsuperscript{28} Comparisons between these two studies are difficult since in one study retraction was performed actively and in the other passively (scapular retraction test). Furthermore, elevation force with manual scapular retraction was always tested after the unassisted condition. Thus, results of passive scapular retraction on elevation strength may be confounded by a learning effect of the test.

It has been theorized that a finding of increased strength or decreased pain with passive alteration of the scapula may be an indicator to include interventions to improve scapular position or muscle function in patients with shoulder pain. In prior studies, strength increases were not associated with decreased pain using the reposition test\textsuperscript{49} or were not reported with the retraction test\textsuperscript{28}. In the current study, we did not concurrently monitor pain during strength testing. We did ask subjects to rate their pain during arm elevation with and without the SAT prior to taking AHD, kinematic, and strength measurements. Interestingly, while only 2 out of 21 subjects with SAIS had a clinically meaningful change in pain with the SAT (at least a 2-points defined in previous work\textsuperscript{13}) a significant increase in AHD was noted at 45° and 90° with passive assistance into upward rotation an posterior tilt using the SAT in subjects with SAIS. Thus, short term changes in AHD with increased scapular upward rotation or posterior tilt may not immediately influence pain.
In this study, measurements of AHD, kinematics and strength were taken while performing the modified SAT with static active arm elevation. Clinically, the SAT is performed during dynamic shoulder elevation. Thus, generalizing this study’s findings to what occurs with the clinical examination using the SAT is limited. Scapular position with and without manual assistance was assessed with electromagnetic motion analysis using skin mounted sensors. In a pilot study, we determined the amount of 3D scapular motion attributed to skin artifact with scapular assistance to be minimal (0.6°± 0.9 in upward rotation and 1.6°± 0.46 in posterior tilt) in 10 subjects when the maximum amount of motion of the skin without appreciable motion of the humerus or scapula with SAT was induced. This error due to skin movement is considerably less than mean change in scapular motion with assistance (6.3°-8.9° upward rotation at rest and 45°; 4.5° posterior tilt averaged across all arm positions) and the MDC<sub>90</sub> found this study. Future study should examine changes in AHD and 3D scapular kinematics with dynamic arm elevation as performed clinically with the SAT, and determine whether this is related to changes in pain (a positive or negative test result). Additionally, factors other than rotator cuff strength should be explored to potentially explain the increase in AHD with manual assistance in scapular posterior tilt during arm elevation.

CONCLUSION

This is the first study to define the impact that passive scapular movement using the SAT has on 3D scapular position, AHD, and shoulder muscle performance in individuals with SAIS and asymptomatic individuals. Manually positioning the scapula with the SAT
increased scapular posterior tilt at rest and with static active arm elevation at 45° and 90° and upward rotation at rest and 45°, regardless of the presence of SAIS. Concurrent with scapular position changes, the AHD increases with manual assistance during active arm elevation at 45° and 90°, also regardless of the presence of SAIS. However, increased amounts of scapular upward rotation at 90° of elevation with manual assistance is associated with less of an increase in AHD and therefore, facilitation of scapular upward rotation with active arm elevation does not appear to be essential to increasing AHD at higher arm angles and may not need to be a component of rehabilitation exercises in patients with SAIS. Scapular assistance with the SAT does not alter shoulder elevation or ER isometric force, regardless of the presence of SAIS. Although only two subjects presented with a clinically meaningful decrease in pain of 2 or more points with the SAT performed during full arc of motion, there was an increase in subacromial space with passive manual assistance using the modified SAT during static active arm elevation at 45° and 90°. Thus treatment to facilitate scapular motion may be beneficial to increase AHD in individuals with SAIS, despite a negative response to the clinical examination of the SAT. This requires further study. Scapular posterior tilt, rather than upward rotation, appears to be a key component of scapular motion that increases AHD and may be beneficial to emphasize during rehabilitation interventions.

**KEY POINTS**

Passive manual correction of the scapula, using the modified SAT, is an effective method to alter the position of the scapula into upward rotation at rest and 45° of active
scapular plane elevation and posterior tilt across all arm angles (rest, 45°, and 90°).
Manual assistance with the SAT is associated with an increase in subacromial space at
45° and 90° of active arm elevation in the scapular plane, in adults regardless of the
presence or absence of SAIS. However, increasing scapular upward rotation at 90° of
elevation may be associated with a reduction in subacromial space. Passive manual
correction with the SAT does not alter shoulder strength with isometric force production.

**Implications**

The SAT is effective at increasing subacromial space with active arm elevation in
individuals with and without SAIS, with an increase in scapular posterior tilt appearing to
be more an important component than upward rotation. Increased scapular upward
rotation with the SAT does not influence AHD with the arm at rest and is associated with
less of an increase in AHD at 90° of active arm elevation. Therefore, scapular posterior
tilt should be emphasized with manual therapy or passive stretching of the shoulder in
patients with SAIS. Furthermore the technique of performing particular exercises, such as
shrugs and scapular retraction should be carefully monitored to promote scapular
posterior tilt.

**Caution**

Caution should be taken when generalizing these findings to what occurs with the
clinical examination method of the SAT as this is typically performed during active arm
elevation through a full arc of motion, not in static positions of active arm elevation as in
this study.
REFERENCES


TABLE 1. Subject Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subjects with SAIS (n=21)</th>
<th>Control subjects (n=21)</th>
<th>t</th>
<th>df</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>44.6 (10.8)</td>
<td>45.19 (11.0)</td>
<td>0.2</td>
<td>40</td>
<td>0.855</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.70 (0.10)</td>
<td>1.7 (0.08)</td>
<td>0.9</td>
<td>40</td>
<td>0.346</td>
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<tr>
<td>Body Mass (kg)</td>
<td>81.7 (16.9)</td>
<td>72.5 (15.7)</td>
<td>1.8</td>
<td>40</td>
<td>0.071</td>
</tr>
<tr>
<td>Penn Shoulder Score, Total*</td>
<td>63.7 (13.6)</td>
<td>97.7 (4.6)</td>
<td>10.8</td>
<td>40</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>pain subscore†</td>
<td>19.4 (4.3)</td>
<td>29.2 (1.3)</td>
<td>10.1</td>
<td>40</td>
<td>&lt;0.001</td>
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<tr>
<td>satisfaction subscore‡</td>
<td>4.7 (2.7)</td>
<td>9.6 (1.2)</td>
<td>7.6</td>
<td>40</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>function subscore§</td>
<td>39.6 (8.4)</td>
<td>58.9 (2.3)</td>
<td>10.1</td>
<td>40</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are mean (SD).

*Penn Shoulder Score total 0-100; 100= no pain, high satisfaction, and full function.
†Pain subscale 0-30; 30 no pain.
‡ Satisfaction subscale 0-10; 10= highly satisfied.
§Function subscale 0-60; 60= full function.
\textbf{TABLE 2.} Intraclass Correlation Coefficients (ICC\textsubscript{3,2}) and Minimal Detectable Change With 90\% Confidence (MDC\textsubscript{90}) of the Acromiohumeral Distance, Scapular Kinematics, and Shoulder Strength

<table>
<thead>
<tr>
<th>Linear Subacromial Space Measurement</th>
<th>Arm Position</th>
<th>Acromiohumeral Distance (mm)</th>
<th>ICC</th>
<th>MDC\textsubscript{90}</th>
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</thead>
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<tr>
<td>Rest</td>
<td>0.98</td>
<td>0.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>45°</td>
<td>0.96</td>
<td>0.9</td>
<td></td>
<td></td>
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<tr>
<td>90°</td>
<td>0.95</td>
<td>0.8</td>
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<table>
<thead>
<tr>
<th>Scapular Kinematics</th>
<th>Arm Position</th>
<th>Upward Rotation (°)</th>
<th>ICC</th>
<th>MDC\textsubscript{90}</th>
<th>Posterior Tilt (°)</th>
<th>ICC</th>
<th>MDC\textsubscript{90}</th>
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<tr>
<td>Rest</td>
<td>0.99</td>
<td>2.2</td>
<td>0.99</td>
<td>1.4</td>
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</tr>
<tr>
<td>45°</td>
<td>0.99</td>
<td>2.3</td>
<td>0.99</td>
<td>1.3</td>
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<tr>
<td>90°</td>
<td>0.98</td>
<td>3.4</td>
<td>0.99</td>
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<table>
<thead>
<tr>
<th>Shoulder Strength (Normalized Peak Torque)</th>
<th>External Rotation (Nm/kg)</th>
<th>Scapular Plane Elevation (Nm/kg)</th>
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<tr>
<td></td>
<td>ICC</td>
<td>MDC\textsubscript{90}</td>
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<tr>
<td></td>
<td>0.98</td>
<td>0.03</td>
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\textit{Abbreviations: ICC, Intraclass Correlation Coefficient model 3,2; MDC_{90}, Minimal detectable change with 90\% confidence bounds.}
**TABLE 3.** Scapular Upward Rotation, Posterior Tilt and Acromiohumeral Distance with and Without Scapular Assistance (SAT) in All Subjects (N=42)

### Scapular Upward Rotation

<table>
<thead>
<tr>
<th>Arm Angle</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
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<tbody>
<tr>
<td>Rest</td>
<td>12.3</td>
<td>12.7</td>
<td>8.5, 16.1</td>
<td>3.4</td>
<td>12.1</td>
<td>-0.3, 7.2</td>
<td>8.9*</td>
<td>4.7</td>
<td>6.7, 11.1</td>
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<tr>
<td>45°</td>
<td>16.4</td>
<td>12.9</td>
<td>12.7, 20.2</td>
<td>10.1</td>
<td>11.8</td>
<td>6.3, 13.9</td>
<td>6.3*</td>
<td>3.6</td>
<td>4.1, 8.5</td>
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<tr>
<td>90°</td>
<td>33.5</td>
<td>12.2</td>
<td>29.7, 37.2</td>
<td>31.2</td>
<td>11.0</td>
<td>27.4, 34.9</td>
<td>2.3</td>
<td>3.8</td>
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### Scapular Posterior Tilt

<table>
<thead>
<tr>
<th>Arm Angle</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Mean</th>
<th>SD</th>
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<tbody>
<tr>
<td>Rest</td>
<td>-9.8</td>
<td>7.6</td>
<td>-12.1, -7.6</td>
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<td>7.5</td>
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<td>-11.3</td>
<td>7.6</td>
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<td>5.1*</td>
<td>2.39</td>
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### Acromiohumeral Distance

<table>
<thead>
<tr>
<th>Arm Angle</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Mean</th>
<th>SD</th>
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<td>0.9</td>
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<tr>
<td>45°</td>
<td>10.2</td>
<td>2.1</td>
<td>9.6, 10.8</td>
<td>8.1</td>
<td>2.0</td>
<td>7.5, 8.6</td>
<td>2.1*</td>
<td>1.1</td>
<td>1.5, 2.7</td>
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<tr>
<td>90°</td>
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<td>1.9</td>
<td>9.0, 10.1</td>
<td>7.8</td>
<td>1.6</td>
<td>7.2, 8.4</td>
<td>1.7*</td>
<td>1.4</td>
<td>1.1, 2.3</td>
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</table>

*p<0.001

*Abbreviations: SD, standard deviation; 95% CI, 95 percent confidence interval*
### TABLE 4. Normalized Peak Torque in External Rotation and Elevation with and without Scapular Assistance (SAT)

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>With SAT</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Without SAT</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Difference</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects SAIS</td>
<td>17</td>
<td>0.19*</td>
<td>0.10</td>
<td>0.15, 0.24</td>
<td>0.20*</td>
<td>0.10</td>
<td>0.16, 0.24</td>
<td>-0.01</td>
<td>0.02</td>
<td>-0.02, 0.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control Subjects</td>
<td>18</td>
<td>0.30*</td>
<td>0.12</td>
<td>0.26, 0.34</td>
<td>0.30*</td>
<td>0.11</td>
<td>0.26, 0.34</td>
<td>0.001</td>
<td>0.03</td>
<td>-0.02, 0.01</td>
<td></td>
<td></td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>With SAT</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Without SAT</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
<th>Difference</th>
<th>Mean</th>
<th>SD</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects SAIS</td>
<td>17</td>
<td>0.33*</td>
<td>0.15</td>
<td>0.25, 0.41</td>
<td>0.33*</td>
<td>0.14</td>
<td>0.25, 0.41</td>
<td>0.004</td>
<td>0.06</td>
<td>-0.03, 0.04</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Control Subjects</td>
<td>18</td>
<td>0.49*</td>
<td>0.19</td>
<td>0.40, 0.57</td>
<td>0.49*</td>
<td>0.18</td>
<td>0.40, 0.57</td>
<td>0.001</td>
<td>0.06</td>
<td>-0.04, 0.04</td>
<td></td>
<td></td>
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</tbody>
</table>

* Significant difference between groups p<0.001

Abbreviations: N, number subjects; SD, standard deviation; 95% CI, 95 percent confidence interval; SAIS, subacromial impingement syndrome
### TABLE 5. Proportion of Subjects Exceeding the Minimal Detectable Change

<table>
<thead>
<tr>
<th>Arm Angle</th>
<th>Mean Increase UR(°)</th>
<th>Proportion of subjects &gt;MDC_{90}</th>
<th>Mean Increase PT(°)</th>
<th>Proportion of subjects &gt;MDC_{90}</th>
<th>Mean Increase AHD (mm)</th>
<th>Proportion of subjects &gt;MDC_{90}</th>
</tr>
</thead>
<tbody>
<tr>
<td>rest</td>
<td>8.9*</td>
<td>39/42</td>
<td>4.5†</td>
<td>33/42</td>
<td>0.8</td>
<td>25/42</td>
</tr>
<tr>
<td>45</td>
<td>6.3*</td>
<td>37/42</td>
<td>4.5†</td>
<td>40/42</td>
<td>2.1*</td>
<td>38/42</td>
</tr>
<tr>
<td>90</td>
<td>2.3</td>
<td>18/42</td>
<td>4.5†</td>
<td>39/42</td>
<td>1.7*</td>
<td>33/42</td>
</tr>
</tbody>
</table>

*statistically significant \( p<0.001 \)
† averaged across all arm angles

**Abbreviations:** SAT: scapular assistance test; MDC_{90}: minimal detectable change with 90% confidence; UR: scapular upward rotation; PT: scapular posterior tilt; AHD: acromiohumeral distance
FIGURE 1. Technique of the Modified Scapular Assistance Test as described by Rabin et al.\textsuperscript{41}
FIGURE 2. A linear measure of the subacromial space at the anterior outlet, the acromiohumeral distance, on ultrasound image
FIGURE 3. Mean (95% confidence intervals) Scapular Upward Rotation with and Without Scapular Assistance (SAT) in Subjects with Subacromial Impingement Syndrome (SAIS) and Control Subjects. Asterisk (*) indicates significant difference between with and without SAT.
FIGURE 4. Mean (95% confidence intervals) Scapular Posterior Tilt with and Without Scapular Assistance (SAT) in Subjects with Subacromial Impingement Syndrome (SAIS) and Control Subjects. Asterisk (*) indicates significant difference between with and without SAT.
FIGURE 5. Mean (95% confidence intervals) AHD with and Without Scapular Assistance (SAT) in Subjects with Subacromial Impingement Syndrome (SAIS) and Control Subjects. Asterisk (*) indicates significant difference between with and without SAT.
FIGURE 6. External Rotation and Elevation Strength (normalized peak joint torque) in Subjects with subacromial impingement syndrome (SAIS) (n=17) and control subjects (n=16) with and without SAT. Asterisk (*) indicates significant difference between SAIS and control subjects.
CHAPTER 4:

DOES SCAPULAR DYSKINESIS ALTER SCAPULAR KINEMATICS AND SUBACROMIAL SPACE WITH AND WITHOUT THE SCAPULAR ASSISTANCE TEST IN ASYMPTOMATIC INDIVIDUALS?

ABSTRACT

DESIGN: Two-group repeated-measures experimental design.

PURPOSE: To determine the influence that: 1) scapular dyskinesis, and 2) passive manual correction with the scapular assistance test (SAT) have on subacromial space and 3D scapular kinematics.

BACKGROUND: Scapular dyskinesis is an abnormal scapular motion or position during active arm motion. Dyskinesis is theorized to contribute to impingement syndrome by decreasing the subacromial space. A corrective passive maneuver of the SAT proposes to increase scapular upward rotation and posterior tilt to increase the subacromial space.

METHODS AND MEASURES: 40 asymptomatic participants were classified with obvious dyskinesis (N=20) or normal motion (N=20) using the scapular dyskinesis test. The anterior outlet of the subacromial space was measured via the acromiohumeral distance using ultrasound imaging and 3D scapular position was assessed with electromagnetic motion analysis with the arm at rest, 45°, and 90° of active elevation with and without the SAT.
RESULTS: There were no differences in acromiohumeral distance or 3D scapular kinematics with static active arm elevation between groups. The SAT increased scapular upward rotation, posterior tilt and acromiohumeral distance in both groups. Participants with dyskinesis demonstrated greater scapular mobility in upward rotation with the SAT, but not an additional increase in AHD.

CONCLUSIONS: Scapular dyskinesis does not alter scapular kinematics or acromiohumeral distance during active elevation in static positions, but did increase passive scapular upward rotation with the SAT in asymptomatic individuals. Other factors such as pain or fatigue may be requisite to alterations. The SAT increased acromiohumeral distance, thus may be helpful identifying individuals where subacromial compression is producing symptoms, regardless of dyskinesis.

KEY WORDS: scapular kinematics, scapular dyskinesis test, scapular assistance test, AHD, impingement
INTRODUCTION

Coordinated movement of the scapula is necessary for proper shoulder motion and function. During humeral elevation, the scapula moves 3-dimensionally (3D) into scapular upward rotation around an anterior to posterior axis, into posterior tilt around a medial to lateral axis in the plane of the scapula, and into external rotation around a superior to inferior axis. Abnormal position or movement of the scapula with active motion has been termed scapular dyskinesis and has been identified in patients and athletes with subacromial impingement syndrome (SAIS) and overhead athletes without shoulder pain. Patterns of decreased scapular upward rotation, posterior tilt and external rotation have been theorized to contribute to SAIS by decreasing the subacromial space.

Evidence suggests scapular dyskinesis reduces subacromial space in asymptomatic athletes. A study by Silva et al found elite adolescent tennis players with scapular dyskinesis had a reduced subacromial space from 0° to 60° abduction compared to athletes without dyskinesis. However, in this study subacromial space, measured by the acromiohumeral distance (AHD), was evaluated with the arm passively positioned. Previous studies have shown that 3D scapular position and AHD differs between passive and active humeral elevation. The construct of scapular dyskinesis should be assessed during active motion, statically held or dynamically through range, since it is defined as an abnormality with active motion. To date, the influence that scapular
Dyskinesis may have on subacromial space during active humeral elevation remains unknown.

Scapular dyskinesis can be assessed clinically or in a laboratory. Using 3D motion analysis to assess scapulothoracic motion in the clinic or athletic field is not feasible due to time constraints and expense. A clinical observation method, termed the scapular dyskinesis test, has been validated as a useful and reliable method to identify active alterations of the scapula, or obvious dyskinesis.40, 55 Athletes with obvious dyskinesis, as defined by the scapular dyskinesis test, demonstrate altered 3D scapular kinematics during active arm elevation as compared to individuals without dyskinesis.55

Scapular dyskinesis may be associated with soft tissue tightness and potentially a loss of passively assisted scapular motion. Altered scapular kinematics have been demonstrated in individuals with reduced length or increased passive tension of the pectoralis minor muscle9, 10 and thoracic spine28. However, scapular dyskinesis may also occur without a loss of passively assisted scapular motion. Determining the cause of scapular dyskinesis should enhance treatment of individuals with dyskinesis. Interventions to address limitations in passive scapular mobility may emphasize scapular and thoracic spine stretching exercises11 and manual techniques to increase mobility5, 6, 13, 32, 51. While treatment of individuals with dyskinesis and normal passive scapular mobility may focus on scapular stabilization exercises and motor control strategies35, 48. Thus, a multidimensional assessment of the scapular motion is considered an important component of a clinical examination of the shoulder in athletes and patients with dyskinesis.
Alterations in scapular kinematics can influence the subacromial space. Passive scapular protraction narrows the anterior outlet of the subacromial space compared to scapular retraction\textsuperscript{54} and restricting scapular mobility by binding the scapula to the thorax reduces subacromial space\textsuperscript{2}. However, the influence of specific scapular rotations that are theorized to increase the subacromial space, scapular upward rotation, posterior tilt and external rotation, have not been directly studied in vivo. The scapular assistance test (SAT) is a clinical method used to manually assist the scapula into upward rotation and posterior tilt during humeral elevation.\textsuperscript{29, 30, 47} The SAT can reliability increase scapular upward rotation and posterior tilt with active arm elevation at 45\textdegree{} and 90\textdegree{} scapular plane elevation and with the arm at rest\textsuperscript{49}, but the potential influence on scapular external rotation is unknown. In symptomatic patients with dyskinesis, the SAT is considered a corrective passive maneuver performed manually by an examiner to decrease pain\textsuperscript{31} presumably due to an increase subacromial space. It is unknown whether individuals with scapular dyskinesis would differ from individuals without dyskinesis in scapular motion or AHD with manual passive assistance of the scapula using the SAT.

The purposes of this study were to: 1) determine the influence of scapular dyskinesis on scapular kinematics and AHD, and 2) compare the influence of passive manual correction with the scapular assistance test (SAT) on AHD and 3D scapular kinematics between individuals with dyskinesis and individuals without dyskinesis. The hypothesis was 3D scapular kinematics and AHD would differ in asymptomatic participants with obvious scapular dyskinesis compared to participants without.
Additionally participants with scapular dyskinesis would demonstrate greater change in 3D scapular position and AHD with the SAT than participants without dyskinesis.

METHODS

Participants

Forty healthy participants (20 with dyskinesis; 20 with normal motion) free from shoulder or upper arm pain for at least 6 months participated in this study. The Virginia Commonwealth University Institutional Review Board approved the protocol for this study and subject consent was obtained prior to data collection. Individuals with symptoms produced by cervical spine motion, systemic connective tissue disease, a history of shoulder fracture, surgery, known shoulder pathology or shoulder pain within the last 6 months were excluded.

Participants with scapular dyskinesis were recruited for participation if they presented with obvious scapular dyskinesis as defined by 2 independent examiners using the scapular dyskinesis test. One examiner was a physical therapist with 15 years of orthopedic clinical experience and the other was a certified athletic trainer with 3 years of experience. Both examiners underwent standardized online training designed by the developers of the test. Participants without scapular dyskinesis (normal group), classified with normal scapular motion with the scapular dyskinesis test, were matched to those with obvious dyskinesis on the basis of age, gender, and arm dominance of shoulder tested (dominant; non-dominant). There were 11 females and 9 males who participated in each cohort. There were 11 participants with scapular dyskinesis in the
shoulder of the dominant arm and 9 in the non-dominant. The dominant arm was defined as the same side as the hand that the participant uses to write.

Using the scapular dyskinesis test, participants performed 5 repetitions of bilateral, active, weighted shoulder flexion (sagittal plane elevation) and shoulder abduction (frontal plane elevation). The amount of weight participants held was based upon body mass, 1.4 kg (3 lb) for those weighing less than 68.1 kg (150 lb) and 2.3 kg (5 lb) for those weighing 68.1 kg or more. Potential participants were asked to elevate their arms overhead as far as possible, thumbs pointing upward, to a 3-second count and then lower to a 3-second count while 2 independent examiners simultaneously observed scapulothoracic motion to individually classify scapular motion into one of three categories: obvious dyskinesis, subtle dyskinesis, and normal. Obvious dyskinesis was present when there was obvious medial or inferior angle winging of the scapula away from the thorax by at least an inch (2.54cm) or strikingly apparent dysrhythmia in at least 3 of 5 trials in weighted shoulder flexion or abduction. Subtle dyskinesis was present when there was mild or questionable evidence of abnormality, or abnormality was not consistently present in both flexion and abduction. Scapular motion was considered normal when there was no evidence of abnormality in at least one plane of motion and subtle or normal motion in the other plane. Participants classified independently by both examiners (100% agreement) with either obvious dyskinesis (DYSK group) or normal scapular motion (normal group) were enrolled in the study. In participants with bilateral dyskinesis, the shoulder with greater scapular winging or dysrhythmia was selected for
testing. Prior to initiating the study, a sample size of 20 participants per group was
determined to provide greater than 80% power to detect clinically meaningful difference
in AHD, defined as the upper boundary of the 90% confidence interval for the minimal
detectable change determined to be 2.0 mm with a common standard deviation of 2.0 mm
in a pilot study.50

**Instrumentation**

The Polhemus 3Space Fastrak electromagnetic-based motion capture system
(Polhemus, Colchester, VT) with a sampling rate of 30 Hz was used with Motion Monitor
software (Innovative Sports Training, Inc, Chicago, IL) to collect 3D kinematic data of
the scapula, humerus, and trunk. The electromagnetic tracking unit consists of a
transmitter, 4 receivers with one attached to a stylus used to digitize anatomical
landmarks, and a computer. The transmitter emits a magnetic field with a measurement
range of 0.45 - 2.4 m detected by receivers affixed to the thorax, scapula, and humerus.
The manufacturer reported accuracy of the electromagnetic tracking device is 0.8 mm
and 0.15°.1 This surface-based electromagnetic tracking method to track scapular motion
has been validated using bone pins.27

A diagnostic US unit, LogiQe (GE Healthcare, Wisconsin, USA) with a 4-12
mHz adjustable linear array transducer set at a frequency of 8.0 mHz was used to capture
images in B-mode for subacromial space measurement. The subacromial space was
defined as the AHD, the shortest linear distance between the humeral head and the
anterior inferior tip of the acromion, and was measured using onscreen calipers. This
measurement represents a linear measure of the anterior outlet of the subacromial space. US-generated AHD measures have demonstrated satisfactory reliability with intraclass correlation coefficients from 0.86-0.91 with the arm at rest, 45°, 60°, and 90° and concurrent validity with radiographs (r = 0.77-0.85)\textsuperscript{3,4}.

**3D Scapular Kinematic Data Collection**

Three electromagnetic sensors were attached to study participants using double-sided tape (3M Health Care, St Paul, MN) and further secured with CoverRoll (Beiersdorf, Norwalk, CT). One sensor was placed on the thorax over the spinous process of T3. The scapula sensor was placed over the broad flat surface of the posterior-lateral acromion and the humerus sensor was placed over the posterior aspect of each humerus distal to the triceps muscle belly, further secured with a Velcro strap. Anatomical landmarks on the subject’s thorax, scapula, and humerus were then palpated and digitized using the hand-held stylus following recommendations from the International Society of Biomechanics (ISB).\textsuperscript{60} Digitization allowed transformation of the sensor position and orientation into anatomically based position and orientation data of the humerus and scapula with respect to the thorax. Euler angle sequences for the humeral (Y-X’-Y”) and scapular (Y-X’-Z”) rotations were based on ISB recommendations.\textsuperscript{60} Scapular rotations (external rotation, upward rotation, and posterior tilt) occur around orthogonal axes as previously illustrated and defined.\textsuperscript{41,42} Scapular movements into the direction of upward rotation, posterior tilt and external rotation and are indicated by positive values. Because the scapula is internally rotated and anteriorly tilted in a resting position, these values are
negative and movement into external rotation and posterior tilt results in reduction of the values. Three-dimensional scapular kinematic data and US imaging of AHD were collected sequentially.

**US Imaging**

Participants were seated in an armless chair positioned in neutral posture attained by asking the subject to sit with feet flat, low back as far back as possible against the chair lumbar support. The 3D position of the probe for US imaging was standardized so that the anatomical landmarks of the humeral head and acromion were consistently identified. Specifically, we wanted to visualize the anterior aspect of the subacromial space since this has been identified as the predominant site of SAIS\textsuperscript{20,45,46}. The US probe was placed at the most anterior aspect of the acromion, identified by palpation, and with the long axis of the US transducer in the plane of the scapula, parallel to the flat surface of the acromion. Images were saved on the US scanner for later AHD measurements. AHD measures were made by a single examiner blinded to SAT condition and arm angle, but examiners were not blinded to group allocation.

**Testing Procedure**

The active arm elevation procedure was used to collect shoulder kinematic data and US imaging of anterior outlet of the subacromial space, with the order randomized by drawing. Participants were seated in an armless chair in neutral posture with the lumbar spine firmly approximating the chair back support. In neutral humeral rotation, the arm was positioned at rest, 45°, or 90° of active shoulder scapular plane elevation. The
scapular plane was defined as 30° anterior to the frontal plane verified with a goniometer. Arm elevation angle was verified with an inclinometer placed parallel to the long axis of the humerus. A second examiner performed the SAT while the first examiner captured an US image or manually triggered the SAT event during continuous scapular kinematic data collection. The SAT is typically performed with the examiner manually assisting the scapula into upward rotation and posterior tilt as the patient elevates the arm. In this study, the SAT maneuver was used as a method to alter scapular position and was applied to the scapula while the subject actively maintained a static arm position. Participants rested the upper extremity in a suspension harness between trials. Two consecutive trials were performed at each arm angle (rest, 45°, and 90°) under each condition (with and without SAT) with both the arm angle and condition randomized by drawing.

**Data Analysis**

Descriptive statistics were computed for all variables. Independent *t*-tests compared mean age, height, and body mass between participants with dyskinesis and normal scapular motion. Test-retest intra-rater reliability for scapular position and orientation, and AHD was calculated using an intraclass correlation coefficient (ICC$_{3, 2}$) analysis at each arm position using the method described by Shrout et al. To examine the effect of dyskinesis, we compared 3D scapular kinematics and AHD between groups using separate mixed-model analysis of variance (ANOVA) with factors of group (dyskinesis; normal), arm angle (rest, 45° and 90°), and interactions. To determine the effects of passive manual correction using the SAT on AHD and 3D scapular position, separate
2×2×3 mixed-model ANOVAs were used with effects of SAT (with SAT; without SAT), presence of dyskinesis (dyskinesis; normal), arm angle, and interactions. Statistical significance was set at $\alpha=.05$. Post hoc testing was performed with linear contrasts with the comparisons of interest to include the main effects of dyskinesis, SAT, and interactions of dependent variables examined at each arm angle. All analyses were performed using SAS Software (JMP 8.1; SAS Institute Inc, Cary, NC).

RESULTS

There were no significant differences in participant characteristics (p>0.05) between groups (TABLE 1). Obvious dyskinesis was identified in the dominant shoulder in slightly more than half (55%; n=11/20) the participants with dyskinesis. Intra-session test-retest reliability of the AHD measures performed during two separate trials of active arm elevation were excellent (ICC$_{3,2}$=0.88-0.93) at 45° and 90° scapular plane elevation and at rest (ICC$_{3,2}$=0.96). Intra-session test-retest reliability of 3D scapular kinematics in upward rotation, posterior tilt, and external rotation performed during two separate trials of active arm elevation were excellent (ICC$_{3,2}$=0.98-0.99). The reliability of the SAT to passively alter the position of the scapula between trials in upward rotation, posterior, and external rotation were excellent (ICC$_{3,2}$=0.97-0.99).

Effects of Scapular Dyskinesis

Scapular kinematics and AHD measures by group are shown in FIGURE 2. Examining the effects of dyskinesis on 3D scapular kinematics, there were no statistically significant
interactions for upward rotation ($p = 0.934$), posterior tilt ($p = 0.524$), external rotation ($p = 0.768$) or main effects of dyskinesis in either upward rotation ($p = 0.729$), posterior tilt ($p = 0.669$), or external rotation ($p = 0.943$). Examining the influence of dyskinesis on AHD, there was no significant interaction ($p = 0.491$) or main effect ($p = 0.754$) of dyskinesis on AHD. Thus participants with dyskinesis did not differ from participants classified as normal in 3D scapular kinematics or AHD with the arm at rest, 45°, or 90° of static active arm elevation. As such, mean scapular kinematics and AHD are shown in TABLE 2.

**Effects of Scapular Assistance Test**

Results of the $2 \times 2 \times 3$ mixed-model ANOVAs to compare the influence of manual correction using the SAT in participants with obvious DYSK and participants classified as normal are shown in TABLE 3, FIGURE 3, and FIGURE 4. With regard to the influence of manual assistance using the SAT on passive scapular upward rotation, the three-way interaction (group $\times$ arm angle $\times$ SAT condition) was not significant. Thus the influence of dyskinesis on upward rotation did not depend on arm angle and SAT condition. There was no significant two-way interaction of group by arm angle; however, there was a significant two-way interaction of group by SAT condition. Thus, the presence of dyskinesis influenced the effect of SAT on scapular upward rotation, regardless of arm angle. As shown in FIGURE 3, the participants with dyskinesis had a mean increase in scapular upward rotation of 10.0° (SE=1.1) with the SAT and participants without had a mean increase of 6.9° (SE=1.1); thus, participants with
scapular dyskinesis demonstrated a significantly greater increase in scapular upward rotation, 3.1° (SE=1.5), with the SAT in compared to participants without dyskinesis. There also was a significant two-way interaction of SAT condition by arm angle. Within both cohorts, the SAT induced scapular upward rotation with the arm at the rest by 13.2° (SE=1.3), with the arm at 45° by 7.3° (SE=1.3) and with the arm at 90° by 4.9° (SE=1.3).

For scapular posterior tilt, there was a significant main effect of SAT condition. Therefore, regardless of arm angle, manual assistance with the SAT increased scapular posterior tilt by 4.8° (SE=1.0), but did not differ between dyskinesis and normal groups. There were no significant three-way interactions, or two-way interactions of SAT condition by arm angle, group by SAT condition and group by arm angle. There was not a significant main effect of group on scapular posterior tilt; thus the presence of dyskinesis did not influence scapular posterior tilt at rest, 45°, or 90° of static arm elevation.

For passive external rotation, there was a significant two-way interaction of SAT condition by arm angle. There was a reduction in external rotation with the SAT at rest (mean decrease 6.5°, SE=0.7), at 45° (mean decrease 4.5°; SE=0.7), and at 90° (mean decrease 3.8°; SE=0.7) of scapular plane elevation. There was no significant three-way interaction, or two-way interaction of group by SAT condition and group by arm angle. There were no significant main effects of group on scapular external rotation; thus the presence of obvious DYSK did not influence scapular external rotation at rest, 45°, or 90° of static arm elevation.
Results for the influence of the SAT on AHD in participants with and without dyskinesis are shown in TABLE 2, and FIGURE 4. There were no significant three-way or two-way interactions of arm angle by SAT, group by SAT, or group by arm angle. Thus, the presence of dyskinesis did not influence AHD, regardless of SAT condition or arm angle. There was a significant main effect of SAT condition. The SAT increased AHD ($t=6.5, p <0.001$) by 1.4mm (SE=0.2), averaged across all arm angles and in both groups. In the entire cohort, mean increase in AHD was 1.8mm (SE=0.36) ($p<0.001$) and 1.6mm (SE=0.36) ($p<0.001$) with the SAT at 45° and 90°, respectively, as compared to without the SAT.

DISCUSSION

Scapular dyskinesis has been proposed to influence scapular motion and in turn subacromial space. Despite the presence of obvious dyskinesis, the 3D position of the scapula, and therefore AHD, was not altered compared to participants without dyskinesis. This conflicts with a prior study by Tate et al\textsuperscript{55} where athletes classified with obvious dyskinesis using the scapular dyskinesis test demonstrated altered 3D scapular kinematics with motion analysis during dynamic full elevation compared to athletes with normal scapular motion. This discrepancy between studies, and the lack of differences in 3D kinematics between participants with dyskinesis and those without in the current study can be attributed to multiple factors including differences in sample population, methods employed, and the potential multi-factored nature of dyskinesis.
In the study by Tate et al\textsuperscript{55}, participants were overhead athletes, many of whom had shoulder pain. Participants in the current study were not restricted to those participating in overhead athletics, and more importantly, were asymptomatic. The presence of pain may be a requisite to alterations in static 3D scapular kinematics in individuals with dyskinesis. Additionally, 3D scapular kinematics in the current study was evaluated during active arm elevation, unloaded, held statically in discrete positions of active arm elevation in the scapular plane which may not detect potential kinematic alterations observed dynamically during weighted full arm elevation in flexion or abduction. Lastly, scapular kinematics in asymptomatic individuals are highly variable\textsuperscript{36, 43}. Therefore, scapular kinematics in individuals with dyskinesis may also be variable. This variability may be attributed to the multi-factorial nature of dyskinesis. Multiple factors have been linked to abnormal scapular motion including decreased pectoralis muscle length\textsuperscript{10}, rotator cuff or shoulder muscle fatigue\textsuperscript{16, 18, 57}, thoracic position\textsuperscript{28}, muscle activity\textsuperscript{22}, and a loss of glenohumeral joint internal rotation range of motion\textsuperscript{8}. Not only can these factors potentially alter scapular kinematics differently, many may not affect scapular kinematics during static movements or at the discrete static angles (rest, 45° and 90°) of scapular plane elevation examined in this study.

With regard to AHD, individuals with obvious dyskinesis did not significantly differ in the AHD across arm positions which conflicts with conclusions of a prior study\textsuperscript{53}. In the current study we found participants with dyskinesis to have 0.4 mm less AHD at 45° and 90° of active elevation than participants without, which was not
This results in a 0.66 mm (SE=0.65) greater reduction in AHD from 0° to 45° in those with dyskinesis which also was not statistically significant (p=0.317). In comparison, Silva et al found a greater reduction in AHD (0.55mm) from the arm positioned at rest to 60° abduction in elite adolescent tennis players with dyskinesis (1.93mm reduction) compared to players without dyskinesis (1.38mm reduction)\(^5\), which was statistically significant (p=0.002). However, the clinical examination used to classify tennis players with scapular dyskinesis was not an established reliable method. Moreover, measurements of AHD were taken with the arm passively positioned at each angle and were not isolated to the anterior aspect of the subacromial space where outlet impingement has been shown to occur\(^12,20\). Prior research has shown that AHD measures taken without muscular activation differs from AHD during active abduction in healthy shoulders\(^23,24\) and does not reveal deficits found with active elevation in patients with SAIS\(^24\). Thus, it is important to evaluate AHD during active elevation to evaluate mechanisms related to scapular dyskinesis and SAIS.

Results of this study support the notion by Kibler et al\(^29,30\), that passive manual correction of the scapula with the SAT increases scapular upward rotation and posterior tilt which influences the subacromial space. The SAT may relieve rotator cuff or subacromial compression, to identify individuals with pain related to subacromial impingement. Interestingly, the SAT also reduced scapular external rotation, or increased scapular internal rotation, across all arm angles. Although internal rotation is theorized to reduce the subacromial space, the AHD in this study increased. Thus, scapular internal
rotation may have little influence on the reduction of subacromial space particularly in the presence of increased scapular upward rotation and posterior tilt.

There were no differences in scapular kinematics or AHD between groups in the effect of the SAT; however, there was greater scapular upward rotation induced with the SAT averaged across all arm angles in participants with dyskinesis (increase 10.0°, SE=1.1) compared to participants with normal scapular motion (increase= 6.9°, SE=1.1). The 3.1mm difference (SE of difference=1.5) in scapular upward rotation with the SAT between groups exceeds the minimal detectable change with 90% confidence, at 45° of active arm elevation; however, the clinical meaning of this finding is unclear.

Increased passive scapular upward rotation with the SAT in individuals with dyskinesis may be indicative of scapulothoracic joint hypermobility, increasing the demand for dynamic control of the scapular stabilizers including the serratus anterior and middle and lower trapezius muscles, thereby contributing to the presence of dyskinesis during loaded, dynamic arm elevation conditions. Increased scapular upward rotation with passive manual assistance of the SAT may be associated with increased soft tissue length or thoracic spine mobility in individuals with dyskinesis. Further study is warranted to determine whether this finding is unique to asymptomatic individuals with dyskinesis.

Limitations

We tested AHD and 3D scapular position at rest and active scapular plane elevation held statically at 45° and 90° in this study. Therefore, results may differ in other
planes of arm elevation, at other arm angles, or when tested dynamically through a full range of shoulder motion. We selected these discrete arm positions for a few reasons. SAIS is characterized by pain during elevation\textsuperscript{45, 46}, typically in an arc of motion where the greater tuberosity passes beneath and approximates the acromion\textsuperscript{21}. Several studies have shown that with humeral elevation greater than 90°, the rotator cuff tendons pass medially beyond the acromion so that they are no longer susceptible to outlet impingement within the subacromial space.\textsuperscript{12, 20, 21} The rotator cuff tendons have been shown to move beyond the anterior acromion at 60° of glenohumeral motion in patients following a rotator cuff repair.\textsuperscript{7} Therefore, testing with the arm at rest, 45° and 90° of active arm elevation provided information within the range of motion where outlet impingement of the rotator cuff tendons is most likely to occur.

Our measures of AHD captured with ultrasound are linear measures of the anterior outlet of the SA space. These do not take into account what may occur at other aspects of the subacromial space which are not typically involved in outlet impingement or changes to overall volume of the space. Lastly, 3D scapular kinematics were evaluated with electromagnetic tracking using surface sensors susceptible to skin artifact with and without passive scapular motion with the SAT. To account for this, we determined the magnitude of skin artifact induced with manual assistance using the SAT to be less than 2° in scapular upward rotation, posterior tilt, and external rotation compared to without SAT in pilot study\textsuperscript{49}. The mean change in scapular motion with the SAT found in this study is considerably greater than the error due to skin movement and
is therefore likely to represent true change. Additionally, we employed a correction factor that accounts for artifact due to skin with elevation without manual assistance derived from a validation study using bone-pin fixation.27 Thus, despite these limitations, the results of this study provide valuable information regarding scapular dyskinesis as a potential mechanism of SAIS.

CONCLUSIONS

Obvious scapular dyskinesis has little effect on scapular kinematics and AHD with the arm held in static positions in asymptomatic individuals. The SAT increased AHD, scapular posterior tilt and upward rotation, but decreased external rotation, regardless of the presence of dyskinesis. There was greater mobility into scapular upward rotation with passive assistance of the SAT in participants with dyskinesis than participants without dyskinesis, indicating potential scapular hypermobility with dyskinesis. Research is needed to determine whether scapular dyskinesis associated with pain differs with regard to 3D kinematics and AHD during dynamic and static active arm elevation.

KEY POINTS

The AHD and 3D scapular kinematics do not differ between participants with and without dyskinesis. Scapular dyskinesis, identified with clinical observation using loaded dynamic conditions, is not apparent during static arm elevation. This may be unique to
individuals without pain. The passive corrective maneuver of the SAT increased scapular upward rotation, posterior tilt, and AHD in all participants. Participants with dyskinesis demonstrate greater passive scapular mobility in upward rotation using the SAT, but not an additional increase in AHD.

**Implications**

Factors related to the presence of dyskinesis in asymptomatic individuals may not influence scapular kinematics in static positions. Pain, load or fatigue may be critical factors. The SAT increased acromiohumeral distance regardless of dyskinesis, and therefore, may be helpful identifying individuals where subacromial compression is producing symptoms.

**Caution**

Individuals with scapular dyskinesis in this study were asymptomatic. Therefore findings of this study cannot be generalized to patients with shoulder pain. Longitudinal study is necessary to determine whether individuals with scapular dyskinesis are more likely to develop pain.
REFERENCES


15. Desmeules F, Minville L, Riederer B, Cote CH, Fremont P. Acromio-humeral distance variation measured by ultrasonography and its association with the


45. Neer CS. Anterior acromioplasty for the chronic impingement syndrome in the


47. Rabin A, Irrgang JJ, Fitzgerald GK, Eubanks A. The intertester reliability of the

48. Roy JS, Moffet H, Hebert LJ, Lirette R. Effect of motor control and strengthening
exercises on shoulder function in persons with impingement syndrome: a single-

49. Seitz A, Michener L, Lynch S, Zirker C, Boardman N. Effects of scapular
assistance test on acromiohumeral distance measured in vivo with

posterior subacromial space changes with elevation measured in vivo using

51. Senbursa G, Baltaci G, Atay A. Comparison of conservative treatment with and
without manual physical therapy for patients with shoulder impingement


**TABLE 1.** Characteristics of participants classified with obvious dyskinesis (DYSK) and normal scapulothoracic motion (Normal) using the scapular dyskinesis test

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal (n=20)</th>
<th>With DYSK (n=20)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>26.7 (5.7)</td>
<td>26.6 (6.4)</td>
<td>0.980</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.73 (0.09)</td>
<td>1.76 (0.09)</td>
<td>0.277</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>71.4 (14.9)</td>
<td>70.8 (11.4)</td>
<td>0.904</td>
</tr>
</tbody>
</table>

*Values are means (standard deviations)*

*Abbreviations: DYSK, obvious dyskinesis*
TABLE 2. Three Dimensional Scapular Kinematics and Acromiohumeral Distance in All Participants (N=40)

<table>
<thead>
<tr>
<th>Arm Angle</th>
<th>Normal Mean</th>
<th>SE</th>
<th>95% CI</th>
<th>With DYSK Mean</th>
<th>SE</th>
<th>95% CI</th>
<th>Difference Mean</th>
<th>SE</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>-0.2</td>
<td>2.5</td>
<td></td>
<td>1.1</td>
<td>2.5</td>
<td></td>
<td>-1.3</td>
<td>3.5</td>
<td>-11.6, 8.9</td>
</tr>
<tr>
<td>45°</td>
<td>10.4</td>
<td>2.5</td>
<td>5.5 15.3</td>
<td>11.8</td>
<td>2.5</td>
<td>6.9 16.8</td>
<td>-1.4</td>
<td>3.5</td>
<td>-11.7, 8.9</td>
</tr>
<tr>
<td>90°</td>
<td>30.6</td>
<td>2.5</td>
<td>25.7 35.5</td>
<td>31.1</td>
<td>2.5</td>
<td>26.2 36.1</td>
<td>-0.5</td>
<td>3.5</td>
<td>-10.8, 9.8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Arm Angle</th>
<th>Normal Mean</th>
<th>SE</th>
<th>95% CI</th>
<th>With DYSK Mean</th>
<th>SE</th>
<th>95% CI</th>
<th>Difference Mean</th>
<th>SE</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>-9.5</td>
<td>1.7</td>
<td>-12.9 -6.0</td>
<td>-11.7</td>
<td>1.7</td>
<td>-15.1 -8.2</td>
<td>2.2</td>
<td>2.5</td>
<td>-5.0, 9.4</td>
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<tr>
<td>45°</td>
<td>-7.6</td>
<td>1.7</td>
<td>-11.0 -4.1</td>
<td>-7.4</td>
<td>1.8</td>
<td>-10.9 -3.8</td>
<td>0.2</td>
<td>2.5</td>
<td>-7.5, 7.1</td>
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<tr>
<td>90°</td>
<td>-8.2</td>
<td>1.7</td>
<td>-11.6 -4.7</td>
<td>-8.9</td>
<td>1.7</td>
<td>-12.4 -5.5</td>
<td>0.8</td>
<td>2.5</td>
<td>-6.5, 8.0</td>
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</table>

<table>
<thead>
<tr>
<th>Arm Angle</th>
<th>Normal Mean</th>
<th>SE</th>
<th>95% CI</th>
<th>With DYSK Mean</th>
<th>SE</th>
<th>95% CI</th>
<th>Difference Mean</th>
<th>SE</th>
<th>95% CI</th>
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<tbody>
<tr>
<td>Rest</td>
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<td>1.7</td>
<td>-34.0 -27.2</td>
<td>-30.3</td>
<td>1.7</td>
<td>-33.8 -26.9</td>
<td>0.3</td>
<td>2.4</td>
<td>-6.9, 7.4</td>
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<tr>
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<td>1.7</td>
<td>-37.8 -31.0</td>
<td>-34.4</td>
<td>1.7</td>
<td>-37.9 -31.0</td>
<td>0.0</td>
<td>2.4</td>
<td>-7.2, 7.2</td>
</tr>
<tr>
<td>90°</td>
<td>-36.0</td>
<td>1.7</td>
<td>-39.4 -32.5</td>
<td>-36.7</td>
<td>1.7</td>
<td>-40.1 -33.2</td>
<td>-0.7</td>
<td>2.4</td>
<td>-7.8, 6.5</td>
</tr>
</tbody>
</table>

**Scapular Upward Rotation (degrees)**

**Scapular Posterior Tilt (degrees)**

**External Rotation (degrees)**

**Acromiohumeral Distance (mm)**

* Abbreviations: DYSK, obvious scapular dyskinesis; SE, standard error; 95% CI, 95 percent confidence interval
TABLE 3. Summary of scapular kinematic and acromiohumeral distance results using 3-factor mixed model ANOVAs for the influence of passive manual assistance with the scapular assistance test in participants classified with obvious dyskinesis (DYSK) and normal.

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>UR</th>
<th>p-value</th>
<th>PT</th>
<th>p-value</th>
<th>ER</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>1, 38</td>
<td>0.7</td>
<td>0.422</td>
<td>0.2</td>
<td>0.667</td>
<td>0</td>
<td>0.998</td>
</tr>
<tr>
<td>Arm Angle</td>
<td>2, 190</td>
<td>432.0</td>
<td>&lt;0.001 *</td>
<td>15.2</td>
<td>&lt;0.001</td>
<td>40.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Group x Arm Angle</td>
<td>2, 190</td>
<td>0.4</td>
<td>0.657</td>
<td>0.8</td>
<td>0.432</td>
<td>0.1</td>
<td>0.900</td>
</tr>
<tr>
<td>SAT condition</td>
<td>1, 190</td>
<td>126.7</td>
<td>&lt;0.001 *</td>
<td>74.1</td>
<td>&lt;0.001</td>
<td>143.0</td>
<td>&lt;0.001</td>
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<tr>
<td>Group x SAT condition</td>
<td>1, 190</td>
<td>4.4</td>
<td>0.038</td>
<td>0.0</td>
<td>0.908</td>
<td>0.1</td>
<td>0.705</td>
</tr>
<tr>
<td>Arm Angle x SAT condition</td>
<td>2, 190</td>
<td>10.8</td>
<td>&lt;0.001 *</td>
<td>0.8</td>
<td>0.462</td>
<td>3.7</td>
<td>0.026</td>
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<tr>
<td>Group x Arm Angle x SAT condition</td>
<td>2, 190</td>
<td>0.1</td>
<td>0.889</td>
<td>0.1</td>
<td>0.912</td>
<td>0.1</td>
<td>0.880</td>
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### Acromiohumeral Distance

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>UR</th>
<th>p-value</th>
<th>PT</th>
<th>p-value</th>
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<th>p-value</th>
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<tbody>
<tr>
<td>Group</td>
<td>1, 38</td>
<td>0.5</td>
<td>0.466</td>
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<tr>
<td>Arm Angle</td>
<td>2, 189</td>
<td>46.1</td>
<td>&lt;0.001 *</td>
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<td></td>
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<tr>
<td>Group x Arm Angle</td>
<td>2, 189</td>
<td>1.5</td>
<td>0.222</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>SAT condition</td>
<td>1, 189</td>
<td>42.5</td>
<td>&lt;0.001 *</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Group x SAT condition</td>
<td>1, 189</td>
<td>0.7</td>
<td>0.390</td>
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<td></td>
<td></td>
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<tr>
<td>Arm Angle x SAT condition</td>
<td>2, 189</td>
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<td>0.066</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Group x Arm Angle x SAT condition</td>
<td>2, 189</td>
<td>0.0</td>
<td>0.958</td>
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<td></td>
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Abbreviations: UR, upward rotation; PT, posterior tilt; ER, external rotation. * statistically significant.
Figure 1. Participant classified with obvious scapular dyskinesis during the weighted flexion task of the scapular dyskinesis test.
Scapular Kinematics and Acromiohumeral Distance

A. Scapular Upward Rotation

B. Scapular Posterior Tilt

C. Scapular External Rotation

D. AHD

Figure 2. Mean scapular kinematics and acromiohumeral distance (AHD) in individuals with scapular dyskinesis (DYSK) and without (Normal). Error bars represent standard error of the mean. Scapular (A) upward rotation (positive values = upward rotation), (B) posterior tilt (positive values = posterior tilt), (C) external rotation (positive values = external rotation), and (D) AHD. Values for scapular posterior tilt and external rotation are negative and remain negative because the scapula is anteriorly tilted and internally rotated at each arm position. Asterisk (*) indicates significant difference between with and without SAT.
Scapular Upward Rotation

A. Normal

B. With DYSK

Scapular Posterior Tilt

C. Normal

D. With DYSK

Scapular External Rotation

E. Normal

F. With DYSK
**Figure 3.** Mean scapular kinematics with and without passive alteration with the scapular assistance test (SAT) in individuals with scapular dyskinesis (DYSK) and without (Normal). Error bars represent standard error of the mean. Scapular upward rotation (positive values= upward rotation) in (A) normal and (B) DYSK, posterior tilt (positive values= posterior tilt) in (C) normal and (D) DYSK, and external rotation (positive values= external rotation) in (E) normal and (F) DYSK. Values for posterior tilt and external rotation are negative and remain negative because the scapula is anteriorly tilted and internally rotated at each arm position. Asterisk (*) indicates significant difference between with and without SAT.
**Figure 4.** Acromiohumeral distance with and without passive alteration with the scapular assistance test (SAT). Error bars represent the standard error of the mean. Participants with (A) normal scapular motion with the scapular dyskinesis test, and (B) obvious scapular dyskinesia with the scapular dyskinesia test. Asterisk (*) indicates significant difference between with and without SAT.
CHAPTER 5: CONCLUSION OF DISSERTATION

The purpose of this dissertation research was to examine the mechanisms associated with rotator cuff disease, specifically rotator cuff tendinopathy. The etiology of rotator cuff tendinopathy is multi-factored and includes various intrinsic and extrinsic mechanisms, evident in the review of literature in Chapter 2. Despite varied mechanisms, patients with rotator cuff tendinopathy are often treated with homogenous treatment regimes (Kuhn, 2009) that do not regard potential differences in contributing mechanisms. Furthermore, randomized controlled trials which compare surgery to rehabilitation indicate not all patients benefit from treatment (Brox et al., 1999; Brox, Staff, Ljunggren, & Brevik, 1993; Haahr & Andersen, 2006; Haahr et al., 2005) with more than a third reporting continued pain and disability (Brox et al., 1999; Brox et al., 1993). A better understanding the mechanisms of rotator cuff disease is paramount to improving treatment outcomes.

Altered scapular kinematics is considered an extrinsic mechanism of rotator cuff disease theorized to reduce the subacromial space leading to subacromial impingement (Ludewig & Cook, 2000). A linear measure of the subacromial space is the acromiohumeral distance (AHD). There is evidence of altered scapular kinematics in patients with subacromial impingement; thus, scapular strengthening and stretching exercises have been advocated as an important component of a rehabilitation
Despite this, no study has examined the influence that specific scapular rotations have on subacromial space in this population. The scapular assistance test is proposed to passively increase scapular upward rotation and posterior tilt, specific scapular motions theorized to increase subacromial space and alter shoulder strength.

This dissertation research focused on studying extrinsic mechanisms of rotator cuff disease, specifically the relationship of scapular kinematics on subacromial space. Chapter 3 of this dissertation research focuses on the influence that a passively induced alteration in scapular position has on scapular kinematics, the AHD, and shoulder strength in individuals with subacromial impingement using the scapular assistance test. Results suggest both scapular kinematics and the AHD can be altered with passive manual correction using the scapular assistance test, but not shoulder strength. Furthermore, the influence of the scapular assistance test did not differ between participants with subacromial impingement and control participants. Additionally, scapular posterior tilt is likely more beneficial than upward rotation to increase AHD, as there was a negative relationship between upward rotation and AHD; the greater the increase in scapular upward rotation the smaller the increase in AHD at 90° of humeral elevation.

Chapter 4 of this dissertation research provided results of the influence that scapular dyskinesis and passive manual correction with the scapular assistance test has on subacromial space and 3D scapular kinematics. Scapular dyskinesis has been demonstrated in both symptomatic and asymptomatic overhead athletes with impingement. (McClure, Tate, Kareha, Irwin, & Zlupko, 2009; Silva, Hartmann, Laurino,
Furthermore, prior research suggests scapular dyskinesis reduces subacromial space in athletes and may contribute to the high prevalence of shoulder pain in this population. (Silva et al., 2010) Results of the second part of this dissertation research suggest the presence of obvious scapular dyskinesis, as defined by the scapular dyskinesis test, in asymptomatic individuals did not result in altered scapular kinematics or AHD. The AHD and 3D scapular kinematics did not differ between participants with and without dyskinesis. Scapular dyskinesis, identified with clinical observation using loaded dynamic condition of the scapular dyskinesis test, is not apparent during static arm elevation. This may be unique to individuals without pain. The passive corrective maneuver of the SAT increased scapular upward rotation, posterior tilt, and AHD in all participants, regardless of dyskinesis.

Clinical Implications

There are clinical implications to the findings of this dissertation research. First, interventions to alter resting scapular position and kinematics by inducing scapular posterior tilt in patients with subacromial impingement may be beneficial. However the focus of manual therapy or scapular exercise techniques in patients with subacromial impingement should be scapular posterior tilt which appears to have an influence on AHD at higher positions of arm elevation, in comparison to scapular upward rotation. Factors related to the presence of dyskinesis in asymptomatic individuals may not influence scapular kinematics in static positions. As such pain may be a critical factor. The SAT did increase the acromiohumeral distance regardless of dyskinesis, and
therefore, this passive maneuver may be helpful identifying individuals where subacromial compression is producing symptoms.

Future Research

Further research is warranted to explore the relationships of pain and pain response with the scapular assistance test on changes in 3D scapular kinematics corresponding alterations in subacromial space in patients with impingement. This may determine the clinical utility of this test to identify individuals for whom subacromial compression contributes to symptoms and identify the minimal clinical important change in the acromiohumeral distance associated with a clinically meaningful reduction in pain. Additionally, scapular dyskinesis in patients with and without pain may differ in 3D scapular kinematics in static and dynamic arm elevation conditions. Understanding differences between these populations may be helpful in developing prevention programs.

Longitudinal study is warranted to determine whether scapular dyskinesis is a risk factor to increasing the prevalence of shoulder pain. Additionally, further research that identifies subgroups of patients with rotator cuff disease, specifically, subacromial impingement syndrome, based on clusters of examination findings such as the presence of obvious dyskinesis is paramount to improving treatment outcomes. This approach has been used in the treatment of individuals with low back pain and has resulted in superior outcomes when compared to a general treatment.(Fritz, Delitto, & Erhard, 2003) Lastly, future research is needed on the various intrinsic mechanisms of rotator cuff disease, such as alterations in muscle and tendon characteristics, as these factors may interplay with
extrinsic factors. Viewing extrinsic and intrinsic mechanisms independently may not fully elucidate our understanding of the disease and limit success of improving treatment strategies.
References


