The Mechanisms of Posterior Shoulder Tightness and Effectiveness of Manual Therapy

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THE MECHANISMS OF POSTERIOR SHOULDER TIGHTNESS AND EFFECTIVENESS OF MANUAL THERAPY

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DEDICATION

I would like to dedicate this dissertation to my family and friends who have supported me along this journey. I am forever thankful and indebted to my advisors, Charles Thigpen, PhD, PT, Ellen Shanley, PhD, PT, Paul Beattie, PhD, PT, Stacy Fritz, PhD, PT, Richard Hawkins MD and David Kwartowitz PhD, who's friendship and expertise will always be cherished. I would also like to thank the faculty and staff at the University of South Carolina and department Exercise Science department for all their support. Finally, I am forever grateful to my mother, Brenda, my family, and friends whose patience, love, and support, made this experience possible.
ACKNOWLEDGEMENTS

Thank you to all the players, parents, coaches and athletic training staffs that participated in this study. I would also like to recognize the Greenville Hospital System and Hawkins Foundation for providing administrative support, and the in-kind support from Sonosite Inc., Carpal Therapy Inc, and Thera-band Inc. This dissertation was partially sponsored by the Legacy Fund of the American Physical Therapy Association’s Sports Section.
ABSTRACT

Posterior shoulder tightness is a common physical impairment in overhand baseball athletes presenting with injury. The etiology of this physical impairment is poorly understood and theorized to be a combination of bony, muscular, and inert soft-tissue contributions occurring at the glenohumeral joint. The ability to discriminate between each tissue’s influences on shoulder range of motion is often challenging to overcome within a clinical environment. Chapter 2 of this manuscript provides a thorough review of the literature discussing the potential mechanisms of posterior shoulder tightness. Previous studies have independently accounted for the relationships between posterior shoulder tightness, and the mechanical contributions of bony anatomy and capsuloligamentous stability. Chapter 3 of this dissertation research is a clinical commentary that discusses the current uses of rehabilitative shoulder ultrasound imaging including specific functions to account for the theorized mechanisms of posterior shoulder tightness.

In overhead throwing athletes, the range of motion deficits of posterior shoulder tightness have been linked to increased prospective injury risk. Therapeutic treatment interventions have shown a promising ability to improve some of these motion deficits although programs are often not tailored to target specific tissues. Chapter 4 is a randomized controlled trial comparing the acute treatment effectiveness of a muscle-directed manual therapy intervention and
posterior shoulder stretching routine versus stretching alone. The results indicate that the added use of muscle-directed manual therapy significantly enhances the amount of ROM gained when compared to stretching alone. These clinical data suggest that musculotendinous stiffness influences the deficits associated with posterior shoulder tightness.

In Chapter 5, we specifically examine the local physiologic contributions of humeral morphology, glenohumeral joint translation, and rotator cuff stiffness with the resolution of posterior shoulder tightness. Of these potential mechanisms, rotator cuff stiffness was the only tissue responsive to the application of muscle-directed manual therapy. Furthermore, the decreases observed in muscle stiffness were concurrent with the supplemental gains in shoulder ROM. These findings indicate that manual therapy treatment directly applied to the rotator cuff is effective at decreasing muscle stiffness and reducing deficits in posterior shoulder tightness.

The results of this dissertation research suggest that rotator cuff stiffness is partially responsible for the presence of posterior shoulder tightness and that muscle-directed manual therapy is effective at decreasing dominant sided deficits in shoulder ROM. Further research is required to determine the potential long-term effects of muscle-directed manual and stretching for the injury prevention and treatment of athletes with posterior shoulder tightness.
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CHAPTER 1: INTRODUCTION

1.1. Problem and Study Significance

Arm injuries in baseball are common, costly, and debilitating being primarily attributed to mechanisms of overuse. Twenty million athletes under the age of 21 play baseball with 45-70% reporting a history of arm pain or injury (Collins and Comstock 2008; Shanley, Michener et al. 2011). Increased age, weight lifting, playing outside the league, pitching with arm fatigue, and the aggregate number of pitches thrown per season have been associated with arm injuries, supporting the theory that these injuries are related to mechanisms of overuse (Lyman, Fleisig et al. 2001; Lyman S, Fleisig GS et al. 2002). The nature of overuse injuries implies that the development of these conditions is preventable; however, there is little empirical evidence to guide in the therapeutic treatment for addressing known risk factors that are associated with these injuries.

Baseball athletes commonly exhibit altered patterns of passive range of motion (ROM) between shoulders. These differences are thought to result from the high mechanical stresses placed on the tissues during repetitive throwing (Borsa, Laudner et al. 2008). The dominant (throwing) shoulder in these athletes commonly displays an increase in external rotation (ER) and humeral retrotorsion. These differences are often concurrent with decreased internal rotation (IR) and horizontal adduction (HA) when compared to their non-dominant
arm (Ellenbecker TS, Roetert EP et al. 2002). While altered shoulder ROM appear to be adaptive changes, when excessive these alterations may become risk factors for injury (Shanley, Michener et al. 2011). In particular, posterior shoulder tightness (PST) has been associated with shoulder pain (Burkhart and Morgan 2001; Laudner, Myers et al. 2006) and activities decreasing that posterior shoulder tightness with resolution of throwing related pain (Tyler, Nicholas et al. 2009). Posterior shoulder tightness has also been identified as a predisposing factor for prospective arm injury (Shanley, Rauh et al. 2011; Wilk, Macrina et al. 2011).

Previous studies have shown that individuals participating in stretching programs targeting PST (loss of internal rotation and horizontal adduction) are amenable to changes that appear to decrease injury risk (Kibler and Chandler 2003; Laudner, Sipes et al. 2008; Tyler, Nicholas et al. 2009). A limiting factor in these studies is that they included a wide range of individuals who did not necessarily display PST. Also, these investigators did not examine the mechanisms contributing to PST nor the interventions used to treat PST. Currently, there is no published research examining the effectiveness of manual therapy interventions in resolving PST. Clear evidence is needed to justify the use of these interventions in an “at risk” population while also elucidating the underlying mechanisms behind their application.

1.2. Theoretical Framework

Injury risk in baseball players has been related to PST, which often
manifests as marked decreases in HA and total arc of motion (TARC = ER + IR) loss with primary deficits in IR (Burkhart and Morgan 2001; Myers, Laudner et al. 2006; Shanley, Rauh et al. 2011; Wilk, Macrina et al. 2011). Capsular restrictions, musculotendinous stiffness and osseous adaptations have been identified as possible mechanical adaptations in these athletes secondary to the high and repetitive stresses incurred during throwing (Burkhart SS, Morgan CD et al. 2000; Crockett, Gross et al. 2002; Burkhart SS, Morgan CD et al. 2003). Currently, however, there is a lack of consensus regarding which of these structures is most responsible for alterations in ROM. It is likely that there are contributions from each, which may vary between individuals. The ability to differentiate between these anatomical variants may better guide in the identification and management of these athletes to reduce the overall rate of arm injury.

Rotational humeral ROM is governed by both soft tissue restraints (rotator cuff & capsuloligamentous) and osseous morphology (humeral torsion). The exact influence and contributions of these structures is unknown. Observational study of injured overhead athletes has shown that these individuals display a decreased TARC on the dominant arm (as compared with the non-dominant arm) with a predominant loss in IR (Myers, Laudner et al. 2006). While these data would indicate that soft-tissue adaptations are responsible for these differences, the study failed to account for the influence of humeral morphology, leaving no clarification of causal relationship. Currently, there are few studies to elucidate the exact mechanisms behind the deficits in humeral ROM; thus, further strides
are needed to understand these relationships.

The stresses of throwing are thought to retard the natural development of humeral antetorsion that occurs during skeletal maturation. As a result, overhead throwers have displayed substantially decreased values of humeral torsion on their dominant arm when compared to their non-dominant side, a clinical finding known as humeral retrotorsion. It is thought that increased humeral retrotorsion results in a shift in total arc of motion such that the dominant arm exhibits a gain in ER equivocal to the loss in IR, thereby retaining the same total arc of motion as compared to the contralateral side. Adaptive capsular and soft-tissue changes in throwers in addition to this bony adaptation are thought to exist when a dominant arm loss in IR exceeds the gain in ER when compared bilaterally,(Wilk, Meister et al. 2002; Shanley, Rauh et al. 2011) however, there is little evidence to substantiate these relationships in athletes with PST (Laudner, Meister et al. 2012).

The posterior rotator cuff (infraspinatus / teres minor) is crucial to the overall function of the shoulder. The primary role of these muscles is to aid in the balance of glenohumeral joint force couples by providing joint concavity-compression as well as an inferiorly directed force on the humeral head to maintain joint congruency (Oyama, Myers et al. 2010). During the throwing motion these muscles serve as a primary decelerator of the upper extremity by resisting internal rotation and horizontal adduction at ball release (Laudner KG, Stanek JM et al. 2006; Myers, Oyama et al. 2007). This repetitive eccentric loading is thought to result in spontaneous shortening of the connective tissue
and increase passive muscle stiffness that ultimately results in reduced
glenohumeral range of motion (Oyama, Myers et al. 2010). Currently, however,
little knowledge is available to guide investigators in the isolated quantification of
posterior rotator cuff muscle stiffness. Further research is needed to identify the
intrinsic soft-tissue contributions of the posterior rotator cuff on influencing
shoulder range of motion and performance.

While rotational shoulder ROM in overhead athletes has been well
documented, little evidence exists describing in-vivo characteristics of
translational glenohumeral movement. Translational, or ‘accessory’,
glenohumeral joint movement is considered to be obligate for overhead athletes
to achieve full range of motion and optimal performance. This motion is
commonly referred to clinically as joint laxity (Borsa, Laudner et al. 2008). The
operational definition of joint laxity for the shoulder is “the degree of humeral
head displacement relative to the glenoid that occurs following the application of
a small force” (Hawkins, Schutte et al. 1996). Current evidence suggests that
there is a wide range of ‘normal’ joint laxity. Despite widespread belief that
throwing athletes acquire hyperlaxity at the anterior glenohumeral joint, the
results of in-vivo studies are often conflicting when compared across the
literature(Ellenbecker, Mattalino et al. 2000; Sethi, Tibone et al. 2004; Borsa,
Wilk et al. 2005; Borsa, Laudner et al. 2008; Laudner, Meister et al. 2012). Thus,
it is inconclusive whether the mechanical adaptations of throwing influence the
magnitude of the translational glenohumeral movement. Furthermore, there is
sparse evidence available examining the effectiveness of therapeutic
interventions on influencing glenohumeral joint translation (Manske, Meschke et al. 2010). The only study available observing the treatment effects of joint mobilizations and stretching in baseball players with posterior shoulder tightness suggests there to be no added clinical benefits to ROM for providing joint mobilizations. Therefore, future studies are needed to explore the best available treatment options for reducing ROM deficits in baseball players with posterior shoulder tightness.

1.3. Purpose of Research

This research proposes a single-blinded randomized clinical trial to examine the effectiveness of therapeutic interventions and to determine the mechanisms by which they act in asymptomatic throwers with PST. Our goal is to measure changes in humeral torsion (HT), anteroposterior (A/P) glenohumeral translation, rotator cuff muscle stiffness and shoulder ROM following the acute application of instrumented soft-tissue mobilizations and/or supervised posterior shoulder stretching. Sixty skeletally mature baseball players displaying PST will be matched by age and hand dominance. Subjects will be randomized into one of two treatment groups, receiving either posterior shoulder stretching and instrumented soft-tissue mobilizations (n = 30), or posterior shoulder stretching alone (n = 30). This research design will improve the current understanding regarding the mechanical treatment effects of a combined soft-tissue and selective tissue-stretching regimen compared to posterior shoulder stretching alone when applied to an “at risk” throwing population.
Differences in passive glenohumeral range of motion will be assessed between intervention groups to determine the treatment effects of these interventions on posterior shoulder tightness. Ultrasound imaging will be used to account for HT differences between sides (dominant and nondominant). We will also measure differences in passive accessory A/P joint translation with the use of a electromagnetic kinematic tracking system (Sethi, Tibone et al. 2004), and observe any changes in rotator cuff muscle stiffness with the use ultrasound elastography.

1.4. Specific Aims and Hypotheses

Specific Aim 1. To compare clinical measures of passive shoulder ROM (IR, ER, TARC, and HA) between throwers with PST receiving instrumented manual therapy and posterior shoulder stretching to those performing only supervised posterior shoulder stretches.

Aim (1.A). To **compare total arc of humeral rotation (ER + IR) of both groups** before and after the application of instrumented soft-tissue mobilizations and/or posterior shoulder stretching while accounting for humeral torsion.

   **Hypothesis (1.A).** Each group will exhibit statistically significant increases in total arc of motion (ER + IR) on the internal rotation side when compared to baseline.

Aim (1.B). To **compare glenohumeral horizontal adduction (HA) of both groups** before and after the application of instrumented soft-tissue mobilizations and/or posterior shoulder stretching when accounting for humeral torsion.
Hypothesis (1.B). Both treatment groups will exhibit statistically significant increases in HA ROM when compared to baseline measures.

Aim (1.C). To compare the external rotation (ER) ROM of both groups before and after the application of instrumented soft-tissue mobilizations and/or posterior shoulder stretching when accounting for humeral torsion.

Hypothesis (1.C). No significant differences in ER rotation will be demonstrated in either treatment group following the intervention.

Aim (1.D). To compare the total arc of motion (ER + IR) between groups following the application of instrumented soft-tissue mobilizations and/or posterior shoulder stretching when accounting for humeral torsion.

Hypothesis (1.D). Individuals receiving instrumented soft-tissue mobilizations and posterior shoulder stretching will display an increased total arc of motion (ER + IR) on the internal rotation side when compared to those performing supervised posterior shoulder stretching alone.

Aim (1.E). To compare the glenohumeral HA between groups following the application of instrumented soft-tissue mobilizations and/or posterior shoulder stretching when accounting for humeral torsion.

Hypothesis (1.E). Individuals receiving instrumented soft-tissue mobilizations and posterior shoulder stretching will exhibit more HA when compared to those performing supervised posterior shoulder stretching alone.
Specific Aim 2. To compare the underlying mechanisms that contribute to PST including humeral morphology, posterior rotator cuff stiffness, and glenohumeral joint translation.

Aim (2.A). To compare glenohumeral joint translation of both groups following the application of posterior shoulder stretching and instrumented soft-tissue mobilizations to posterior shoulder stretching alone.

Hypothesis (2.A). Following the indicated treatment interventions both groups will not exhibit statistically significant increases in glenohumeral joint translation.

Aim (2.B). To compare posterior rotator cuff stiffness of both groups following the application of posterior shoulder stretching and instrumented soft-tissue mobilizations to posterior shoulder stretching alone when accounting for humeral torsion.

Hypothesis (2.B). Following the application of both treatment interventions each group will exhibit statistically significant decreases in posterior rotator cuff stiffness when measured by ultrasound elastography.

Aim (2.C). To compare glenohumeral joint translation between groups following the application of posterior shoulder stretching and instrumented soft-tissue mobilizations to posterior shoulder stretching alone when accounting for humeral torsion.

Hypothesis (2.C). No differences in glenohumeral joint translations will be apparent between groups as soft-tissue mobilizations are not thought to influence capsuloligamentous tissue.
Aim (2.D). To **compare posterior rotator cuff stiffness between groups** following the application of posterior shoulder stretching and instrumented soft-tissue mobilizations to posterior shoulder stretching alone when accounting for humeral torsion.

**Hypothesis (2.D).** The individuals receiving instrumented soft-tissue mobilizations in addition to posterior shoulder stretching will demonstrate greater decreases in posterior rotator cuff stiffness when compared to those receiving supervised posterior shoulder stretching alone.

1.5. Clinical Implications

The results of this study will elucidate the mechanisms of PST by concurrently examining the osseous, muscular, and capsuloligamentous restraints of shoulder ROM. This will begin to provide a better understanding behind the mechanisms thru which therapeutic interventions influence shoulder ROM and identify the underlying physiologic impairments that relate to arm injury. This information may be used clinically to improve therapeutic outcomes, selectively identify appropriate treatment interventions, and better prognosticate patient outcomes in those with PST.

1.6. Limitations and Assumptions

The following limitations and assumptions apply to this study design:

1. Glenohumeral joint ROM is influenced by a combination of osseous, muscular, and inert-soft-tissue contributions.
2. Individuals 15 years or older are skeletally mature and demonstrate stable measures of HT.

3. Ultrasound imaging calculations of HT correspond to the actual degree of humeral torsion.

4. Ultrasound elastography calculations of rotator cuff stiffness are representative of true rotator cuff stiffness.

5. Passive accessory joint glides are a valid measure of capsular restraint.

6. The electromagnetic kinematic tracking system provides true quantitative assessment of glenohumeral translation.

1.7. Delimitations

1. 60 male baseball players (ages 15 years and up) were recruited from local high schools and colleges from the Greenville/Spartanburg SC area.

2. All subjects exhibited posterior shoulder tightness and had no activity limiting pain within 3 months of testing. This includes participation in all practices and games without modification of playing status or position based on symptoms (i.e. pitching versus first base etc.).

3. Ultrasound imaging was utilized to calculate the influence of humeral retrotorsion on measures of shoulder ROM.

4. A/P translation of the glenohumeral joint was measured using an electromagnetic tracking system.

5. Ultrasound elastography was used as an indirect method of calculating in vivo tissue stiffness.
6. Ultrasound imaging was used as an indirect method to estimate HT.

1.8. Power Analysis

Power was calculated based on the preliminary pilot study of 8 baseball players with PST (TARC, IR, and HA deficit > 15°). Considering the values of presented Table 1, a moderate to large effect size (Cohen’s d) was be observed with the combined treatments of ISTM and stretching (n = 4) when compared with players stretching only (n = 4). This allows for clinically relevant conclusions to be drawn for any of the observed differences among tissue mechanisms including; humeral torsion, A/P translation, and rotator cuff stiffness. These power calculations assume a 2-tailed Type I error rate of .05 based on the independent samples t-test of ROM gained between groups (ROM Gain = Posttest ROM – Pretest ROM) for selected paired comparisons. Horizontal adduction displayed the greatest variability therefore, a priori power calculations estimated the effect size to be d = 0.61, with a corresponding Power of 0.62 (alpha = 0.05). This resulted in an estimated sample size of 44 participants (n = 22 in each group) (Faul, Erdfelder et al. 2009). These data suggests that a conservative sample size of 60 (n = 30 for each group) would provide sufficient power to detect differences for all dependent ROM variables between groups for all research questions TABLE 1.1.
TABLE 1.1. ESTIMATED STUDY POWER (N = 60)

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Range of Motion</th>
<th>Mean Difference</th>
<th>Stdev</th>
<th>Power</th>
<th>Effect Size (d)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Arc of Motion Gain</strong></td>
<td>9.2°</td>
<td>4.8°</td>
<td>0.83</td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td><strong>Internal Rotation Gain</strong></td>
<td>8.2°</td>
<td>5.8°</td>
<td>0.81</td>
<td>0.70</td>
<td></td>
</tr>
<tr>
<td><strong>Horizontal Adduction Gain</strong>*</td>
<td>7.1°</td>
<td>5.0°</td>
<td>0.62</td>
<td>0.61</td>
<td></td>
</tr>
<tr>
<td><strong>Mechanisms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Humeral Torsion Gain</td>
<td>-0.6°</td>
<td>2.1°</td>
<td>0.18</td>
<td>0.33</td>
<td></td>
</tr>
<tr>
<td>Total A/P Translation Gain</td>
<td>-0.2 cm</td>
<td>&lt;0.1 cm</td>
<td>0.32</td>
<td>0.35</td>
<td></td>
</tr>
<tr>
<td>Anterior Translation Gain</td>
<td>-0.2 cm</td>
<td>&lt;0.1 cm</td>
<td>0.31</td>
<td>0.34</td>
<td></td>
</tr>
<tr>
<td>Posterior Translation Gain</td>
<td>-0.1 cm</td>
<td>&lt;0.1 cm</td>
<td>0.17</td>
<td>0.20</td>
<td></td>
</tr>
<tr>
<td>Rotator Cuff Stiffness Loss</td>
<td>0.3 kPa</td>
<td>0.1 kPa</td>
<td>0.63</td>
<td>0.58</td>
<td></td>
</tr>
</tbody>
</table>

*An estimated Sample Size of 44 was calculated based on the relative Power and Effect Size of Horizontal Adduction
1.9. References


CHAPTER TWO: LITERATURE REVIEW

2.1. Introduction

Previous studies have determined that injured baseball athletes often exhibit clinical deficits in glenohumeral range of motion (Myers, Laudner et al. 2006; Dines, Frank et al. 2009; Shanley, Rauh et al. 2011; Wilk, Macrina et al. 2011). Currently, there is a lack of consensus among investigators regarding the specific tissue(s) responsible for these alterations in glenohumeral motion. Researchers have attributed these differences to thickened posterior inferior joint capsule (Thomas, Swanik et al.; Tyler, Nicholas et al. 2000; Burkhart SS, Morgan CD et al. 2003), posterior rotator cuff tightness, (Myers, Laudner et al. 2006), and osseous adaptations (Crockett, Gross et al. 2002; Osbahr, Cannon et al. 2002; Reagan KM, Meister K et al. 2002; Ruotolo, Price et al. 2006; Myers, Oyama et al. 2009). Considering the location of suspected mechanisms, these deficits commonly fall into the broad category of posterior shoulder tightness (PST) and will be referred to as such for the purposes of this manuscript.

Therapeutic interventions have shown promising ability to improve clinical measures of PST (McClure, Balaicuis et al. 2007; Manske, Meschke et al. 2010; Maenhout, Van Eessel et al. 2012) and concomitant pain however, research has failed to establish a clear link in identifying the specific tissues responsible for these deficits, along with the specific populations that respond to conservative treatment. Given the high incidence of arm injuries among these athletes and
the lack of knowledge regarding the development of PST, further research is required to better understand the mechanisms that contribute to this condition. An enhanced understanding of the mechanical contributions of PST will help guide clinicians in the selection and application of focused treatment interventions in effort to improve clinical outcomes.

2.2. Passive Shoulder Range of Motion

The mechanical demands placed on the throwing shoulder of baseball athletes have been speculated to contribute significantly to alterations and adaptations in shoulder range of motion (ROM). Previous research examining the side-to-side differences in overhead athletes supports this theory as throwing athletes often exhibit increased external rotation (ER), decreased internal rotation (IR), and loss of horizontal adduction (HA) on the dominant side (Ellenbecker, Roetert et al. 2002; Myers JB, Laudner KG et al. 2006; Tokish JM, Curtin MS et al. 2008). Total arc of rotation (TARC) is typically preserved and equal bilaterally in healthy baseball athletes, however it is often shifted towards ER when compared to the non-dominant side (Ellenbecker, Roetert et al. 2002; Reagan, Meister et al. 2002; Myers, Oyama et al. 2009). Based upon the results of retrospective (Myers, Laudner et al. 2006; Ruotolo, Price et al. 2006; Dines, Frank et al. 2009) and prospective (Shanley, Rauh et al. 2011; Wilk, Macrina et al. 2011) studies, investigators have determined that dominant side-to-side losses in TARC, on the IR side, have been associated with increased injury rates among baseball players. These findings suggest that preservation of TARC is critical to
Several authors have documented humeral ROM in baseball players (Borsa, Dover et al. 2006; Myers, Laudner et al. 2006; Laudner, Sipes et al. 2008; Myers, Oyama et al. 2009; Shanley, Rauh et al. 2011; Wilk, Macrina et al. 2011). Clinical measures of TARC (ER + IR) are most often reported with the subject lying supine and the shoulder abducted to 90 degrees within the coronal plane. This method has demonstrated acceptable intrarater reliability [Intraclass correlation coefficients (ICCs)\((2,1)\) = 0.95-0.98], (Laudner, Sipes et al. 2008) interrater reliability (ICCs\((2,k)\) = 0.95-0.99), (Shanley, Rauh et al. 2011), and intersession reliability (ICCs\((2,k)\) = 0.93-0.97) (Myers, Oyama et al. 2009)].

Specific measurement techniques of passive shoulder ROM have varied among investigators, making it difficult for direct comparisons across studies. Particularly, HA measures have been reported using both side-lying (Tyler TF, Roy T et al. 1999; Tyler, Nicholas et al. 2000; Myers, Laudner et al. 2006) and supine methods (Laudner, Stanek et al. 2006; Myers, Oyama et al. 2007; Shanley, Rauh et al. 2011). However, more recent investigation into these techniques has shown that the supine method shows higher reliability and lower standard error measures (SEMs) when compared to the side-lying technique (Laudner, Stanek et al. 2006; Myers, Oyama et al. 2007). Laudner et al. (2006) reported high intratester (ICC\((2,1)\) = 0.93, SEM = 1.64) and intertester (ICC\((2,k)\) = 0.91, SEM = 1.74) reliability using the supine scapular stabilization technique when compared to the sidelying method. Later research has supported these comparisons reporting higher intrasession (ICC\((2,1)\) = 0.91 vs 0.83),
intersession (ICC(2,k) = 0.75 vs 0.42), and intertester (ICC(2,k) = 0.94 vs 0.69) reliability for the supine technique in relation to the sidelying method (respectively) (Myers, Oyama et al. 2007). These results imply that supine measures are likely more accurate and indicative of true HA measures.

2.3. Humeral Morphology

Humeral antetorsion is a natural physical maturation process thought to occur at the proximal humeral physis. There is evidence to support this theory as 80% of overall humeral growth has been shown to occur at the proximal physis (Pritchett 1991). Baseball athletes consistently exhibit decreased humeral torsion on the throwing shoulder when compared to the non-throwing shoulder, a clinical finding termed humeral retrotorsion (Crockett, Gross et al. 2002; Osbahr, Cannon et al. 2002; Reagan, Meister et al. 2002; Yamamoto, Itoi et al. 2006). These differences among overhead athletes are not surprising as it is estimated that 90% of the proximal physeal development occurs after the age of 11 (Pritchett 1991). These differences are likely the result of the large torsion moments that are placed on the shoulder during the act of throwing (Sabick, Kim et al. 2005; Myers, Oyama et al. 2009), which can reach up to 90 N*m (Fleisig, Andrews et al. 1995). Many authors assert that humeral retrotorsion is primarily responsible for the observed side-to-side shift in TARC towards ER in overhead athletes (Reagan, Meister et al. 2002; Wilk, Meister et al. 2002; Myers, Oyama et al. 2009). However, much of the previous research has failed to account for the influence of humeral torsion, thereby, confounding the results of these studies.
While consensus is lacking, there continues to be compelling evidence to suggest that humeral retrotorsion significantly influences shoulder ROM.

The objective measurement of humeral torsion has been analyzed using a range of various imaging modalities that include computed tomography (CT) (Crockett, Gross et al. 2002), radiographs (Osbahr, Cannon et al. 2002; Reagan, Meister et al. 2002), and ultrasonography (Ito, Eto et al. 1995; Whiteley, Ginn et al. 2006; Yamamoto, Itoi et al. 2006; Myers, Oyama et al. 2009). Due to the associated cost and ionizing radiation exposure of radiographs and CT, ultrasonography appears to be gaining favor among clinicians and researchers.

Using the ultrasound imaging assessment procedures first introduced by Ito et al. (1995), humeral torsion is estimated with the subject lying supine and the upper extremity positioned in 90 degrees of shoulder abduction and elbow flexion (Ito, Eto et al. 1995). The ultrasound system is then used to align the greater and lesser tuberosities within the coronal plane, with the resulting forearm angle representing the epicondylar axis at the elbow, and the corresponding degree of humeral torsion. Previous, studies using the this method have established high intersession and inter-rater reliability with ICCs$_{(2,k)}$ ranging from 0.96-0.98, and an average SEM of 2.3° (Myers, Oyama et al. 2009). It is significant to note that these values were reflective of separate healthy college-aged student sample and not the overhead athletes that participated in the study. Nonetheless, studies examining active baseball players have also reported excellent inter-rater reliability (ICCs$_{(2,k)} \geq 0.94$) when using this method (Whiteley, Ginn et al. 2006).
2.4. Glenohumeral Joint Translation

The extreme physiologic demand of overhead throwing requires an exceptional balance between mobility and stability at the glenohumeral joint. Many authors agree that there is a minimal amount of capsular laxity is necessary to generate the forces required during throwing (Burkhart SS, Morgan CD et al. 2003; Borsa, Laudner et al. 2008). Some believe that baseball athletes demonstrate altered arthrokinematics and passive humeral ROM due to reactive scarring and ligamentous contracture of the posterior/inferior capsule as a result of repetitive tissue stress (Burkhart SS, Morgan CD et al. 2003). These adaptations are hypothesized to reduce posterior/inferior joint laxity, however empirical evidence is lacking to substantiate these claims among overhead athletes. The clinical assessment of capsular mobility at the glenohumeral joint is often determined by using a method of passive accessory joint glides to estimate the magnitude of humeral translation (Maitland 1980; Hawkins, Schutte et al. 1996).

To quantify joint translation investigators have used an array of objective techniques. These techniques include manual joint translation under stress radiography (Hawkins, Schutte et al. 1996; Ellenbecker, Mattalino et al. 2000), electromagnetic kinematic tracking (Tibone, Lee et al. 2002; Sethi, Tibone et al. 2004); mechanical loading of stress arthrometers and telos force applicators (Borsa, Sauers et al. 2001; Sauers, Borsa et al. 2001; Borsa, Laudner et al. 2008). While mechanical applicators are helpful in providing standard loads at the shoulder, these techniques do not correspond to clinical applications.
Additionally, the inability for subjects to relax in the apparatus may explain the wide range of A/P translations observed in these studies (Borsa, Sauers et al. 2001; Sauers, Borsa et al. 2001). Conversely, studies using manual techniques have been unsuccessful in standardizing joint load making comparisons difficult across trials and between subjects. These limitations may have hindered the investigators ability to detect translational differences between dominant and non-dominant shoulders.

Similarly, excessive anterior joint capsule laxity has also been proposed as a mechanism for altered joint mechanics among throwing athletes. However, despite widespread belief that these athletes acquire hyperlaxity of the anterior glenohumeral joint (Burkhart SS, Morgan CD et al. 2003), empirical data are often inconsistent between studies (Ellenbecker, Mattalino et al. 2000; Borsa PA, Scibek J et al. 2004; Sethi, Tibone et al. 2004; Laudner, Meister et al. 2012). Of the two available studies supporting this hypothesis, several limitations exist (Sethi, Tibone et al. 2004; Laudner, Meister et al. 2012). These studies failed to account for players with posterior shoulder tightness, side-to-side comparisons and measurement of total A/P translation. Based on these limitations, further research is warranted to account for differences in capsular mobility, should they exist.

2.5. Posterior Rotator Cuff Stiffness

Investigators have cited posterior rotator cuff muscle stiffness as a potential mechanism of PST that may lead to the development of shoulder injury
(Myers, Laudner et al. 2006; Oyama, Myers et al. 2010). These hypotheses are significantly limited due to lack of objective data to support these claims. A driving factor behind these limitations is the difficulty to differentiate in-vivo muscle stiffness from capsular restrictions. To our knowledge no attempts have been made to directly quantify in-vivo tissue stiffness of the posterior rotator cuff in throwing athletes.

Recently, investigators used ultrasound imaging to document changes in cross-sectional-area (CSA) of the infraspinatus muscle before and after an eccentric loading activity (Oyama, Myers et al. 2010). An isokinetic dynamometer was used to fatigue healthy volunteers for a dosage of 9 sets of 25 repetitions to simulate the mechanical stresses placed on the shoulder during pitching. Humeral ROM and muscle CSA were assessed immediately pre/post, and at a 24 hr follow-up.

The authors reported significant increases in infraspinatus CSA during the immediate follow-up and at 24 hours post-activity. Additionally, the results show that humeral IR and HA remained unchanged at the immediate post-test, but were significantly decreased at the 24-hour follow-up. The authors suggest that the changes in infraspinatus CSA and ROM may mimic the mechanisms of muscular stiffness in individuals with PST. However, these associations may be unrelated, as the immediate increase in CSA did not correspond to immediate decreases in humeral ROM. The authors continue to speculate that perhaps it is more likely that increased cellular permeability and presence of inflammatory markers were responsible (not CSA) for the ROM differences exhibited 24hrs
post-test. These results demonstrate the current lack of understanding regarding the characteristics posterior rotator cuff and its’ influence on glenohumeral ROM. To our knowledge no other studies have been able to effectively quantify these mechanisms and their relationship to humeral ROM.

2.6. Effects of Conservative Interventions on Posterior Shoulder Tightness

Due to the association of PST and injury, previous studies have investigated the effectiveness of therapeutic techniques in restoring humeral ROM. A sample of 54 healthy college-aged individuals was recruited with those exhibiting at least a 10-degree side-to-side loss in IR participating a four-week regimen of “sleeper” (n = 15) or “cross-body” (n = 15) stretches (McClure, Balaicuis et al. 2007). The two intervention groups were instructed to perform the stretches on the limited side only, once daily for 5 repetitions, holding each for 30 seconds. These effects were compared to the control group (n = 24) that did not display side-to-side differences, and who were instructed not to stretch for the duration of the study. The analysis showed that individuals performing cross-body stretches displayed a statistical increase in IR (+6°) when compared to the control group. This suggests that in a healthy, non-athletic population cross-body stretches are more effective in restoring IR loss than sleeper stretches. However, this study has several limitations. The small sample size reduces the statistical power and prohibits the investigators ability to detect other ROM differences that may have existed. The authors stipulate that those in the sleeper-stretching group trended towards significance differences in IR, citing the limited sample
size as rationale for this lack in statistical differences. Additionally, HA was not included as an independent variable, thus constraining the investigator’s ability to identify individuals with PST. Finally, the author’s excluded both athletes and those with shoulder pain who also displayed PST.

Other investigators have examined the effects of stretching among baseball athletes. A comparison of side-to-side differences was observed following the acute performance of the “Fauls Modified Passive Shoulder Stretching Routine” in 30 asymptomatic baseball players (Sauers, August et al. 2007). This routine consists of 12 sidelying and supine shoulder activities, which were performed only on the athlete’s throwing arm for 5 repetitions of 3-7 seconds. The authors found statistical increases in dominant-sided glenohumeral ER (+5°), IR (+6°), and HA (+2°) with no changes on the non-dominant side, suggesting that the Faul’s stretching method is acutely effective at gaining ER, IR and HA in healthy baseball players. However, the inclusion of athletes without PST and large number of activities performed by each subject limit the definitive conclusions that can be drawn from this study.

More specific comparisons were later performed examining the acute effects of “sleeper stretches” on TARC and humeral adduction in college baseball players (n = 33) (Laudner, Sipes et al. 2008). These comparisons were made to a control group of healthy, active college students (n = 33) that did not participate in the stretching intervention, or have recent participation in overhead sports (within 5 years). The intervention was applied by the principle investigator for dosage of 30-seconds for 3 repetitions on the dominant throwing shoulder. The
results showed significant temporal increases in IR (+3°) and HA (+2°) only among the baseball athletes that underwent treatment, indicating that the acute manual application of sleeper stretches is effective in decreasing GIRD and PST among baseball players. Within the discussion, the authors did acknowledge some of the inherent limitations of this study. First, the investigators emphasize the insufficient sample size used in this study based on a priori power analysis that required a group of 58 athletes to reach a power of 0.80, which may have limited their ability to detect additional differences in ROM. Additionally, the authors contend that the passive therapist-applied stretches may be more effective than conventional self-stretching, emphasizing the importance of maintaining proper stretching technique.

Limitations not reported by the investigators consist of the inclusion of athletes that did not necessarily display PST, or current symptoms, which inhibits the generalizibility of the results to pathologic populations. In addition, while reaching statistical significance, these differences do not fall outside the margin of minimal detectable change (MDC) for PST reported by previous investigators (Kolber and Hanney 2010). Furthermore, little is known regarding the minimal clinical important differences (MCIDs) in ROM that are necessary to impact clinical outcomes in patients with shoulder injury making the generalizations difficult to injured athletes.

Recently, researchers reported on a 6-week regimen of “sleeper stretching” for treating overhead athletes (volleyball, tennis, squash, water polo, and badminton) with PST (defined as ≥ 15° dominant-sided in deficit in IR).
(Maenhout, Van Eessel et al. 2012). The data show a significant improvement in side-to-side deficits for IR (14\(^\circ\)) and HA (11\(^\circ\)) when compared to healthy control who did not perform stretching or have PST \((P < .05)\). This study did not consider TARC or the influence of HT. Therefore, while the treatment group did gain IR, it is unknown how much HT may have influenced this change. Not accounting for TARC inhibits the ability to understand the responsibility that bony morphology may have had on these IR deficits. Despite these limitations this study shows that “sleeper stretching” is an effective treatment interventions for restoring IR deficits in athletes with PST.

PST has been proposed as a mechanism of overuse in individuals with internal impingement, and has served as a treatment focus for resolving symptoms in patients with shoulder pain. A cohort design was used to investigate the effects of physical therapy on resolving ROM deficits and pain in 22 recreational athletes (age 41 ±13 years) with symptomatic internal impingement (Tyler, Nicholas et al. 2009). Comparisons were made between patients who reported complete resolution of symptoms to those who continued to complain of residual pain following a variable course of physical therapy (7 ±2 weeks). Interventions included posterior glenohumeral joint glides, active-assisted cross-body adduction, sleeper stretch, ER, scapular strengthening exercises and a home exercise program. Physical therapy was performed for 3 times per week until complete resolution of symptoms was achieved, or patient progress had plateaued. The results of this study show that patients with internal impingement had baseline side-to-side deficits in IR (35\(^\circ\)), ER (23\(^\circ\)), and sidelying HA (35\(^\circ\)).
Following the physical therapy intervention there was a statistically significant improvement in IR, ER loss, and HA among the entire sample, with a significant gain in HA among patients with complete symptom relief when compared to those with residual pain (35° vs 18°; P < 0.05). Based on these results the authors suggest that resolution of symptoms was related to correction of HA deficit, and not IR loss. However, these conclusions may be skewed based upon the limitations of this study. The investigators failed to account for potential confounding variables in their analysis, which include humeral torsion, sport of participation, duration of symptoms, and age. Furthermore, the small sample size, between-subjects variability, and number of interventions utilized make the determination of relationships difficult. Based upon these limitations, additional study is required to understand the associations between the resolution of PST and shoulder pain.

To date, only one study has examined the effectiveness of manual therapy for resolving the deficits of PST (Manske, Meschke et al. 2010). Investigators measured changes in shoulder ROM (IR, ER, and HA) following a 4-week regimen of glenohumeral joint mobilizations plus crossbody stretching (n = 20) or cross-body shoulder stretching only (n =19) in baseball players. Each player within this study had at least a 10° IR deficit on the dominant side. Results following the 4-week intervention show no differences between groups receiving mobilizations plus stretching and stretching alone (P > .05). However, at 4-weeks post-intervention the mobilization group maintained the IR gains in shoulder ROM to a greater degree than did the stretch only group. It is important to
consider the possible influence of confounding factors, as activity exposure was not regulated within this posttreatment phase. Overall, the results indicate that glenohumeral joint mobilizations serve no acute benefits for reducing deficits in PST when compared to stretching alone. There may be a greater lasting treatment effect associated with mobilizations however further study is required to determine these relationships.

The previous studies share limitations that constrain the number of conclusions that can be determined from the results. Failing to account for the influence of humeral morphology serves as a major limitation, as it is unclear the magnitude to which HT influences the effectiveness of therapeutic interventions. Additionally, not all these studies included individuals with PST, potentially constraining the effect size of the selected interventions. Finally, these studies lack the ability to observe any intrinsic mechanical tissue changes that may have occurred during application of these interventions. This limitation prohibits the clinician’s ability to focus therapeutic interventions on the tissue(s) most responsible for causing PST.

2.7. Conclusion

In efforts to reduce the number of overuse arm injuries in baseball players a greater understanding behind the mechanisms that contribute to PST is warranted. Many investigators theorize that while some athletes with PST may exhibit primary alterations in bony architecture, many likely have capsuloligamentous or musculotendinous stiffness that are superimposed on
osseous adaptations. These tissue alterations are thought to contribute to the high prevalence of internal impingement (Tyler, Nicholas et al. 2000), type II superior labrum anterior to posterior (SLAP) lesions (Burkhart SS, Morgan CD et al. 2000) and ulnar collateral ligament injuries (Dines, Frank et al. 2009) reported among these athletes. Furthermore, based on a small number of studies, conservative therapeutic treatment of PST appears to be effective in reducing ROM deficits and resolving shoulder pain. However, the existing literature has been limited in its ability to discriminate between capsuloligamentous restraint, muscle tightness and osseous morphology in athletes with PST. These limitations currently prohibit the selection of focused treatment interventions and the identification of prognostic factors. Based on the current evidence, further research is required to determine the specific mechanisms that contribute to PST in overhand athletes.
2.8. References


3.1. SYNOPSIS

Clinical applications of rehabilitative ultrasound imaging (RUSI) are rapidly emerging as potential tools to assist rehabilitation specialists in the evaluation and treatment of musculoskeletal disorders. This commentary highlights the recent research findings and illustrates the potential clinical application of RUSI including emerging technologies and novel applications in the management of shoulder disorders.

**Key Words:** rehabilitation, rotator cuff, ultrasonography, ultrasound examination.
3.2. INTRODUCTION

Recent estimates suggest that over 20% of the population currently suffers from shoulder pain.\textsuperscript{80} Symptoms of debilitating pain, weakness, and loss of function make it necessary to explore the best means available to inform clinical practice. Traditionally, physical therapists have used a variety of physical examination and therapeutic techniques to identify, stage, and treat shoulder pain. The diagnostic yield of these procedures is variable and often limited as they rely heavily upon patient report and clinician judgment to determine the severity and source(s) of patient symptoms. Recently, ultrasound imaging (USI) has been embraced as a potential option to aid clinical decision-making thru the delivery of live imaging at a relatively affordable cost. As a result of the prevalence, complexity and subsequent burden of shoulder injuries, investigators are exploring the potential role of USI to augment the therapeutic management of these patients.

Clinical uses of USI fall into the two distinct categories of traditional ‘diagnostic’ ultrasound imaging, and ‘rehabilitative’ ultrasound imaging (RUSI).\textsuperscript{85} RUSI is defined as; “\textit{a procedure used by physical therapists to evaluate muscle and related soft tissue morphology and function during exercise and physical tasks…Additionally, RUSI is used in basic, applied, and clinical rehabilitative research to inform clinical practice.}”\textsuperscript{76} The Orthopedic Section of the American Physical Therapy Association has recently recognized a Special Interest Group on ‘Imaging’ with an initiative of “defining, communicating, and promoting the unique role of the physical therapist in imaging.”\textsuperscript{68} These initiatives have brought
with them efforts to standardize imaging procedures through the establishment of credentialing organizations for educating rehabilitation specialists in the use of RUSI.\textsuperscript{2}

Specifically at the shoulder, investigators have used RUSI to assess anatomical landmarks,\textsuperscript{70} analyze muscle morphology,\textsuperscript{61} classify tissue integrity,\textsuperscript{35} and measure vascular properties of the soft-tissues.\textsuperscript{65} Considering these applications, this modality could feasibly augment rehabilitation evaluation, patient prognosis, therapeutic interventions and clinical outcomes. However, prior to the large-scale implementation of RUSI, it is imperative to understand the practicality and clinometric properties of this modality.\textsuperscript{31} Therefore, the purpose of this commentary is to provide a general summary of quantitative musculoskeletal RUSI measures and propose clinical applications for the therapeutic management of shoulder pain. For specific discussions regarding technical descriptions of ultrasound imaging physics and capabilities the reader is encouraged to seek more thorough resources.\textsuperscript{85}

### 3.3. Skeletal Characteristics

The bony architecture of the glenohumeral joint allows for a wide range of motion to occur at the shoulder. This unique morphology and exceptional range of motion is thought to potentially be a predisposing factor for injury by placing excessive stress and demand on the supporting tissues. Recent literature has described the use of RUSI at the shoulder to identify anatomical variants of bony morphology\textsuperscript{14} and osseous landmarks\textsuperscript{18} that have been linked to symptoms. Of
these key measures: 1. the degree of humeral torsion and 2. the magnitude of
the acromio-humeral distance are among the most frequently reported.

*Humeral Torsion*

The basic factors that influence range of motion (ROM) at the
glenohumeral joint include bony morphology and musculotendious stiffness. The
challenge facing clinicians lies in determining the contributions of bony
morphology, as traditional goniometric measures do not discriminate between
bony architecture and soft-tissue restrictions. To help distinguish these
influencing factors RUSI has been employed to determine the contributions of
humeral torsion. Humeral torsion represents the relative osseous rotation from
proximal to distal articular surfaces, and is calculated with RUSI by aligning the
apices of the greater and lesser tuberosities and measuring the corresponding
forearm inclination angle (FIGURE 3.1 & 3.2). Rehabilitation specialists
investigating the measurement characteristics of this technique (TABLE 3.1)
report acceptable intra-rater and inter-rater reliability [intra-class correlation
coefficients (ICC$_{2,k}$) > 0.90] in asymptomatic subjects as well as overhead
athletes. Additionally, a recent study of asymptomatic baseball players
demonstrated the criterion validity of RUSI measures of humeral torsion to the
‘gold standard’ of computed tomography (CT), thus confirming ultrasound as a
viable option for quantifying humeral torsion.

When considering the bony anatomy, humeral torsion is a characteristic
shown to heavily influence shoulder ROM, particularly in the overhead
athlete where arm injuries are prevalent. Investigators estimate that most
non-throwing individuals commonly have a small degree of antetorsion of the humerus (approximately 20-30°). However, in overhead athletes, the stresses placed on the humerus during throwing are thought to retard the natural development of humeral antetorsion that occurs during skeletal maturation, a clinical finding known as ‘humeral retrotorsion’. This contention has been supported by recent studies in which throwing athletes have consistently displayed humeral retrotorsion of the dominant arm when compared to their non-dominant side and the arms of other non-throwing athletes.

The clinical impact of this anatomical variation is illustrated when considering the injury prevalence of overhand athletes, which is markedly higher for individuals displaying increased dominant humeral retrotorsion (mean difference = 7.2°, P = 0.027), glenohumeral internal rotation deficit (GIRD) and posterior shoulder tightness (horizontal adduction). Particularly, a side-to-side decrease in internal rotation of the dominant arm (≥ 20°) has consistently shown to increase the incidence of injury in high school and professional baseball pitchers. Clinically, RUSI of humeral torsion may be useful in identifying those patients presenting with GIRD as a result of bony morphology and not soft-tissue restrictions. Additionally, this technique may also allow for treatment and ROM goals to be tailored based on the contribution of bony morphology.

*Acromiohumeral Distance*

While the etiology, pain generating factors, and level of tissue involvement of subacromial impingement is debatable, there is an abundance of literature...
to suggest that changes to the subacromial space are associated with patient function and clinical outcome.\textsuperscript{20, 55, 69, 71} To quantify the dimension of the subacromial space investigators have utilized radiographs,\textsuperscript{62, 66} magnetic resonance imaging\textsuperscript{28, 29} and RUSI\textsuperscript{4, 18} to determine acromiohumeral distance (AHD). AHD is operationally defined as the shortest linear distance between the most inferior aspect of the acromion and the adjacent humeral head.\textsuperscript{18} RUSI measurement of AHD is obtained with the transducer oriented in the scapular plane and placed on the lateral aspect of the acromion to capture the lateral edge of the acromion and superior portion of the humeral head (FIGURE 3.3 and 3.4).

In asymptomatic individuals the average resting AHD values are typically greater than 7mm.\textsuperscript{71} Decreased AHD values are associated with the presence of a large rotator cuff tear, superior migration of the humeral head, and poor surgical outcomes.\textsuperscript{20, 55, 69, 71} These decreased findings are often a result of the prolonged loss of muscle force coupling representative of end-stage rotator cuff disease. Recent literature also suggests that this measure may also be useful in individuals with less severe rotator cuff disorders and shoulder ROM impairments.\textsuperscript{49} In the absence of a chronic full-thickness rotator cuff tear, the dynamic assessment of AHD during active elevation can provide insight into impairments associated with movement abnormalities influencing the subacromial space during humeral elevation, such as subacromial impingement syndrome. More recent studies have shown that the range of motion where the rotator cuff tendons are most vulnerable to extrinsic impingement from the acromion, glenohumeral elevation up to 60°, is much lower than previously
believed.\textsuperscript{6, 8, 45} Therefore, in the classic “painful arc” of motion from 80° to 120° of elevation the rotator cuff tendons have moved medially beyond the anterior-inferior aspect of the acromion and are no longer susceptible to extrinsic impingement. Therefore, we recommend that USI for AHD measurement in this patient population be performed during active contraction and up to 60° of glenohumeral elevation to provide meaningful information related to subacromial impingement.

RUSI has been used by physical therapists to examine the influence of modifiable factors on AHD. These factors include standing posture,\textsuperscript{34} scapular position,\textsuperscript{70} posterior shoulder tightness and GIRD\textsuperscript{49}. Patients diagnosed with shoulder impingement who assumed an upright posture displayed means increases in AHD of 1.2mm (>10%) measured by RUSI, suggesting that postural re-education may be an effective treatment to increase the distance of this anatomic region.\textsuperscript{34} Similarly, positioning the scapula in a more externally rotated and posterior titled position (Scapular Assistance Test) increased AHD in individuals diagnosed with shoulder impingement,\textsuperscript{70} inferring that rehabilitation strategies aimed at improving scapular position and neuromuscular control may influence subacromial space. Furthermore, exercises aimed at improving posterior shoulder flexibility may be clinically valuable as this treatment approach was shown to increase AHD in overhead athletes displaying GIRD and posterior shoulder tightness.\textsuperscript{49}

This measure has proven to be reliable and valid (TABLE 3.1), however, it is important to note that more information is needed to determine minimal
clinically important differences (MCID) of this measure, as this represents a key gap in the knowledge base. However, this evidence does provide insight into the negative relationship of RUSI generated AHD measures and modifiable impairments associated with shoulder disorders that include, poor posture, forward scapular position, posterior shoulder tightness, and GIRD.

3.4. Musculotendinous Characteristics

Several techniques have been reported to quantify anthropometric and intrinsic musculotendinous factors known to influence muscle function including muscle thickness, muscle volume, cross-sectional area (CSA), fiber bundle length, pennation angle, and contractile density.\(^{27,41}\) Additionally, the emergence of novel technologies including Power Doppler and ultrasound-elastography offer promising ways of quantifying iv-vivo tissue characteristics.\(^{1,17,21,39,65}\) These techniques are promising to impact clinical practice by assessing changes in musculotendinous characteristics,\(^{15,16}\) muscle atrophy\(^{89,90}\) and evaluating treatment effects.\(^{22,33,38}\) The following summarizes RUSI measurement properties that have been used for quantifying musculotendinous characteristics of the shoulder girdle and proposes potential clinical applications of these techniques.

**Muscle Thickness**

Altered scapular movement patterns have been identified in individuals with shoulder dysfunction.\(^{46-48,50}\) The trapezius muscle is a primary scapula stabilizer and RUSI has been shown to quantify muscle thickness and
differences among contractile states in individuals with and without shoulder pain.\textsuperscript{57, 58} Resting lower trapezius muscle thickness averages were approximately 3.1mm (±0.8mm) in healthy individuals with acceptable measurement reliability and validity (TABLE 3.1) when measured 3cm lateral to the edge of the spinous processes of T7-8 (FIGURE 3.5).\textsuperscript{56-58} This measure may provide clinicians with information regarding lower trapezius muscle performance for patients with shoulder pain related to alterations in scapular function. In addition, RUSI assessment of scapulothoracic muscle thickness may be useful as a biofeedback tool for neuromuscular re-education. While, RUSI biofeedback measures of muscle thickness have proven an effective treatment strategy in those with low back pain,\textsuperscript{30, 63, 77, 85} there are no existing data to support this application at the shoulder. Future studies are needed to determine the relationships of scapulothoracic muscle thickness with the clinical examination findings and patient reported outcomes as well as the efficacy and effectiveness of biofeedback applications for the treatment shoulder pain.

\textit{Rotator Cuff Tendon Thickness}

Rotator cuff disease is the most prevalent shoulder disorder presenting for non-operative and post-operative rehabilitation. RUSI has been used by physiotherapists to reliably obtain measures of tendon characteristics (thickness) in a group of healthy college students and laborers (Table 3.1).\textsuperscript{7} These investigators reported an average supraspinatus tendon thickness of 6.6 mm with acceptable mean-differences of test-retest reliability (0.24 mm ± 0.37 mm) when measured at the base of the “tuberculum majus plateau” (greater tuberosity) of
the humeral head (FIGURE 3.6). In healthy individuals, side-to-side comparisons of supraspinatus tendon thickness showed negligible differences (mean differences = 0.1 mm), suggesting that thickness asymmetry is an abnormal finding when using RUSI. This contention was supported by Joensen et al\textsuperscript{32} who discovered that patients with unilateral shoulder tendinopathy display greater measures of supraspinatus tendon thickness on their symptomatic side. Criterion-referenced validity of this measure was supported with concurrent clinical findings of decreased muscle strength and increased palpable tenderness predicting supraspinatus tendon thickness measures that exceeded 15\% of their asymptomatic side (positive predictive value of 0.94). Considering these results, tendon thickness measured by RUSI may be a useful clinical indicator of tendon integrity and/or staging of pathology. The serial tracking of tendon thickness throughout the course of therapeutic care may also provide additional insight into patient progress and prognosis, however more studies are required to determine the utility of this measure.

Muscle Volume and Atrophy

Deltoid muscle function is a key impairment that is almost always addressed during the rehabilitation of patients with shoulder pain. For example, as many as 42\% in individuals with episodes of anterior shoulder instability present with axillary nerve injury which often results in deltoid muscle atrophy and decreased shoulder function.\textsuperscript{81} Deltoid muscle performance is also critical in the non-operative management of full thickness rotator cuff tears and post-operative outcome for candidates of reverse total shoulder arthroplasty.\textsuperscript{67} With
these considerations, measures of deltoid morphology may be valuable to
determine patient functional capacity. Audenaert et al.,\textsuperscript{3} recently demonstrated
the validity of RUSI in assessing deltoid muscle volume by comparing ultrasound
derived measures described below with the corresponding amount of fluid
displacement methods ex-vivo (TABLE 3.1). By using the following formula,
ultrasonography demonstrated high criterion validity ($r = 0.98$) with the water
displacement method.

$$\text{VOLUME} = \left( \frac{\text{LENGTH} \times \text{HEIGHT}}{2} \right) \times \text{THICKNESS}$$

Additionally, RUSI estimates of deltoid volume were strongly associated ($r = 0.89$, $p < 0.001$) with isokinetic peak torque for shoulder abduction,
demonstrating the relevance of ultrasound-derived deltoid muscle volume to
functional performance.$^3$ Based on these reports, RUSI measurement of deltoid
volume may be useful as a prognostic indicator for non-operative and operative
management of shoulder pain. Sequential tracking of deltoid hypertrophy/atrophy
in individuals with shoulder dysfunction may lend insight into mechanisms
underlying recovery of shoulder function and effectiveness of interventions.
However, more research is needed to elucidate viable applications and full
clinical relevance of this measure.

\textit{Rotator Cuff Cross-Sectional Area \& Muscle Atrophy}

Similar to deltoid atrophy, rotator cuff muscle atrophy is associated with
chronic rotator disease.$^{24, 25, 78}$ Due to this relationship, rehabilitation programs
are often tailored to improve rotator cuff muscle strength and endurance. To
assess the integrity of the rotator cuff and presence muscle atrophy, RUSI
measures are captured similar to the scapular “Y” method of computed
tomography (CT) and MRI (FIGURE 3.7). With the suprascapular notch
serving as a standardized landmark, images are obtained within the short axis of
the supraspinatus muscle to visualize the contents of the suprascapular fossa.
Occupation ratios are calculated using two ellipses; one to quantify all contents
within the suprascapular fossa, and a second surrounding the hyperechoic
supraspinatus muscle (FIGURE 3.8) to estimate the degree of muscle atrophy
(FIGURE 3.9). High correlations of these measures have been reported
between RUSI and MRI (TABLE 1), providing evidence of criterion-referenced
validity.  

Similar imaging methods have been used to calculate CSA of the
supraspinatus and infraspinatus muscles in healthy individuals. Supraspinatus CSA was estimated with the probe oriented perpendicular to the
muscle’s line of action (short-axis) at a standardized location mid-way between
the medial border of the scapular spine and the lateral acromion. An ellipse is
then drawn around the hyoechoic supraspinatus muscle. Physical therapists
investigating these techniques reported acceptable reproducibility and validity of
this characteristic using RUSI (TABLE 3.1). Atrophy via this view using
CT/MRI is an important prognostic factor for patients with rotator cuff disease.
Therefore, RUSI application is likely to provide meaningful tissue level
information aiding in improving outcomes for non-operative and post-operative
rehabilitation strategies for rotator cuff disease.

Oyama et al (2011) used RUSI to observe acute increases in infraspinatus
CSA following eccentric rotator cuff training within a group of collegiate baseball players. Changes in infraspinatus CSA were obtained along 3 points a standardized template within the short axis of the muscle (FIGURE 3.10 & 3.11) and demonstrated acceptable measurement reliability (TABLE 3.1). CSA changes are thought to be a product of vasodialation, increased cellular permeability and inflammatory markers and were concurrent with losses in glenohumeral range of motion. These adaptive CSA changes along with acute losses of passive motion suggest that flexibility activities may help avoid range of motion deficits following these types of activities and mechanical stresses. Assessment of CSA may be an important prognostic factor for treatment and may provide evidence for effective rotator cuff strengthening interventions.

Fatty Infiltration

Associated with rotator cuff atrophy (measured by CSA and volume), fatty infiltration is considered to be an irreversible sequelae to severe rotator cuff disease. This finding has shown to have a detrimental relationship to glenohumeral force coupling, anatomic surgical repair and functional outcome. Measurement of fatty infiltration is determined by estimating the density of hyperechoic fibroadipose bundles that are invested between the perimysium of the muscle. By adapting Goutallier’s (1994) original 4-part classification system of fatty infiltration of the rotator cuff, researchers have applied RUSI to a 3-part classification system based on tissue appearance. Fatty infiltration is categorized using RUSI by echogenicity and structural organization as compared to the superficial deltoid and trapezius muscles. These muscles provide a gradient
standard from which to base the assessment of fatty infiltration for the rotator cuff. Qualitative comparisons of echogenicity are classified as grade “0” - isoechoic (normal); “1” - mildly hyperechoic (mild infiltrate); or “2” - markedly hyperechoic (marked infiltrate) when compared to the superficial deltoid or trapezius muscles.

RUSI fatty-infiltration classification has been documented in the assessment of muscle integrity for the supraspinatus and infraspinatus muscles (TABLE 3.1). Investigators using this technique reported high sensitivity of RUSI in accurately identifying marked infiltrate of the infraspinatus (13 out of 15) when compared to MRI findings. Rater agreement has shown to be increased (Kappa ≥ 0.83) when these classifications are dichotomized.

These studies suggest early detection of this disease process may allow for rehabilitative interventions to be administered in the prevention of further degradation and/or provide prognostic indicators for clinical decision-making in patients with full-thickness rotator cuff tears. While more research is required to refine these techniques, they may serve to enhance the physical examination and prognosticate the functional capacity of individuals with rotator cuff disease.

**Fiber Bundle Length & Pennation Angle**

In addition to quantifying gross muscle morphology, the assessment of intrinsic contractile tissue characteristics may provide valuable information as these properties have shown to directly influence muscle performance. The supraspinatus muscle serves as a unique example when considering intrinsic contractile properties as it is thought to be responsible for withstanding multi-
directional load demands. Researchers using conventional USI to perform morphologic study of the supraspinatus found that the muscle is not uniformly continuous and consisted of anterior and posterior regions, which are then further subdivided into superficial, middle, and deep portions. These investigations included descriptions of fiber bundle length and pennation angles, which may also be useful in providing insight into the initiation and propagation of tears.

More recently, preliminary studies using RUSI have shown that individuals with supraspinatus tears have fiber bundle length and pennation angles that are significantly decreased in comparison to healthy individuals. Therefore, it may be reasonable to assume that measurable gains in fiber bundle length and increases in pennation angle may be useful for reporting patient progress with conservative or post-surgical interventions. However, prior to implementation further research is required to determine these associations and to clarify these clinical applications.

Ultrasound-Elastography

Shoulder ROM is transient in most patients as evidenced by changes due to interventions, overhead activity, and resolution of shoulder pain in those subjects with a clinical diagnosis of internal impingement. The improvements in passive motion suggest that rotator cuff stiffness changes were likely responsible for the change in ROM but it is unclear which tissues change and what interventions are most appropriate. Understanding musculotendinous stiffness may allow for better treatment pathways and more effective therapeutic interventions.
Ultrasound elastography is a post-processing tool that was initially developed to improve the detection of malignant tumors\textsuperscript{13, 23, 85} by using the raw data gathered from the ultrasound-imaging unit to perform cross-correlational analyses for estimating tissue displacement and strain.\textsuperscript{85} There is limited empirical evidence to support the current musculoskeletal use of this method due to the hurdles of implementation which include difficulty accessing the raw electrical ultrasound data (not available on most conventional systems),\textsuperscript{85} variability among processing procedures,\textsuperscript{40} and the use of commercial hardware and software programs which still lack proper validation. Considering these limitations, a majority of the available clinical data has been performed in Achilles tendons, and suggests that severe tendinopathies are often more stiff than healthy tendons.\textsuperscript{15} Understanding tissue stiffness patterns holds great potential to document soft-tissue integrity and disease staging.

Of the available evidence at the shoulder, investigators have examined supraspinatus tendon strain patterns during isometric and isotonic contractions in patients with shoulder pain.\textsuperscript{39} These results indicate that the bursal side of the cuff experiences greater strain during isometric contractions compared to the articular side. Interestingly, this relationship was transposed during isotonic contractions suggesting that greater articular-sided strain with isotonic rotator cuff activity. It is important to note that validity concerns of this method have been raised\textsuperscript{74} as the normative deformation ranges exceeded previously documented failure strains. However, the emergence of these technologies offers exciting possibility into the in-vivo quantification of soft-tissue stiffness and may augment
rehabilitation evaluation and treatment\textsuperscript{40} of shoulder dysfunction.

\textit{Blood Flow}

As understanding the mechanical characteristics of musculotendinous tissue is important, secondary measures of muscle function such as blood flow have also been related to tissue integrity\textsuperscript{5, 21, 54, 65}. Power Doppler is an USI function commonly used to depict arterial and venous blood flow within tissues\textsuperscript{21} and may be clinically helpful to identify potential sources of vascular compromise and/or tissue healing status\textsuperscript{5, 21, 65}. This feature enhances the range of conventional color Doppler imaging by allowing examiners to quantitatively assess dynamic blood volume over time, which may be clinically relevant to inform physical therapists of the absence or presence of vascular properties\textsuperscript{54}. Importantly, an increase in vascular flow appears to be a normal physiologic response in the asymptomatic rotator cuff, as power Doppler has been used to demonstrate dynamic increases in vascular supply of the supraspinatus tendon immediately following shoulder-fatiguing exercises\textsuperscript{1}. These studies also indicate that asymptomatic individuals often exhibit decreased vascular supply throughout the rotator cuff tendon with increased age\textsuperscript{65}.

In overhead athletes, power Doppler was used to document the relative decrease in axillary artery blood supply that occurs in the dominant arm of those with shoulder instability\textsuperscript{5}. Compared to healthy athletes, those diagnosed with shoulder instability saw just 25\% of the mean increase in axillary artery blood flow immediately after throwing\textsuperscript{5}. This difference may provide rationale for the phenomenon known as “dead-arm” syndrome more commonly reported within
this population. Furthermore, reversal of this disparity may potentially provide an objective means of documenting patient progress during a course of therapeutic care, however further research is needed to verify this.

Power Doppler has been also been used to examine the post-operative vascular characteristics of the supraspinatus tendon 6 months after surgical repair. Among the six regions examined (peribursal, peritendinous, musculotendinous, intratendinous, pericortical, and suture anchor site) the peritendinous region displayed the most robust blood flow, while the lowest overall vascular activity remained at suture anchor site. Consistently however, the vascular signal progressively decreased throughout all six regions over time, suggesting that decreased vascular flow as a potential marker of rotator cuff healing. Therefore, serial tracking of vascular activity could potentially provide information into the rehabilitation phase progression of post-operative rotator cuff patients by providing quantitative estimates of healing status. Further research is warranted to confirm these uses.

3.5. FUTURE CONSIDERATIONS

The clinical and research application of RUSI holds the possibility to impact patient outcomes through assessing tissue level characteristics in the prevention and treatment of shoulder disorders. Assessments of tissue morphology and muscle biofeedback are of particular interest for rehabilitation professionals as these techniques could potentially provide information into the intrinsic morphological state and responses to therapeutic interventions. RUSI
may be useful in developing tailored interventions, however, the performance of high quality studies is required to fully develop and elucidate its’ capabilities. Additionally, standardization of methods and clinical techniques are required to create clear communication between research investigators, rehabilitation specialists and the rest of the medical community.

3.6. SUMMARY

The goal of this commentary is to provide the reader with an overview of the literature and to describe some potential clinical applications of RUSI for physical therapists. Based on the current body of literature, it is likely that RUSI’s impact for the management of shoulder dysfunction has yet to be realized. There is a growing body of evidence to support the current development of technologies and integration of RUSI into rehabilitation practice. However, more data is needed to determine the clinical scope and measurement limitations of RUSI for the shoulder.
3.7. REFERENCES


33. Juul-Kristensen B, Bojsen-Moller F, Holst E, Ekdahl C. Comparison of muscle sizes and moment arms of two rotator cuff muscles measured by


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### TABLE 3.1. RUSI Clinometric Properties

<table>
<thead>
<tr>
<th>RUSI Measure</th>
<th>Study Sample</th>
<th>Reliability</th>
<th>Validity (Criterion)</th>
<th>Error</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Humeral Torsion</strong></td>
<td>Overhead Throwing Athletes(^{54, 87})</td>
<td>Intra-Rater</td>
<td>(r = 0.80, R^2 = 0.64, P &lt; 0.01) (CT)</td>
<td>Std Error = 1(^{1})</td>
</tr>
<tr>
<td><strong>Acromiohumeral Distance</strong></td>
<td>Rotator Cuff Disease &amp; Glenohumeral Internal Rotation Deficit &amp; Posterior Shoulder Tightness(^{4, 19, 50, 72})</td>
<td>Intra-Rater Humeral Elevation @ 0(^{0}) ICC(<em>{2,1}) = 0.80-0.94 @ 45(^{0}) ICC(</em>{2,1}) = 0.91 @ 60(^{0}) ICC(_{2,1}) = 0.92</td>
<td>(r = 0.80-0.85) (Radiographs)</td>
<td>MDC(_{90}) @0(^{0}) = 0.60 mm @45(^{0}) = 0.90 mm @90(^{0}) = 0.90 mm</td>
</tr>
<tr>
<td><strong>Muscle cross-sectional area</strong></td>
<td>Rotator Cuff Disease &amp; Overhead Athletes(^{3, 34, 63})</td>
<td>Intra-Rater (\dagger ICC_{2,1} \geq 0.90)</td>
<td>(\Upsilon r = 0.98) (Volumetric Water Displacement)</td>
<td>(\Upsilon SEM = 0.25 mm) (\dagger SEM = 0.74 mm) (\dagger SEM = )</td>
</tr>
<tr>
<td><strong>Fatty Infiltration &amp; Atrophy</strong></td>
<td>Rotator Cuff Disease(^{35, 65})</td>
<td>Intra-Rater (\dagger K = 0.76; \dagger K = 0.67) Inter-Rater (\dagger K = 0.71; \dagger K = 0.68)</td>
<td>(\dagger K_{W} = 0.78) (MRI) (\dagger K_{W} \geq 0.71) (MRI) (\dagger Atrophy r = 0.90)</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>Tendon Thickness</strong></td>
<td>Rotator Cuff Disease and Asymptomatic(^{8, 33})</td>
<td>Intra-Rater Mean-differences = (\dagger 0.24 mm \pm 0.37 mm)</td>
<td>N / A</td>
<td>Std Error = 0.8 mm (95% CI = 0.57 mm)</td>
</tr>
<tr>
<td><strong>Muscle Thickness</strong></td>
<td>Symptomatic and Asymptomatic(^{34, 59, 60})</td>
<td>Inter-Rater (\ast ICC_{2x} = 0.88)</td>
<td>(\ast r = 0.77) (MRI)</td>
<td>SEM = 0.30 mm</td>
</tr>
</tbody>
</table>

\(\dagger\) Supraspinatus; \(\dagger\) Infraspinatus; \(\ast\) Lower Trapezius; \(\Upsilon\) Deltoid
<table>
<thead>
<tr>
<th>RUSI Measure</th>
<th>Target Demographic</th>
<th>Patient Positioning</th>
<th>Probe placement</th>
<th>Measurement Landmarks</th>
</tr>
</thead>
</table>
| **Humeral Torsion**              | - Overhead Throwing Athletes  
- Shoulder Arthroplasty                                                              | Supine on plinth with shoulder at 90° of abduction and 90° of elbow flexion         | Within short-axis of the long-head of biceps tendon at the proximal humerus     | Proximal: parallel orientation of greater and lesser tuberosities  
Distal: Proximal ulnar border                                                   |
| **Acromiohumeral Distance**      | Rotator Cuff Disease & Glenohumeral Internal Rotation Deficit & Posterior Shoulder Tightness | Seated with an upright posture and the arm in a dependent position at the side.     | Longitudinally within scapular plane just inferior to lateral edge of acromion   | Linear distance from lateral edge of the acromion and superior border of the humeral head |
| **Muscle Cross-Sectional Area**  | Rotator Cuff Disease & Overhead Athletes                                            | Supraspinatus: Seated with arm at the side  
Infraspinatus: Prone with shoulder at 90° abduction and neutral rotation          | Supraspinatus: Short-axis view at the suprascapular notch  
Infraspinatus: Obliquely from Superior medial border towards lateral border     | Supraspinatus: Ellipse around the hypoechoic (dark) muscle within the supraspinatus fossa  
Infraspinatus: Ellipse around the hypoechoic (dark) muscle within the infraspinatus fossa |
| **Fatty Infiltration & Atrophy** | Rotator Cuff Disease                                                              | Seated or prone                             | Supraspinatus: Short-axis view at the suprascapular notch  
Infraspinatus: Short-axis view at the spinoglenoid notch                          | Relative echogenicity (brightness) of rotator cuff to superficial deltoid or trapezius.  
1. Isoechoic (normal)  
2. Mildly Hypoechoic  
3. Markedly Hyperechoic                                                           |
| **Tendon Thickness**             | Rotator Cuff Disease                                                              | Seated with arm at the side or shoulder extended & externally rotated (Modified-Crass Position) | Longitudinally within scapular plane just inferior to lateral edge of acromion   | Linear distance from the base of the tuberculum majoris plateau and the bursal edge of the tendon |
| **Muscle Thickness**             | Symptomatic and asymptomatic patients                                            | Lower Trapezius: Prone with shoulder at 90° abduction and neutral rotation         | Short-axis: 3cm lateral to the spinous process at thoracic level of inferior scapular angle | Linear distance from costal border to superficial edge for both medial and lateral sites |

†Supraspinatus; ‡Infraspinatus; *Lower Trapezius; ¥Deltoid
3.9. FIGURES

FIGURE 3.1. Ultrasound Image of Humeral Torsion. The probe is positioned until the apices of the tuberosities are visualized. The humerus is then rotated to allow the tuberosities to be oriented in parallel.

FIGURE 3.2. Humeral Torsion Measurement. The ultrasound probe is oriented proximally to capture the apices of the tuberosities. Once the tuberosities are aligned in parallel (proximal line), the corresponding forearm angle is measured. This angle represents the osseous humeral rotation and position of the epicondylar axis (distal line).
FIGURE 3.3. Acromiohumeral Distance. The AHD is calculated by measuring the linear distance from the most lateral edge of the acromion to the most proximal portion of the humeral head (white line).

FIGURE 3.4. Measurement Of Acromiohumeral Distance. The patient assumes an upright posture with the arm in a dependent position. The probe is placed within the coronal plane along the most lateral aspect of the acromion to capture the image.
FIGURE 3.5. Lower Trapezius Muscle Thickness. The medial and lateral muscle thickness sites are identified by measuring the linear distance between superficial and deep facial borders (white lines).

FIGURE 3.6. Supraspinatus Tendon Thickness. The tendon thickness is measured from the base of the greater tuberosity to the most superior margin of the tendon (white line).
FIGURE 3.7. Scapular 'Y' View. This MRI image depicts the cross-section of the supraspinatus fossa (black circle). Note the dark supraspinatus muscle surrounded by lighter non-contractile tissue.

FIGURE 3.8. Cross-Sectional Area of Supraspinatus. This figure represents a healthy supraspinatus muscle. Note the light colored central tendon surrounded by the dark (hypoechoic) muscle, each contained within the supraspinatus fossa.
FIGURE 3.9. FATTY ATROPHY OF THE SUPRASPINATUS. The outer ellipse signifies the entire area supraspinatus fossa, while the inner ellipse represents only the dark contractile tissue of the supraspinatus muscle.

FIGURE 3.10. Template For Infraspinatus Cross-Sectional Area. Three landmarks are identified (X): 1. acromion, 2. inferior angle, and 3. superior-medial border. The midpoint distance between the acromion and inferior angle is identified and a perpendicular line is drawn towards the superior-medial border within the short-axis of the muscle. The probe is moved along the perpendicular line to capture CSA.
FIGURE 3.11. INFRASPINATUS CROSS-SECTIONAL AREA. The white line represents the facial boundary of the infraspinatus muscle in short-axis/cross-section.
CHAPTER 4: MANUAL THERAPY IMPROVES RANGE OF MOTION DEFICITS IN BASEBALL PLAYERS WITH POSTERIOR SHOULDER TIGHTNESS


4.1. ABSTRACT

Background: Baseball players displaying deficits (dominant versus non-dominant) in shoulder range of motion (ROM) are at increased risk of arm injury. Currently, there is a lack of consensus regarding the best available treatment options to restore shoulder ROM.

Hypothesis: The use of instrumented soft tissue mobilizations (ISTM) and stretching will result in greater ROM gains in baseball players displaying posterior shoulder tightness (PST) when compared to stretching alone.

Study Design: Randomized Controlled Trial, Level of evidence, 1.

Methods: Shoulder ROM and humeral torsion were assessed in 60 baseball players (age 19 ±2 years) with ‘total arc of motion (TARC) + internal rotation (IR)’, and ‘horizontal adduction (HA)’ deficits as qualifiers for PST (nondominant – dominant ≥15°). Participants were randomly assigned to receive “ISTM plus stretching” (n =30), or “stretching only” (n =30). Deficits in ROM were compared between groups before and after a single treatment session. Treatment
effectiveness was determined using mixed-model ANOVAs (group x time) and a number-needed-to-treat (NNT) analysis with 95% confidence intervals (CI) for injury risk.

**Results:** At pretest, players displayed significant ($P < .001$) dominant-sided deficits in IR (-26°), TARC (-18°), and HA (-17°). Following treatment, both groups showed improvements in ROM, however, players receiving “ISTM plus stretching” had additional gains of +5° of IR ($P = .010$), +6° of TARC ($P = .010$), and +7° of HA ($P = .004$). The injury risk for ‘TARC + IR’ deficits was not different between players receiving “ISTM plus stretching” and “stretching only” [failure rate: 36% versus 43%, respectively, $P = .187$; NNT of 14.3 (95% CI: 10.3, 17.1)]. For ‘HA’ injury risk deficits, treatment failure rates were decreased with ISTM [failure rate: 7% versus 33%, $P = .010$; NNT of 2.2 (95% CI: 2.1, 2.4)].

**Conclusion:** The added use of ISTM with stretching resulted in greater ROM gains and decreased injury risk in baseball players with PST.

**Clinical Relevance:** Players with PST may respond differently to treatment based on their ROM deficit(s). Clinicians should consider ISTM for reducing ROM deficits and injury risk in baseball players with PST.

**Key Terms:** posterior shoulder tightness (PST); glenohumeral internal rotation deficit (GIRD); instrumented soft-tissue mobilization (ISTM); baseball.

**What is known about the subjects:** Recent prospective data shows that baseball players with dominant-sided deficits in shoulder ROM are at an increased risk of injury.

**What this study adds to existing knowledge:** This is the first study to show that
instrumented manual therapy in conjunction with a shoulder stretching routine significantly reduces ROM deficits and injury risk in baseball players “at risk” of injury when compared to stretching alone.

4.2. INTRODUCTION

The dominant (throwing) shoulder in baseball players consistently displays alterations in glenohumeral range of motion (ROM). Over time, the forces endured by the throwing shoulder are thought to increase shoulder external rotation and subsequently decrease internal rotation through the adaptation of humeral torsion. However, baseball players who display deficits in shoulder ROM (dominant vs. non-dominant) in excess of the normal adaptations are at an increased risk of injury. Prospective studies have demonstrated that players with deficits as low as a 5° in total arc of motion, 20° in internal rotation and 15° in horizontal adduction are 2-9 times more likely to sustain an arm injury. Together this evidence suggests that deficits indicating posterior shoulder tightness are associated with increased injury risk and that clinical treatment should focus on resolving these ROM deficits.

Intervention strategies to resolve posterior shoulder tightness generally focus on altering the capsuloligamentous and muscular adaptations associated with the throwing shoulder. However, recent literature suggests that ROM measures should be interpreted in the context of the osseus adaptation given that humeral torsion (HT) is moderately related to the degree of shoulder internal
rotation\textsuperscript{23,26} and horizontal adduction, but less so with external rotation.\textsuperscript{19} This suggests that when interpreting clinical ROM measurements it may be useful to consider the influence of HT.\textsuperscript{5,19,36}

Manual therapy and stretching interventions show a promising therapeutic ability to resolve posterior shoulder tightness.\textsuperscript{7,13,16,18,32} However, there is little available evidence to guide clinicians in the selection of best available treatment option(s) for improving these impairments. Given the increased injury risk associated with ROM deficits reflecting posterior shoulder tightness, identifying the most effective means of resolving these deficits may provide the best opportunity to decrease injury risk. Therefore, the purpose of this study is to compare shoulder ROM deficits between baseball players receiving instrumented soft tissue mobilization (ISTM) and stretching to a group receiving stretching alone. We hypothesize that players receiving a combination of ISTM and stretching will display greater resolution of ROM deficits.

4.3. MATERIALS AND METHODS

Participants

Baseball players with dominant-sided deficits in shoulder ROM on their dominant throwing shoulder were recruited for this study (Table 4.1). The eligibility requirements for this study included male baseball players ages 15 years and up with current participation on an organized baseball team as a pitcher or position player. Inclusion criteria included the presence of at least one ROM deficit; including a dominant-side deficit of 15° in total arc of motion (with at least 15°
loss in internal rotation) and/or HA. These criteria were selected as previous research indicates that a side-to-side difference of $20^\circ$ in ROM is associated with prospective injury.\textsuperscript{29,35} Therefore, as a conservative screening criteria, we selected a threshold of $\geq 15^\circ$ in an attempt to reduce injury risk factors, while remaining large enough to detect potential changes in ROM.

Participants were excluded if they; had a recent history of activity limiting shoulder pain (within 3-months), were not actively participating in all team activities, and/or had a previous surgical history on either shoulder. Two hundred and seventy-six players within the Greenville SC area were screened for eligibility, with 127 meeting requirements and 60 agreeing to participate and subsequently becoming enrolled within the study (FIGURE 4.1).

**Study Design & Procedures**

This single-blinded randomized controlled trial was approved by the Greenville Hospital System and University of South Carolina Institutional Review Board and conducted during the 2013 professional and scholastic season. Prior to participation each athlete/guardian completed informed consent form.

To determine eligibility for participation all players completed an initial assessment of shoulder range of motion and an activities questionnaire to screen for the presence of activity limiting arm pain. Qualifying players were then asked to complete a Pennsylvania Shoulder Scale (PSS) and Functional Arm Scale for Throwers (FAST) and return on a separate day for testing to ensure the presence of ROM deficits was consistent over at least a 24-hour period. The PSS is a 100-point scale that was used to determine the level of pain and disability for each
participant (lower score = more pain and disability). Players with > 30% disability on the PSS were not eligible for participation. The FAST is a 100-point functional scale developed for overhead throwers and was chosen to provide a sport specific assessment of function (higher scores = more sport-related pain and disability). Participants were then randomly allocated (by random drawing) into one of two intervention groups.

Those allocated to the ‘instrumented soft-tissue mobilizations (ISTM) and stretching’ group received 4-minutes of supervised posterior shoulder stretching followed by 4-minutes of supervised posterior shoulder stretching. Those in the ‘stretching only’ group received only 4-minutes of supervised posterior shoulder stretching.

**Shoulder Range of Motion Assessment**

*Internal and External Range of Motion.* A Baseline Digital Inclinometer (Fabrication Enterprises, Inc; White Plains, NY) was used for all ROM measures throughout the course of this study. For all shoulder ROM measures, the participants were positioned in supine on a plinth in 90° of shoulder abduction and elbow flexion. The same clinician provided stabilization and performed all ROM measures. Internal rotation (IR) was assessed using a towel-roll under the arm to maintain the position of the humerus, and the shoulder was passively rotated until the examiner felt movement at the corocoid process. A second investigator then aligned the digital inclinometer along the ulnar border and recorded the corresponding angle in degrees (FIGURE 4.3). External rotation was measured in a similar manner with the shoulder passively rotated to
the first resistance without overpressure (FIGURE 4.4). Two trials of each measure were taken and used for measurement reliability and statistical analysis. Measurement reliability for this study was acceptable for internal rotation: intraclass correlation coefficient \((ICC_{(2,1)} = 0.98\) [95% confidence interval \((95\% CI): .98, .99;\) standard error of measure \((SEM) = 1.3^\circ;\) minimal detectable change \((MDC_{95}) = 3.7^\circ\) and ER: \(ICC_{(2,1)} = .98\) (95% CI: .98, .99; SEM = 1.5^\circ; MDC_{95} = 4.0^\circ). Passive total arc of motion was calculated for each arm (total arc of motion = external rotation + internal rotation).

**Horizontal Adduction Range of Motion.** Horizontal adduction was collected using methods described by both Laudner et al\(^{12}\) and Myers et al,\(^{21}\) due to the previously established measurement reliability. For measurement the examiner stabilized the scapula in full retraction at the lateral scapular border and passively horizontally adducted the arm while maintaining neutral rotation and continued until resistance was felt. Once end range was reached, a second examiner measured the corresponding humeral horizontal adduction angle using the digital inclinometer at the humeral diaphysis relative the horizontal plane. Our test-retest reliability for horizontal adduction was \(ICC_{(2,1)} =0.99\) (95% CI: .99, .99; SEM = 1.3^\circ; MDC_{95} = 3.7^\circ). Reductions in ROM deficits were calculated for each variable ROM variable and used for statistical analysis (ROM deficit = mean nondominant value - mean dominant value).

**Humeral Torsion Assessment**

Humeral torsion was assessed using valid\(^{18}\) measures previously described by Whiteley et al\(^{34}\) and Yamamoto et al.\(^{38}\) A sonographer with 5 years
of experience in musculoskeletal ultrasonography performed all the imaging for this study. Participants were positioned supine on a plinth in 90° of shoulder abduction and elbow flexion. A SonoSite - Edge (SonoSite Inc. Bothell, WA, USA) ultrasound imaging unit with 4cm linear array transducer (6-15MHz) was used to collect all measures. The probe was placed on the participant’s shoulder at the level of the biceps groove and oriented perpendicular the plane of the plinth and verified with a bubble level. A second examiner then passively rotated the subject’s humerus until the apices of the greater and lesser tuberosities were oriented parallel to coronal plane (FIGURE 4.2). The second examiner then measured the corresponding angle using the digital inclinometer (Figure 4.3). For this sample the measurement reliability for humeral torsion was ICC(2,1) = .99 (95% CI: .98, .99; SEM = 1.3°; MDC95 = 3.5°).

**Interventions**

Measurements of ROM (external rotation, internal rotation, and horizontal adduction) were performed on both shoulders immediately before and after the treatment interventions, which were only administered to the dominant throwing shoulder. The primary investigator was blinded to group assignment and left the testing area for a standardized time of ten minutes while treatment was provided by one of two orthopedic physical therapists, each with over 15 years of clinical experience. Upon completion of the interventions the primary investigator returned for subsequent posttest measures. Specific group interventions were as follows:

*“Stretching Only” Group.* The ‘stretching only’ group (n =30) was given
standardized instruction and visual demonstration in the performance of sleeper and cross-body adduction stretching, which are common treatment interventions for ROM deficits that have been well documented in previous studies.\textsuperscript{11, 14, 16, 32}

The sleeper stretch was performed with the subject side-lying on a towel roll with the dominant throwing shoulder on the treatment table so that the scapula was retracted and stabilized. The humerus was positioned in 90° of shoulder elevation with the elbow flexed to 90° then rotated the shoulder internally using the opposite hand until a stretch was felt along the posterior aspect of the shoulder (FIGURE 4.4). The stretch was held for a duration of one minute as the treating therapist timed and assessed for appropriate stretching technique. Participants performed the stretch twice and were given 30 seconds of rest in between each repetition.

The cross-body adduction stretch was performed in the same starting position described above. Subjects were asked to grasp the dominant elbow with the opposite hand, pulling the arm across the front of the body until a stretch was felt in the posterior shoulder (FIGURE 4.5). The players were asked to perform 2 repetitions for one minute each, while an investigator timed the hold and evaluated the technique. Participants were again given 30 seconds of rest in between each repetition.

\textit{“ISTM plus Stretching” Group}. Immediately following the stretching described above, the “ISTM plus Stretching” group (n =30) also received instrumented soft-tissue mobilizations targeting the infraspinatus and teres minor muscles. Subjects were placed in prone for instrumented soft-tissue
mobilizations (SASTM; Carpal Therapy Inc, Indianapolis, IN) with the dominant arm positioned in neutral rotation at 90° of shoulder elevation and elbow flexion (FIGURE 4.6). A towel was placed under the participant’s shoulder to maintain alignment of the humerus within the scapular plane. Emollient was applied to the posterior axillary border to allow the tool to glide smoothly and all participants in this group were treated using the same instrument (SASTM # 4). Treatment strokes were administered at approximately a 45° angle to the skin’s surface for 2 minutes in both parallel and perpendicular directions to the fiber alignment of the infraspinatus and teres minor muscles (FIGURE 4.6 and FIGURE 4.7). To ensure standardized dosage between participants, a metronome was set at a rate of 45 hertz for each treatment to monitor frequency of strokes.

**Statistical Analysis**

A one-way analysis of variance (ANOVA) was performed to compare age, height, weight, subjective outcomes (PSS and FAST), and level of competition between the two treatment groups. Separate 3-way mixed-model ANOVAs (group x arm x time) were used to determine the underlying treatment effects for each dependent variable. Post-hoc planned comparisons were made between sides and groups for the dependent variables of interest (external rotation, internal rotation, total arc of motion, and horizontal adduction) using tests for simple main-effects differences. Pearson correlation coefficients ($r$) were calculated to assess the association between HT and the ROM deficits.

To further evaluate the clinical effectiveness of these interventions, a number-needed-to-treat (NNT) statistic with associated 95% confidence interval
(CI) was calculated for players after being dichotomized into successful/unsuccessful outcome. Players qualifying for injury risk (≥ 15° deficit for ‘total arc of motion plus internal rotation’, and/or ‘horizontal adduction’) at pretest who no longer qualified following treatment were considered to have a successful outcome. In contrast, players were considered to have an unsuccessful outcome if they did not fall below the injury risk threshold. We further examined injury risk qualification by comparing pretest and posttest percentage changes of success using a chi-square statistic, and these procedures were repeated for each category of injury risk qualification. G-Power software (version 3.1.6) was used to calculate required effect size and power based on a moderate a priori estimation of .30 and .80, respectively. PASW Statistic 18 software (SPSS Inc., Chicago, IL, USA) was used with statistical significance set a priori at α = .05.

4.4. RESULTS

Pre-Treatment Comparisons

There were no between group differences for age, height, weight, arm dominance, playing position, level of competition, or subjective outcomes scores (PSS and FAST) (TABLE 1). The total sample displayed significant dominant-sided deficits in internal rotation (26° ± 11°), total arc of motion (18° ± 11°), and horizontal adduction (17° ± 11°) at initial assessment (P < .001). There were no baseline differences between groups for ROM on either arm. However, the “stretching only” group did exhibit greater nondominant humeral torsion (6° ± 3°;
*P* = .038) when compared to the “ISTM plus stretching” group. When examining the relationship between humeral torsion difference and ROM at initial assessment, the data show that humeral torsion was related to internal rotation (*r* = .386, *P* < .001) and horizontal adduction (*r* = .287, *P* = .013) deficits for both groups.

**Post-Treatment Comparisons**

There was a significant interaction (group x side x time) for dominant internal rotation (*F*(1, 59) = 7.05, *P* = .010), total arc of motion (*F*(1, 59) = 7.10, *P* = .010), and horizontal adduction (*F*(1, 59) = 9.25, *P* = .004), indicating that those within the “ISTM plus stretching” group gained more ROM immediately following the intervention (TABLE 4.2). Specifically, the ‘ISTM and stretch’ group had deficit reductions of 12° (± 9°) for internal rotation, 14° (± 10°) for total arc of motion, and 14° (± 8°) for horizontal adduction (*P* < .001). Those within the “stretching only” group had deficit reductions of 7° (± 5°) for IR, 8° (± 6°) for total arc of motion, and 7° (± 8°) for horizontal adduction (*P* < .001). Our results also show that a majority of total arc of motion gained (+14°) on the dominant shoulder was predominantly in internal rotation (+12°) as indicated by an *R*² value of .71, suggesting that external rotation was not significantly influenced by these interventions. The effect sizes and power between groups were close to our conservative *a priori* estimation of .30 and .80; (internal rotation - effect size: .33; power: .75; total arc of motion - effect size: .33, power: .75; horizontal adduction - effect size: .37 with power: .85), indicating that the added benefits of ISTM exceeded the upper bound of measurement error for ROM change. While both
groups displayed ROM deficits reductions, the “ISTM plus stretching” group exhibited added ROM gains of 5° (± 2°) in internal rotation, 6° (± 2°) of total arc of motion, and 7° (± 2°) of horizontal adduction when compared to the “stretching only” group (FIGURE 4.8). No changes were observed in external rotation for either group with these interventions (P > .05). Following the interventions, humeral torsion was related only to the remaining horizontal deficits (r = .471, P = .009) in the “stretching only” group.

Of the 60 players participating in this study, 36 (60%) were successfully removed from ‘all categories’ of injury risk qualification (≥ 15° deficit for ‘total arc of motion plus internal rotation’, and/or ‘horizontal adduction’). When comparing between treatment groups, the percentage of success for ‘all categories’ of injury risk was not different for players receiving “ISTM plus stretching” and those receiving “stretching only” (70% vs. 50%, respectively, P = .187). Likewise, the percentage success rates of players for ‘total arc of motion plus internal rotation’ injury risk were not different between treatment groups (64% vs 57%, P = .521). For the injury risk qualification of horizontal adduction, a greater percentage of players receiving “ISTM plus stretching” experienced a successful outcome than did players receiving “stretching only” (89% vs. 44%, respectively, P = .021).

The NNT statistics were calculated for the rates of unsuccessful outcomes based on injury risk qualification (TABLE 4.3). For the injury risk qualification of ‘all categories’, the failure rates were 30% for players in the “ISTM plus stretching” group and 50% for players in the “stretching only” group, resulting in a NNT of 5.0 (95% CI: 4.7, 5.4). This indicates that for a player with ‘both
categories’ of PST, approximately 5 players would need to be treated with ISTM and stretching, to prevent 1 unsuccessful outcome with stretching only. For the injury risk qualification of ‘total arc of motion plus internal rotation’, the failure rates were 36% for players in the “ISTM plus stretching” group and 43% for players in the “stretching only” group, resulting in a NNT of 14.3 (95% CI: 10.7, 17.9). Therefore, for a player qualifying with deficits in ‘total arc of motion plus internal rotation’, approximately 14 players would need to be treated with ISTM and stretching, to prevent 1 unsuccessful outcome with stretching only. Finally, for the injury risk qualification of ‘horizontal adduction’, the failure rates were 11% for players in the “ISTM plus stretching” group and 56% for players in the “stretching only” group, resulting in a NNT of 2.2 (95% CI: 2.1, 2.4). As a result, for a player qualifying with deficits in ‘horizontal adduction’, approximately 2 players would need to be treated with ISTM and stretching, to prevent 1 unsuccessful outcome with the stretching only intervention.

4.5. DISCUSSION

The primary results of this study indicate that baseball players with posterior shoulder tightness receiving ISTM plus stretching experience greater gains in ROM and possible decreases in injury risk when compared to those receiving stretching alone. The gains in internal rotation, total arc of motion, and horizontal adduction gains with “ISTM plus stretching” were approximately double those displayed with “stretching only” (Figure 4.8). Furthermore, this is the first study to report the added treatment benefits of ISTM for resolving posterior
shoulder tightness in a sample of “high risk” baseball players. The injury risk differences observed between categories of posterior shoulder tightness would indicate that players are likely to respond differently to treatment based on the specific impairments of posterior shoulder tightness (e.g. total arc plus internal rotation and/or horizontal adduction). Together these findings indicate that a single treatment session may effectively resolve shoulder ROM deficits and potentially decrease injury risk in a time-efficient manner.

The results of this study and previous research suggest that the ROM deficits associated with posterior shoulder tightness are responsive to conservative therapeutic interventions. In fact, the internal rotation gains of +12° observed within the ‘ISTM and stretch’ group are comparable to 4-6 week stretching programs ranging from +12-15°. In contrast, Laudner et al examined the acute gains in internal rotation for healthy baseball players following a single bout of sleeper stretching. The authors report fewer internal rotation (+3°) gains compared to our results, however, players did not specifically display baseline internal rotation deficits. Furthermore, we combined the use of sleeper and cross-body adduction stretching, which resulted a longer total end range time (4 minutes versus 1.5 minutes). These differences in player characteristics and therapeutic dosage may account for the dissimilar results between studies.

Recently, deficits in horizontal adduction have recently been linked to greater injury risk among baseball players. The results of this study indicate that players receiving “ISTM plus stretching” gained significantly more horizontal
adduction compared to “stretching only” (+14° versus +7°, respectively). Studies by Maenhout\textsuperscript{14} and Tyler\textsuperscript{32} and colleagues show similar gains in horizontal adduction (+11-17°) over the course of 6-7 treatment weeks. These gains are much greater than the reported outcome for the previous acute treatment study by Laudner and colleagues, which only showed marginal gains of +3° for horizontal adduction. Collectively, it may be reasonable to infer from these studies that when identifying patients with posterior shoulder tightness, the application of ISTM and stretching will significantly improve total arc of motion, internal rotation and horizontal adduction.

In this study, the ROM treatment response was influenced by the relative humeral torsion (side-to-side) difference. A key difference between these results and prior studies was the consideration of internal rotation deficits in context of total arc of motion for qualification of ROM deficits. We observed similar relationships between humeral torsion and internal rotation ($r = .386, P > .001$) and horizontal adduction ($r = .287$, $P = .013$) deficits for both groups as previous literature.\textsuperscript{5, 19, 22, 23} Furthermore, in the “stretching only” group the remaining posttest horizontal adduction deficit was correlated with humeral torsion ($r = .471$, $P = .009$) and greater injury risk (Table 3). Considering these results, when stretching is ineffective ISTM may be beneficial for reducing horizontal adduction deficits and subsequent injury risk.

The NNT statistic may impact clinical decision making after identifying players by injury risk category. When comparing the application of “ISTM plus stretching” to “stretching only” the NNT was approximately 2 for players with
‘horizontal adduction’ injury risk (deficits $\geq 15^\circ$). This indicates that for every 2 players (with ‘horizontal adduction’ injury risk) treated with “ISTM plus stretching”, an unsuccessful outcome will be avoided in 1 of those 2 players that would have otherwise occurred if treated with “stretching only”. In contrast, the NNT for was approximately 14 for ‘total arc of motion plus internal rotation’ injury risk (deficits $\geq 15^\circ$). This indicates that for every 14 players (with ‘total arc of motion plus internal rotation’ injury risk) treated with “ISTM plus stretching”, an unsuccessful outcome will be avoided in 1 of those 14 players that would have otherwise occurred if treated with “stretching only”. Considering these differences, it may be more beneficial to treat players with horizontal adduction deficits with ISTM versus ‘total arc of motion plus internal rotation’ deficits if time and resources are limited in a clinical setting to decrease the associated injury risk. Further research is required to determine the benefits of using injury risk categories with other treatment interventions for clinical decision making.

Manske et al$^{15}$ examined the additive treatment effects of glenohumeral joint mobilizations and stretching in a randomized control trial over 4-weeks. In contrast to our results, they concluded no additive treatment benefit for posterior joint mobilizations as both groups showed similar gains in internal rotation. Our mean data show that players receiving ISTM plus posterior shoulder stretching gained significantly more internal rotation and horizontal when compared to the “stretching only” cohort. These differences may indicate that manual therapy techniques directed at the musculotendinous tissue, as performed in our study, are perhaps more beneficial for improving ROM deficits than posterior joint
mobilizations focused on altering capsuloligamentous restraint.

Interestingly, we also observed that the PSS (91.8 ± 13.5) and FAST (14.0 ± 13.7) scores suggested that on average, players were participating with at least low-levels of pain and/or disability. This was unanticipated finding, as all players were currently participating without restrictions or modifications to position and indicated not having pain when asked (do you currently have arm pain? yes/no) on an activities questionnaire. Perhaps this finding indicates a subclinical level of impairments that is present in players with ROM deficits of at least 15°, however we have no normative data for comparison to support this hypothesis.

The specific mechanism(s) driving the ROM deficit reductions with the application of ISTM is unknown. Alterations in musculotendinous morphology and neural modulation are potential sources to explain the acute changes in ROM. There is limited research which suggests that muscular stiffness of posterior shoulder (infraspinatus, teres minor, and teres major), is related to internal rotation deficits in healthy individuals. The ROM changes in this study support this mechanism as players significantly gained internal rotation and horizontal adduction with ISTM application directed at posterior shoulder. Advocates of ISTM suggest that myofascial adhesions are being released citing animal studies, however, the marked acute increases in ROM observed in this study would indicate that this was not the primary mechanism. Evidence of stretching programs for the quadriceps and triceps surae muscles have shown decreased muscle stiffness (N/cm) and diminishing reflex sensitivity suggestive of a central mediated response. Based on the current study design, we are
unable to determine if these mechanisms were associated with our observed outcomes. Future studies should examine the musculotendinous morphology and neural mediation to better understand the underlying mechanisms and improve the treatment selection.

**Limitations of the Study**

The study limitations should be considered when examining these results. First, the isolated and long-term benefits of ISTM are unknown. Future research should be conducted to understand the dose and temporal responses to a comprehensive manual therapy and posterior shoulder-stretching regimen. Also, we are unable to determine which specific tissue(s) were responsible for these ROM changes. The inability to quantify mechanical tissue changes currently serves as a void within the literature. A better understanding of these mechanisms may help to focus therapeutic interventions on the tissue(s) most likely to respond. Future studies should be conducted to collectively identify the tissues responsible for changes in passive shoulder ROM within these players. Lastly, while the results observed within this study may not be generalizable to those with injury, it is reasonable to assume that the use of ISTM could be safely applied to a patient population.

**Clinical Significance**

Based on the findings of this study, we recommend the use of ISTM in conjunction with posterior shoulder stretching for improving ROM and potential injury risk among baseball players with posterior shoulder tightness. Previous research has shown that players often develop deficits in shoulder ROM
following competitive exposures and that these deficits (5° for total arc of motion, 20° for internal rotation and 15° for horizontal adduction) are demonstrated risk factors for arm injury. To address these deficits, our results indicate that ISTM plus stretching may acutely double the ROM gains of stretching alone. Furthermore, while injury risk benefits of ISTM and stretching appear to be equitable for players with total arc of motion and internal rotation deficits, horizontal adduction deficits were more responsive to the combined application of ISTM plus stretching. Therefore, clinicians should consider stretching only for players presenting with total arc of motion and internal rotation deficits, and ISTM plus stretching for players presenting horizontal adduction deficits. Given the association between ROM deficits and arm injury clinicians treating patients with posterior shoulder tightness should consider ISTM in addition to a posterior shoulder stretching program. It may also be clinically beneficial to categorize and treat players based on their specific ROM deficits, and recommend future study be directed towards understanding the effectiveness of these interventions for injury prevention and symptom resolution.

4.6. CONCLUSION

The results of this study show that baseball players exhibiting ROM deficits can acquire clinically meaningful gains in ROM with the acute application of ISTM and posterior shoulder stretching. The addition of ISTM with stretching appears to significantly augment treatment effectiveness when compared to stretching alone. This suggests that the combination of these interventions may be more
beneficial to restore ROM in baseball players with ROM deficits than isolated stretching. Additional studies are required to determine the lasting benefits of these interventions and the underlying mechanism(s) posterior shoulder tightness.
4.7. References


### TABLE 4.1. Subject Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>ISTM &amp; Stretching (n=30)</th>
<th>Stretching Only (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± SD), y</td>
<td>18.8 ± 2.6</td>
<td>18.6 ± 2.1</td>
</tr>
<tr>
<td>Height (mean ± SD), cm</td>
<td>184.1 ± 6.0</td>
<td>182.1 ± 6.7</td>
</tr>
<tr>
<td>Weight (mean ± SD), lbs</td>
<td>187.2 ± 24.3</td>
<td>177.8 ± 20.9</td>
</tr>
<tr>
<td>Arm Dominance (Right / Left)</td>
<td>27 Right, 3 Left</td>
<td>29 Right, 1 Left</td>
</tr>
<tr>
<td>PSS Score (mean ± SD)(^a)</td>
<td>91.3 ± 6.4</td>
<td>92.1 ± 8.4</td>
</tr>
<tr>
<td>FAST Score (mean ± SD)(^b)</td>
<td>15.4 ± 13.9</td>
<td>12.9 ± 13.6</td>
</tr>
<tr>
<td>Level of Competition</td>
<td>9 High School, 21 Collegiate/Pro</td>
<td>12 High School, 18 Collegiate/Pro</td>
</tr>
<tr>
<td>Playing Position</td>
<td>11 Pitchers, 18 Position Players</td>
<td>13 Pitchers, 17 Position Players</td>
</tr>
</tbody>
</table>

\(^a\)PSS Scores are reported as raw totals of possible 100 points; \(^b\)FAST Scores are reported as % of disability
<table>
<thead>
<tr>
<th>Variable</th>
<th>ISTM &amp; Stretching (n = 30)</th>
<th>Stretching Only (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pretest</td>
<td>Posttest</td>
</tr>
<tr>
<td><strong>External Rotation (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dominant</td>
<td>110.5 ± 9.6</td>
<td>112.3 ± 9.1</td>
</tr>
<tr>
<td>Non-Dominant</td>
<td>105.6 ± 8.6</td>
<td>105.1 ± 8.2</td>
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<tr>
<td><strong>Internal Rotation (°)</strong></td>
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<td></td>
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<tr>
<td>Dominant</td>
<td>20.7 ± 10.9</td>
<td>32.8 ± 10.5</td>
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<tr>
<td>Non-Dominant</td>
<td>44.5 ± 11.3</td>
<td>45.6 ± 10.2</td>
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<tr>
<td><strong>Total Arc of Rotation (°)</strong></td>
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<tr>
<td>Dominant</td>
<td>131.2 ± 13.7</td>
<td>145.2 ± 13.3</td>
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<tr>
<td>Non-Dominant</td>
<td>150.2 ± 10.4</td>
<td>150.8 ± 10.6</td>
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<td><strong>Horizontal Adduction (°)</strong></td>
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<tr>
<td>Dominant</td>
<td>-2.2 ± 9.3</td>
<td>11.3 ± 8.0</td>
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<tr>
<td>Non-Dominant</td>
<td>14.6 ± 7.8</td>
<td>13.7 ± 6.6</td>
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<td><strong>Humeral Torsion (°)</strong></td>
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<tr>
<td>Dominant</td>
<td>13.9 ± 8.6</td>
<td>13.3 ± 8.1</td>
</tr>
<tr>
<td>Non-Dominant</td>
<td>33.0 ± 7.4</td>
<td>33.5 ± 6.6</td>
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</table>

*Δ = Posttest - Pretest ROM in degrees; * Indicates statistically significant differences between treatment groups, $F_{(1,59)}$: ($P < .050$)
<table>
<thead>
<tr>
<th>Risk of Injury: Any Criteria</th>
<th>ISTM Plus Stretching (n = 30)</th>
<th>Stretching Only (n = 30)</th>
</tr>
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<tbody>
<tr>
<td>Treatment Failure Rate</td>
<td>9/30 (30%)</td>
<td>15/30 (50%)</td>
</tr>
<tr>
<td>Absolute Risk Reduction (95%CI)</td>
<td>.20 (.186, .214)</td>
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<tr>
<td>Number-Needed-To-Treat (95%CI)</td>
<td>NNT with ISTM plus stretching (versus stretching only) to prevent another unsuccessful outcome: 5.0 (4.7, 5.4)</td>
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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk of Injury: ‘Total Arc of Motion plus Internal Rotation’ Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Arc plus Internal Rotation Gain</td>
</tr>
<tr>
<td>Treatment Failure Rate</td>
</tr>
<tr>
<td>Absolute Risk Reduction (95%CI)</td>
</tr>
<tr>
<td>Number-Needed-To-Treat (95%CI)</td>
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<table>
<thead>
<tr>
<th>Risk of Injury: ‘Horizontal Adduction’ Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horizontal Adduction Gain</td>
</tr>
<tr>
<td>Treatment Failure Rate</td>
</tr>
<tr>
<td>Absolute Risk Reduction (95%CI)</td>
</tr>
<tr>
<td>Number-Needed-To-Treat (95%CI)</td>
</tr>
</tbody>
</table>

*Indicates statistically significant differences between groups (chi-square test; \( P < .05 \)). The absolute risk reduction (ARR) was calculated as \(|\text{Rate ISTM Plus Stretching} - \text{Rate Stretching Only}|\). The NNT was calculated as \(1/(\text{ARR})\).
4.9. FIGURES

FIGURE 4.1. Study Design

Screened for Presence of PST (N = 276)

Qualified for study (n = 126)

Enrolled (n = 60)

Randomization

(30) - Experimental ISTM & Stretch

(30) - Control Stretching Only
FIGURE 4.2. Humeral Torsion Value. Alignment of greater and lesser tuberosity apices relative to the epicondylar axis.

FIGURE 4.4. Sleeper Stretch

FIGURE 4.5. Cross-Body Adduction Stretch
FIGURE 4.6. Instrumented Soft-Tissue Mobilizations. Parallel treatment direction

FIGURE 4.7. Instrumented Soft-Tissue Mobilizations. Perpendicular treatment direction
FIGURE 4.8. Range Of Motion Gain. *Indicates statistically significant differences between treatment groups ($P < .050$)
5.1. ABSTRACT

**Background:** Posterior shoulder tightness (PST) has been associated with increased injury risk among baseball players. There is a current lack of consensus regarding the specific tissues responsible for these deficits in range of motion (ROM).

**Hypothesis:** Changes in rotator cuff stiffness will be related to acute ROM deficit reductions.

**Study Design:** Randomized Controlled Trial, Level of Evidence, 1.

**Methods:** 60 asymptomatic baseball players (19 ± 2 years) with PST (defined as dominant total arc of motion and/or horizontal adduction deficit ≥ 15°) were enrolled, receiving a single treatment of posterior shoulder stretching and instrumented soft-tissue mobilizations (n = 30), or stretching alone (n = 30). Shoulder ROM, instrumented glenohumeral joint translation, humeral torsion, and rotator cuff stiffness were examined before and after the intervention. A 3-way analysis of variance (group x side x time) was used to determine the treatment effects of each dependent variable. Bivariate Pearson correlation coefficients (r)
were used to determine the relationships between ROM deficits and mechanisms.

**Results:** Rotator cuff stiffness decreased with manual therapy ($F_{(1,59)} = 3.90$, $P = .050$) and was related to deficits reductions of IR ($r = .35$, $P = .034$) and HA ($r = .44$, $P = .008$). No treatment effects were observed for A/P translation or humeral torsion between groups or over time ($P > .05$). Players receiving ISTM plus stretching displayed additional increases in total arc of motion ($+5^\circ \pm 2^\circ$, $P = .010$), internal rotation ($+6^\circ \pm 2^\circ$, $P = .010$), and horizontal adduction ($+7^\circ \pm 2^\circ$, $P = .004$) when compared to stretching only.

**Conclusion:** Of the three local mechanisms of PST assessed in this study 1) bony morphology, 2) capsuloligamentous stability, and 3) musculotendious stiffness; posterior rotator cuff stiffness was the only tissue to respond concurrently with deficit reductions.

**Clinical Relevance:** Soft-tissue interventions applied to the posterior shoulder may provide added benefits to self-stretching by reducing muscle stiffness and ROM deficits of PST. Future studies should examine the long-term effects of these treatments over multiple days and throughout the course of a competitive season.

**Key Terms:** posterior shoulder tightness, glenohumeral internal rotation deficit (GIRD), instrumented soft-tissue mobilization (ISTM), baseball.

**What is known about the subjects:** Recent evidence shows that baseball players with dominant-sided deficits in shoulder range of motion are at an increased risk
of injury. However, there is no current evidence to guide clinicians for treating the known tissue(s) responsible for these deficits.

*What this study adds to existing knowledge:* This is the first study to consider each of the mechanical contributions to posterior shoulder tightness (bony morphology, capsuloligamentous stability, and rotator cuff stiffness) and demonstrates that instrumented manual therapy in conjunction with shoulder stretching significantly improves ROM deficits and rotator cuff stiffness in baseball players displaying PST.

### 5.2. INTRODUCTION

Baseball players often exhibit deficits (dominant versus nondominant) in shoulder range of motion (ROM).\(^5\),\(^{24}\),\(^{31}\) Recently, investigators have discovered that large ROM deficits are strongly related to the development of upper extremity injuries.\(^{36}\),\(^{45}\) Specifically, glenohumeral internal rotation deficit (GIRD, internal rotation deficit > 20\(^\circ\)),\(^9\) total arc of motion (TARC, total arc of motion deficit > 5\(^\circ\)),\(^{46}\) and horizontal adduction loss (horizontal adduction deficit > 16\(^\circ\))\(^{34}\) have been prospectively linked to the development of upper extremity injuries in baseball players. Based on these results the clinical prevention of future injury is reliant upon addressing these ROM deficits. In consideration of this clinical impact and consistency in terminology, we have operationally defined these conditions as posterior shoulder tightness (PST) for the purposes of this manuscript.

While the clinical presentation of PST is becoming more clearly defined, there is a lack of consensus regarding the specific tissue(s) responsible for
contributing the deficits of PST.\textsuperscript{6} Many believe that structural adaptations to the bone and/or soft-tissues are responsible for the deficits in ROM, and there is some limited evidence to support these theories.\textsuperscript{8, 11, 25, 28-30, 32, 37, 42} Baseball players commonly exhibit dominant humeral torsion differences that are related to specific impairments of PST [internal rotation (IR) and horizontal adduction (HA)].\textsuperscript{11, 28-30, 32} Others attribute thickening and contractures of the posterioinferior joint capsule to the presence of PST.\textsuperscript{8, 37, 42} Finally, there is some to suggest that PST may be the result of increased posterior rotator cuff stiffness.\textsuperscript{18} Regardless of the proposed mechanisms, a better understanding of the underlying mechanisms responsible PST is important for advancing the effectiveness of therapeutic interventions.\textsuperscript{22, 40} An improved understanding will likely aid in the development of tailored treatment interventions designed to resolve the potential injury risk factor(s). To our knowledge, there are no studies that have collectively examined the local physiologic mechanisms of PST (bony, musculotendinous, and capsuloligamentous). Ultimately, this knowledge should improve clinicians’ ability to select interventions to effectively address the modifiable risk factor(s) that characterize PST.

The purpose of this randomized controlled trial is to account for each of the suspected physiologic mechanisms of PST and track the specific tissue changes that occur with therapeutic intervention. We hypothesize that instrumented soft-tissue mobilizations (ISTM) plus stretching will significantly improve deficits in PST, and that changes in rotator cuff stiffness are primarily responsible for acute gains in ROM compared to stretching alone.
5.3. METHODS

Study Design

This randomized controlled trial was conducted during the 2013 scholastic baseball season. All participants/guardians completed an informed consent form approved by the Greenville Hospital Systems Institutional Review Board.

Participants

Sixty local baseball players with PST were enrolled in this study (TABLE 5.1). Inclusion criteria were; males ages 15 years or older, pitchers or position players, current participation with an organized baseball team, and PST on the dominant throwing arm, as defined by our criteria. Exclusion criteria were a history of shoulder pain within the past 3-months that led to the inability to participate in some or all team activities, and previous surgical history of either shoulder.

PST was defined as a side-to-side deficit ≥ 15° in TARC (and IR), or HA on the dominant throwing shoulder. These criteria were chosen as previous research has indicated that a dominant-sided ROM deficit of 15-20° is markedly associated with prospective injury. Therefore, we set a deficit threshold of ≥ 15° to identify those at higher risk and potentially prevent future injury.

Testing Procedures

Participants underwent an initial screening including passive shoulder ROM and completion of an activities questionnaire to determine eligibility for participation. Qualifying participants were asked to complete a Pennsylvania Shoulder Scale (PSS) and Functional Arm Scale for Throwers (FAST) and return
at least 24-hours after screening for testing to ensure the presence of PST over multiple days. The PSS is a 100-point scale that was used to determine the level of pain and disability for each participant (lower score = greater pain and disability). Players with > 30% disability on the PSS were not eligible for participation. The FAST is a 100-point functional scale developed for overhead throwers and was chosen to provide a sport specific assessment of function (higher scores = more sport-related pain and disability). Participants were randomly allocated by means of random drawing into one of two intervention groups (Figure 1). Each participant underwent pretest and posttest measures of passive shoulder ROM, HT, rotator cuff stiffness, and A/P glenohumeral translation immediately before and after treatment. The PI was blinded to group assignment and was not present during treatment for a standardized length of 10 minutes while treatment was provided by one of two physical therapists, each with over 15 years of orthopedic clinical experience. Players allocated to the experimental group (n = 30) received 4-minutes of supervised posterior shoulder stretching followed by 4-minutes of ISTM. Players in the control group (n = 30) received only 4-minutes of supervised posterior shoulder stretching.

**Shoulder Range of Motion**

*Range of Motion Assessment.* ROM measures were performed as previously described\(^3,36\) using a Baseline Digital Inclinometer (Fabrication Enterprises, Inc; White Plains, NY). Participants were positioned in supine on a plinth in 90° of shoulder abduction and elbow flexion with a towel-roll under the arm to maintain the position of the humerus. One clinician stabilized the scapula,
while a second examiner measured all angles for each of the ROM measures. IR and ER were assessed by passively rotating the shoulder until the examiner felt movement at the coracoid process. A second investigator then aligned the digital inclinometer along the ulnar border and recorded the corresponding angle in degrees. HA was also assessed with the subject in supine while the scapula was stabilized in full retraction and the humerus passively horizontally adducted maintaining neutral humeral rotation until resistance was felt. A second investigator measured the corresponding humeral HA angle using the digital inclinometer at the humeral diaphysis relative the vertical plane.

Measurement reliability for this sample was acceptable for shoulder IR [intra-class correlation coefficients (ICC(2,1)) = .99; 95% confidence interval (95% CI): .98, .99; standard error of measure (SEM) = 1.3°; minimal detectable change (MDC95) = 3.7°], ER (ICC(2,1) = .98; 95% CI: .98, .99; SEM = 1.5°; MDC95 = 4.0°), and HA (ICC(2,1) = .99; 95% CI: .99, .99; SEM = 1.3°; MDC95 = 3.7°). Passive TARC was calculated for each arm using methods previously described by adding the mean ER and IR for the respective side. To determine clinical meaningfulness, we used the relative injury risk deficits (dominant versus nondominant) of IR, TARC and HA for data analysis.

**Humeral Torsion Assessment**

Humeral torsion (HT) was assessed by an examiner with 5 years of experience in musculoskeletal ultrasonography using validated techniques. Participants were positioned supine on a plinth in 90° of shoulder abduction.
and elbow flexion. A SonoSite - Edge (SonoSite Inc. Bothell, WA, USA) ultrasound imaging unit with 4cm linear array transducer (6-15MHz) was used to collect all measures. The probe was placed on the participant’s shoulder at the level of the biceps groove and oriented perpendicular the plane of the floor and verified with a bubble level (FIGURE 5.2). A second examiner then passively rotated the subject’s humerus until the apices of the greater and lesser tuberosities were oriented parallel to coronal plane (FIGURE 5.3). The second examiner then measured the corresponding angle using the digital inclinometer (Figure 2B). The reliability for HT was acceptable (ICC2,1 = .99; 95% CI: .98, .99; SEM = 1.3°; MDC95 = 3.5°). Posttest humeral torsion was only measured the first ten subjects to establish that there were no bony changes occurring with these interventions ($F_{(1,9)} = .63, P = .443$).

**Tissue Elastography**

A novel technique was used to measure compressive rotator cuff stiffness (infraspinatus muscle) previously demonstrating construct validity when compared to the known stiffness of a polyvinyl alcohol cryogel (PVA-C) phantom modulus.$^{39}$ Bilateral stiffness was measured with the player in prone with the testing arm placed in 90° of shoulder abduction, elbow flexion and neutral rotation (Figure 4). An ultrasound imaging system (SonoSite – Edge® SonoSite Inc. Bothell, WA, USA) was used to assess rotator cuff stiffness by placing the transducer within the long-axis of the infraspinatus muscle at the standardized viewing location of the spinoglenoid notch (FIGURE 5.5). Once the appropriate imaging position was obtained, the probe placement was traced with an indelible
marker to ensure consistency between testing periods. A mechanical stress of approximately 10 N (1.0 kg) was delivered and recorded by a force transducer mounted on the ultrasound probe (Figure 3B). A cine-loop was synchronously recorded and stored on the hard-drive of the ultrasound unit to capture the tissue strain of the infraspinatus muscle. Compressive strain was calculated by measuring the change in infraspinatus muscle thickness from rest to maximal stress. Three trials were collected and averaged for data analysis.

Young’s elastic modulus (\(E\)) was used for post-processing estimation of tissue stiffness based on the function of applied stress (\(\sigma\)) and resultant tissue strain (\(\varepsilon\)). Higher values of Young’s Modulus are indicative of increased tissue stiffness while lower values correspond to decreased tissue stiffness. We used the following formulas to measure rotator cuff stiffness;

\[
\sigma = \frac{F}{A}; \quad \varepsilon = \frac{\Delta L}{L_0}; \quad E = \frac{\sigma}{\varepsilon}
\]

Stress (\(\sigma\)) was calculated as an applied force (\(F\)) over a surface area (\(A\)), while strain (\(\varepsilon\)) was the change in the tissue length (\(\Delta L\)) from the original resting length (\(L_0\)). This relationship was computed given a known transducer size (area) and the measurement of the applied force at the ultrasound transducer. The resting length (\(L_0\)) and change in length (\(\Delta L\)) was measured from the resulting ultrasound images and expressed in kilopascals (kPA). This method demonstrated acceptable reliability (ICC\(_{2,1}\) = .714; 95% CI: .58, .83; SEM = 0.53 kPa; MDC = 1.5kPa).

**Glenohumeral Joint Mobility**

Glenohumeral joint translation was assessed with an electromagnetic
tracking system using procedures previously described by Tibone et al.\textsuperscript{38} and Sethi et al.\textsuperscript{33} (Flock of Birds, Ascension Technology Corp., Burlington, VT). Kinematic data were processed using the Motion Monitor (Innovative Sports Training Inc., Chicago, IL, USA) analysis software and reduced using Matlab programming (MathWorks Inc., Natick, MA, USA).

An electromagnetic receiver transmitted a 3-dimensional global coordinate system for kinematic analysis. Position sensors were affixed transcutaneously to the thorax, acromion process and proximal humerus of both shoulders (Figure 3A). Cartesian coordinates from the 2 sensors of the 1) acromion process and 2) humeral head were used for joint translation measurement. A/P translation was calculated by subtracting the absolute vector of humeral displacement from the absolute vector displacement of the scapula, producing a measure of isolated humeral head translation. This relative vector includes motions of X, Y, and Z coordinates to account for the obligate out-of-plane movement that occurs with respect to the osseous joint congruency. For this sample, a secondary regression analysis shows that anteroposterior (A/P) translation on the x-axis accounted primarily for the total humeral head translation ($R^2 \geq .87$, $P < .001$) while Y, and Z planes of movement were not statistically significant ($P > .05$).

We selected a procedure identical to the anterior-posterior drawer tests initially described by Gerber and Ganz\textsuperscript{15, 33} to simulate clinical relevance for measuring joint translation. A/P translations were performed with the subject lying supine with the humerus positioned in 90\degree of abduction and neutral rotation (FIGURE 5.4). For each trial the start position was attained by seating the
humeral head within the glenoid fossa using joint compression and axial loading of the humerus as previously described by Hawkins et al.\textsuperscript{17} Once the start (centered) position was achieved, an anterior-directed force was slowly applied to the humerus until a capsular end-point was reached, then followed subsequently by the posterior-directed force. Each shoulder was taken through five successive trials of A/P translations with the average of trials 2-4 used for data analysis. Anterior, posterior, and total A/P translation were analyzed and demonstrated acceptable intrasession reliability ($\text{ICCs}_{(2,1)} = .98$; 95\% CI: .96, .99; $\text{SEM} = 0.1 \text{ cm}$; $\text{MDC}_{95} = 0.3 \text{ cm}$).

**Treatment Interventions**

Measurements were performed on both shoulders immediately before and after the treatment interventions. The PI was blinded to group assignment and was not present during treatment for a standardized length of 10 minutes while a co-investigator applied the treatment. Upon completion of the interventions the PI was asked to return and complete subsequent posttest measures. The control group ($n = 30$) was given standardized instruction in the performance of sleeper and cross-body adduction stretches, which have shown to effectively decrease PST in previous studies.\textsuperscript{20, 22, 23, 41} The participants performed each stretch for one minute of 2 repetitions and a 30-second rest period between repetitions. The treating therapist timed the treatment and assessed for appropriate stretching technique.

The experimental group ($n = 30$) performed the previous stretches then immediately received instrumented soft-tissue mobilizations targeting the
infraspinatus and teres minor muscles. Subjects were placed in prone for ISTM (SASTM; Carpal Therapy Inc, Indianapolis, IN) with the dominant arm positioned in neutral rotation at 90° of shoulder abduction and elbow flexion (Figure 4). Emollient was applied to the posterior axillary border and treatment strokes were administered for two minutes following a metronome set at 45 hertz. The treatment angle was held consistent at approximately a 45° angle to the skin’s surface in direction both parallel and perpendicular to the fiber alignment of the infraspinatus and teres minor muscles.

**Statistical Analysis**

A one-way ANOVA was used to compare pretest age, height, weight, ROM, HT, A/P translation, rotator cuff stiffness, subjective outcomes (PENN and FAST), and level of competition between treatment groups. Separate linear 3-way mixed-model ANOVAs (group x arm x time) were used to determine the treatment efficacy of the interventions by measuring each ROM and mechanical tissue change variable over time. Planned post-hoc comparisons were made for the three-way interactions effects including group for all ROM variables of interest (IR gain, TARC gain, and HA gain) and the mechanical tissue changes (HT, rotator cuff stiffness loss, and A/P translation gain). Gains in ROM and mechanical tissue change were calculated as posttest measure – pretest measure.

Bivariate Pearson correlation coefficients (one-tailed) were then used to determine the effectiveness of the intervention as related to injury risk by calculating the relationship of posttest ROM deficits to the mechanical tissue
changes. Only relationships between the ROM deficits of interest (IR, TARC and HA) were included in the analysis. Posttest ROM deficits were calculated as nondominant ROM – dominant ROM measure (IR deficit, TARC deficit, and HA deficit). Simple linear regression models for ROM were then calculated based including only the significantly correlated mechanical variables to account for the influence on ROM deficits. PASW Statistic 18 software (SPSS Inc., Chicago, IL, USA) was used all statistical procedures and statistical significance was set a priori at α = .05.

5.4. RESULTS

Pretest Comparisons

There were no baseline group differences for any of the demographic variables (TABLE 5.1), or dependent variables (shoulder ROM, A/P translation, or rotator cuff stiffness) (TABLE 5.2). The control group displayed greater humeral retrotorsion (+6° ± 3°) on the nondominant arm when compared to the experimental group ($F_{(1,59)} = 4.45$, $P = .038$). At baseline, there were no group differences in TARC, IR, ER, HA or side-to-side deficits ($P > .05$). Overall, players displayed significant ($P < .001$) dominant-sided deficits in IR (26° ± 11°), TARC (18° ± 11°), and HA (17° ± 11°), representing the magnitude of PST within this sample.

Range of Motion Treatment Comparisons

There was a significant three-way interaction effect (group x side x time) for IR ($F_{(1,59)} = 7.05$, $P = .010$), TARC ($F_{(1,59)} = 7.10$, $P = .010$), and HA ($F_{(1,59)} =$
9.25, \( P = .004 \). Post hoc analysis revealed that the experimental group gained 5° (± 2°) IR, 8° (± 6°) TARC and 7° (± 2°) HA when compared to the control group. The average data indicate that ROM deficits were more effectively reduced with the combined application of ISTM and stretching (70% versus 55%).

**Posterior Rotator Cuff Stiffness**

There was a significant 3-way interaction effect (group x side x time) for rotator cuff stiffness (\( F_{(1.59)} = 3.90, P = .050 \)). Post hoc testing indicates that rotator cuff stiffness of the dominant arm decreased significantly in the experimental group (Figure 5A) and not the control group (-0.4 ± 0.09 kPa, versus -0.1 ± 0.09 kPa; \( P = .002 \)). This magnitude of change shows that the application of ISTM effectively decreased dominant rotator cuff stiffness to levels beyond the nondominant arm.

**Glenohumeral Translation**

There were no significant interactions involving group or time for A/P translation, meaning the treatment interventions did not influence the amount of A/P translation (\( F_{(1,59)} = .69, P = .410 \); Table 2). There was a significant main effects difference for arm dominance (\( F_{(1,59)} = 25.71, P < .001 \)) suggesting that A/P translation is appreciably diminished in the dominant arm of players with PST. At baseline the average A/P joint translation on the dominant arm was 2.7 cm (± 0.2 cm) compared to 4.1 cm (± 0.3 cm) on the nondominant arm. Despite these baseline differences A/P joint translation was not influenced by these interventions.

**Humeral Torsion**
There were no significant interactions involving group or time for HT as bony morphology was not acutely influenced with these treatment interventions \((F_{(1,9)} = .63, P = .443; \text{Table 2})\). Baseline differences were present between dominant and nondominant arms (TABLE 5.2), however this did not change between groups or over time \((P > 0.05)\).

**Posttest Relationships Between ROM Deficits and Physiologic Mechanisms**

Within the experimental group, there was a significant, moderate correlation between posterior rotator cuff stiffness and posttest ROM deficits in IR and HA \((\text{IR}, r = 0.35, P = .03; \text{HA}, r = 0.44, P = .008)\) (FIGURE 5.6). This suggests that as posterior rotator cuff stiffness was reduced there was a concurrent restoration of ROM symmetry. HT also displayed a significant, moderate correlation with posttest IR deficit \((r = 0.36, P = 0.024)\). Simple linear regression revealed that posterior rotator cuff stiffness and HT were independent contributors to post-treatment IR deficits explaining 23% of the variability \((P = .037)\). This indicates that the resolution of IR deficits was influenced by the change in muscle stiffness mediated through HT. HA deficits were not significantly correlated with HT \((P = .130)\), suggesting that rotator cuff stiffness was the only mechanism measured to influence changes in HA deficits. There were no other significant correlations between ROM and any of the mechanical variables \((P > .05)\).

There were no relationships between rotator cuff stiffness and ROM for the control group. However, there was a negative posttest relationship between posterior translation and HA deficit \((r = -0.38, P = .018)\), signifying that decreased
posterior translation was related to the large and remaining HA deficits after stretching. This relationship was not observed within the experimental group. Furthermore, the influence of HT was present among the influencing factors of HA in the control group \((r = .47, P = .004)\) indicating that increased humeral retrotorsion was associated resolution of HA ROM deficits. Simple linear regression revealed that posterior translation and HT were independent contributors to post-treatment HA explaining 32% of the remaining deficits \((P = .006)\). This suggests that when muscle stiffness remains, HA deficits are related to the magnitude of posterior translation and HT.

5.5. DISCUSSION

This is the first study to account for each of the local mechanisms of PST, (muscle stiffness, joint translation, and bony morphology). Our findings support our original hypothesis that the combined application ISTM plus stretching improves deficits in PST, and that changes in rotator cuff stiffness are primarily responsible for ROM gains. More specifically, the supplemental benefit of ISTM resulted in added gains for IR, TARC, and HA \((IR = 5° \pm 2°, P = .011; TARC = 8° \pm 6°, P = .007; \text{and} \ HA = 7° \pm 2°, P = .003)\). The decreased ROM deficits and injury risk were observed concurrently with the decreases in dominant rotator cuff stiffness.

**Posterior Rotator Cuff Stiffness**

Our results suggest that the use of ISTM was effective in decreasing rotator cuff stiffness, as differences were isolated only to the dominant upper
extremity in players receiving ISTM. These decreases in rotator cuff stiffness were also concurrent with the deficit reductions in IR and HA. While there is limited evidence for comparison, Hung et al\textsuperscript{18} reported similar relationships between shoulder ROM and muscle stiffness (posterior deltoid, infraspinatus and teres major and minor) in patients with adhesive capsulitis. These results provide preliminary evidence to suggest that the mobility of the musculotendinous unit(s) is a primary mechanism influencing ROM impairments at the shoulder.

The significance of these mechanical factors is important when considering injury risk. Baseball players in the experimental group displayed comparative deficit reductions in IR and HA compared to the control group. Regression analysis shows that changes in rotator cuff stiffness partially explained the resolution of PST with $R^2$ values $< 25\%$, suggesting other factors impacted the changes observed in ROM. Among the likely explanations for the observed changes in ROM is a centrally mediated neural response.\textsuperscript{1, 4} Recent studies examining the benefits of stretching have reported decreases in muscle stiffness and reflex sensitivity.\textsuperscript{1, 4} In low back pain, the use of manual therapy has reportedly been shown to elicit down regulation of the muscle spindles.\textsuperscript{16} Considering these results, neuromodulation of the resting muscle tension may account for some of the unexplained variability observed within our sample. Despite these limitations, changes in rotator cuff stiffness did occur concurrently with the application and added ROM benefits of ISTM. Future studies should further examine the musculotendinous unit and neural-mediated influence to better understand the physiologic mechanisms and improve the specificity of the
According to our results, muscle stiffness may also influence posterior translation and HA deficits. Interestingly, following the intervention, the control group continued to exhibit HA deficits ($10^\circ \pm 11^\circ$). These deficits in HA appear to be associated with decreased posterior translation and greater HT, which was not observed in the experimental group. Perhaps the lack of change in rotator cuff stiffness is responsible for these relationships. By providing concavity compression, the rotator cuff is inherently responsible for the active stability of the glenohumeral joint. Considering this mechanism, the present muscle stiffness may have constrained the joint, thereby limiting passive physiologic (posterior translation) and osteokinematic motion (HA).

**Joint Mobility**

Baseline differences did exist between dominant and non-dominant upper extremities, however there were no observed changes in A/P translation over time or between groups. This lack of differences in glenohumeral translation suggests that capsuloligamentous changes were not responsible for the acute gains in ROM. Pretest comparisons (TABLE 5.2) demonstrate that players had less total A/P translation on the dominant arm when compared to the nondominant arm. This is in contrast to previous data suggesting no differences between arms or that greater total A/P translation is present on the dominant arm. These opposing results may be contributed to the specific deficits associated with PST, as this the first study to document total A/P translation within a sample of players with PST. Perhaps chronic adaptations to throwing
influenced the capsular mobility of players within our sample and are partially responsible for the side-to-side differences.

Our results are also in contrast to Laudner et al\textsuperscript{19} who reported that greater anterior translation was associated with measures of PST. However, their sample only displayed a TARC loss of 8° and IR loss of 16°, which is less than in our study. The differences between studies highlight the variability among throwers and may be related to the ability to account for the total dominant A/P translation, the influence of HT, not accounting for nondominant arm as in our study, and age differences between studies. The lack of side-to-side comparisons limits the ability to determine if PST was responsible for these differences or if HT influenced these measures. Our results show no differences in anterior translation between arms or over time. Future studies should investigate the potential capsular adaptations in baseball players over the course of multiple exposures and consecutive seasons.

We observed decreased dominant arm posterior translation at baseline which is consistent with altered translation measured via stressed ultrasonography in professional pitchers.\textsuperscript{7} Additionally, the athletes in the control group demonstrated HA deficits, which were moderately associated with the decreases in posterior translation. Within this group, the remaining posterior rotator cuff stiffness on top of the capsular mobility restrictions may have limited the obligate posterior translation needed for gaining HA. Further investigation is needed to understand the serial development and changes that occur between glenohumeral translation and overhead throwing.
Humeral Torsion

Humeral torsion did not change due to the intervention but was moderately associated with the resolution of IR and HA ROM deficits. HT differences have been clearly linked to alterations in dominant ROM in baseball players, particularly for IR and HA. Players within this sample exhibited greater differences than has been previously reported within the literature ($21^\circ \pm 11^\circ$). This difference was consistent between groups allowing for us to account for the influence of HT in order to clarify the factors contributing to PST. The relationships between side-to-side differences in HT and IR deficit suggest that when rotator cuff stiffness is resolved, HT is the primary factor limiting IR. In contrast when posterior rotator cuff was increased HT had a greater influence on HA deficits. This suggests that ISTM may be helpful to establish baseline ROM measures for throwers when HT can’t be assessed. In contrast when posterior rotator cuff stiffness remains, side-to-side differences in HT appear to influence HA deficits to a greater degree.

Clinical Relevance

Considering the added benefits to ROM and decreased rotator cuff stiffness, we recommend clinicians consider the use of directed ISTM when treating players with PST. Specifically, the focused application of manual therapy techniques to the posterior rotator cuff appears to resolve PST at least in part by decreasing muscle stiffness. When assessing for the presence of PST, clinicians should carefully consider the soft-tissue mobility of the posterior rotator cuff, addressing deficits with a combined static stretching and ISTM regimen.
Limitations

The limitations of this study should be considered when interpreting these results. Subjects in this study displayed heterogeneous characteristics of PST when considering baseline TARC, IR and HA deficits. Despite this variability, those subjects within the experimental group displayed significantly fewer ROM deficits following the intervention when compared to the control group. We feel this diverse presentation in ROM deficits is consistent with the clinical setting and strengthens the generalizability of these interventions. However, not all subjects displayed deficits across all 3 ROM measures possibly contributing to the lack of overall relationships between resolution of ROM deficits and muscle stiffness. Secondly, while the compressive rotator cuff stiffness changed, the lack of significant predictive value for each of the mechanical contributions to PST suggests the likelihood of other influencing factors. Of these potential factors the neural modulation of resting tissue tension may have conceivably influenced the musculotendinous restraint. However, of the potential factors assessed posterior rotator cuff stiffness was the only one that changed concurrent with ROM increases. Lastly, this study was conducted in self-reported, asymptomatic baseball players. However, these players surprisingly displayed PSS and FAST scores that suggest less than optimal shoulder function. While these players did not have enough dysfunction to limit their participation they may have represented a subclinical population. Future studies should confirm these results in those athletes with confirmed pathology, pain and disability to allow for clinical application.
5.6. SUMMARY

The contributing mechanisms of PST in throwers remain elusive. This study shows that the added use of ISTM significantly decreases ROM deficits and posterior rotator cuff stiffness when compared to stretching alone. When considering the local mechanisms of PST assessed in this study 1) bony morphology, 2) capsuloligamentous stability, and 3) musculotendinous stiffness; posterior rotator cuff stiffness was the only tissue to respond to treatment concurrently with ROM improvements. However, the amount of variability explained by these mechanisms was not strongly predictive of the observed gains in ROM or the reduced deficits of PST. Future studies should continue to explore the musculotendinous contributions to PST and other potential mechanisms that contribute to alterations in the shoulder ROM.
5.7. REFERENCES


5.8. TABLES

### TABLE 5.1. Subject Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>ISTM &amp; Stretching (n=30)</th>
<th>Stretching Only (n = 30)</th>
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<tr>
<td>Age (mean ± SD), years</td>
<td>19 ± 2.6</td>
<td>19 ± 2.1</td>
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<tr>
<td>Height (mean ± SD), cm</td>
<td>184 ± 6.0</td>
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<tr>
<td>Weight (mean ± SD), lbs</td>
<td>187 ± 24.3</td>
<td>178 ± 20.9</td>
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<tr>
<td>Arm Dominance (Right / Left)</td>
<td>27 Right, 3 Left</td>
<td>29 Right, 1 Left</td>
</tr>
<tr>
<td>PSS Score (mean ± SD)a</td>
<td>91 ± 6.4</td>
<td>92 ± 8.4</td>
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<tr>
<td>FAST Score (mean ± SD)b</td>
<td>15 ± 13.9</td>
<td>13 ± 13.6</td>
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<tr>
<td>Level of Competition</td>
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<td>12 High School, 18 Collegiate/Pro</td>
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<tr>
<td>Playing Position</td>
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<td>13 Pitchers, 17 Position Players</td>
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</table>

*a*PSS Scores are reported as raw totals of possible 100 points; *b*FAST Scores are reported as % of disability

*There were no differences between groups with statistical significance set *a priori* at $\alpha = 0.05$. 

## TABLE 2. Range of Motion and Mechanical Changes

<table>
<thead>
<tr>
<th>Variable</th>
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<th>ISTM &amp; Stretch Groups</th>
<th>Stretch Only Group</th>
<th>$\Delta_{\text{post-pre}}$</th>
<th>$P$ Value</th>
<th>$\Delta_{\text{post-pre}}$</th>
<th>$P$ Value</th>
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<td>Posttest</td>
<td></td>
<td></td>
<td>Pretest</td>
<td></td>
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<tr>
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<td>112.3 ±9.1</td>
<td>+1.8</td>
<td>.070</td>
<td>114.7 ±10.3</td>
<td>.181</td>
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<td></td>
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<td>.444</td>
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<td>Internal Rotation ($^\circ$)</td>
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<td>32.8 ±10.5</td>
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<td>D</td>
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<td>14.6 ±7.8</td>
<td>13.7 ±6.6</td>
<td>-1.0</td>
<td>.282</td>
<td>19.6 ±12.5</td>
<td>.178</td>
</tr>
<tr>
<td>Humeral Torsion ($^\circ$)</td>
<td>D</td>
<td>13.9 ±8.6</td>
<td>13.3 ±8.1</td>
<td>-0.6</td>
<td>.453</td>
<td>13.0 ±11.2</td>
<td>.992</td>
</tr>
<tr>
<td></td>
<td>ND</td>
<td>33.0 ±7.4</td>
<td>33.5 ±6.6</td>
<td>+0.6</td>
<td>.552</td>
<td>38.8 ±13.0</td>
<td>.780*</td>
</tr>
<tr>
<td>A/P Translation (cm)$^b$</td>
<td>D</td>
<td>3.0 ±1.8</td>
<td>3.2 ±1.6</td>
<td>+0.2</td>
<td>.181</td>
<td>2.6 ±1.2</td>
<td>.030*</td>
</tr>
<tr>
<td></td>
<td>ND</td>
<td>3.8 ±1.9</td>
<td>3.9 ±1.6</td>
<td>+0.1</td>
<td>.879</td>
<td>4.3 ±2.1</td>
<td>.425</td>
</tr>
<tr>
<td>Stiffness (kPa)$^c$</td>
<td>D</td>
<td>1.6 ±0.6</td>
<td>1.2 ±0.3</td>
<td>-0.4</td>
<td>&lt; .001*</td>
<td>1.4 ±0.4</td>
<td>.212</td>
</tr>
<tr>
<td></td>
<td>ND</td>
<td>1.5 ±0.4</td>
<td>1.5 ±0.3</td>
<td>0.0</td>
<td>.483</td>
<td>1.5 ±0.3</td>
<td>.11</td>
</tr>
</tbody>
</table>

$\Delta_{\text{post-pre}}$: Posttest – Pretest change values

$^a$Indicates vector of total glenouhumeral joint translation from A/P (mm)

$^b$Indicates Young’s Elastic Modulus stiffness value of the Infraspinatus (kPa)

$^c$Indicates statistically significant 3-way interaction (Side x Time x Group) between treatment groups, ($F_{(1,59)}$, $P < .050$)
5.9. FIGURES

FIGURE 5.1. Study Design

1. Screened for Presence of PST (N = 276)
2. Qualified for study (n = 126)
3. Enrolled (n = 60)
4. Randomization
   - (30) - Experimental ISTM & Stretch
   - (30) - Control Stretching Only
FIGURE 5.2. Humeral Torsion Value. Alignment of greater and lesser tuberosity apices relative to the epicondylar axis.

FIGURE 5.5. Ultrasound Elastography. Figure demonstrates the placement and direction of compressive stress while the amount of tissue deformation is registered by the ultrasound image.
FIGURE 5.6. Rotator Cuff Stiffness Change. The red line indicates the stiffness changes in the ‘ISTM & Stretching’ group and the black line represents the changes in the ‘Stretching Only’ group.
CHAPTER 6: CONCLUSION OF DISSERTATION

The purpose of this dissertation was to examine the underlying mechanisms of posterior shoulder tightness (PST) by determining the clinical effectiveness of common treatment interventions to improve range of motion (ROM) deficits and injury risk in baseball players with PST. The primary mechanism of PST has been widely debated and frequently reported within the literature. PST is a ROM impairment commonly found in overhead athletes and individuals with shoulder pain. As described in Chapter 2, clear relationships have been established in individuals with PST reporting shoulder pain (Tyler, Nicholas et al. 2010) and the incurrence of prospective injury (Shanley, Rauh et al. 2011; Wilk, Macrina et al. 2011). A better understanding of the mechanical contributors to PST is vital to effectively detecting and treating modifiable risk factors of injury.

Local mechanical restraints to shoulder ROM include bony morphology (Crockett HC, Gross LB et al. 2002; Osbahr, Cannon et al. 2002), capsuloligamentous stability (Crawford and Sauers 2006) and musculotendinous restraint (Myers, Oyama et al. 2009). Recent studies have independently examined the potential influence each of these contributors on shoulder ROM. Despite these comparisons no studies have collectively considered each mechanism within a sample of individuals with PST. To effectively determine the
etiology underlying PST it is essential to collectively consider each of the potential anatomical contributors.

Therapeutic interventions have shown a promising ability to improve shoulder ROM (McClure, Balaicuis et al. 2007; Laudner, Sipes et al. 2008; Manske, Meschke et al. 2010) and pain in overhead athletes (Tyler, Nicholas et al. 2010). Despite these improvements, patients with PST are often treated with homogenous treatment regimens that do regard the potential differences of contributing mechanisms. In fact, recent work suggests that joint mobilizations do not influence the ROM gains when performed with a posterior shoulder stretching regimen (Manske, Meschke et al. 2010), which is contrary to previous the belief that PST is driven by capsuloligamentous contracture (Burkhart, Morgan et al. 2003).

This dissertation research investigates the clinical and mechanical benefits of manual therapy and conventional stretching for resolving PST in an at-risk population. Chapter 3 of this dissertation is a randomized clinical trial investigating the clinical effectiveness of ISTM with a posterior shoulder stretching routine in baseball players with PST. Results suggest that players receiving both ISTM and posterior shoulder stretching had clinically meaningful gains in ROM when compared to those stretching alone. The magnitude of change observed with the acute treatment of ISTM and stretching was twice that of players performing stretching alone. Seventy percent of the athletes that had previously qualified for being at risk of injury were not at risk following the acute treatment of ISTM and stretching. The clinical relevance of these results are that
the added use of ISTM with stretching can elicit significant gains in ROM and dramatically decrease injury risk in individuals with PST.

Chapter 4 of this dissertation research is a randomized controlled trial examining the acute tissue changes occurring within local mechanical contributors of PST. Previous literature has suggested that PST results from primarily from posterior capsular adaptations of the glenohumeral joint (Burkhart, Morgan et al. 2003), however there is currently no clinical data to support these theories (Tibone, Lee et al. 2002; Sethi, Tibone et al. 2004; Crawford and Sauers 2006). Within this study, humeral torsion, glenohumeral joint translation, and rotator cuff stiffness were measured immediately before and after an acute application of ITSM and stretching, or stretching alone. The results of this study indicate that of the tissues measured, rotator cuff stiffness was the only tissue to undergo mechanical change concurrently with ROM gains. This suggests that musculotendinous stiffness is at least partially responsible for the impairments associated with PST.

6.1. Clinical Implications

There are several clinical implications to consider in this dissertation. First, the combined use of ISTM and posterior shoulder stretching appears to effectively reduce the deficits associated with PST. Effectively reducing these ROM deficits is key to lowering prospective injury risk, and ROM impairments associated with the presence and resolution of shoulder pain in overhead athletes (Myers JB, Laudner KG et al. 2006; Tyler, Nicholas et al. 2010). The
focused application of manual therapy techniques to the posterior rotator cuff appears to resolve PST at least in part by decreasing muscle stiffness. Utilization of ISTM may decrease rotator cuff stiffness and subsequent injury risk of PST by specifically reducing deficits in internal rotation and horizontal adduction. Clinicians should consider a focused manual intervention directed towards the posterior rotator cuff when treating players with PST.

6.2. Future Research

Research is warranted to further investigate the relationship between rotator cuff stiffness and ROM deficits in individuals with PST. Specifically, considerations should be made to account for the influence of centrally mediated neural regulation on resting muscle stiffness. Past studies have used electromyography to investigate the influence of central mediation of muscle stiffness and stretch reflex sensitivity for the triceps surae and quadriceps muscles (Avela and Komi 1998; Blackburn, Padua et al. 2008). This application would be the first at the shoulder to consider the only other physiologic mechanism to potentially influence shoulder ROM.

Longitudinal study is needed to establish whether soft-tissue mobilizations are effective in reducing PST among overhead athletes and patients populations. Furthermore, future research should investigate the subgroups of PST, specifically including deficits in total arc of motion, internal rotation, and horizontal adduction. This approach may help to identify the specific impairments and effective treatment interventions associated with the various definitions of
PST. Lastly, further research is needed to determine the impact of preventative soft-tissue mobilization regimens for reducing the incidence of injury in overhead athletes with PST.
6.3. References


REFERENCES


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