


Summer 8-10-2019

Humeral Retroversion, Range of Motion, and Strength Adaptations in Tennis Players

Daniel Hannah

Follow this and additional works at: <https://dsc.duq.edu/etd>

 Part of the [Orthopedics Commons](#), [Other Rehabilitation and Therapy Commons](#), [Sports Medicine Commons](#), and the [Sports Sciences Commons](#)

Recommended Citation

Hannah, D. (2019). Humeral Retroversion, Range of Motion, and Strength Adaptations in Tennis Players (Doctoral dissertation, Duquesne University). Retrieved from <https://dsc.duq.edu/etd/1818>

This One-year Embargo is brought to you for free and open access by Duquesne Scholarship Collection. It has been accepted for inclusion in Electronic Theses and Dissertations by an authorized administrator of Duquesne Scholarship Collection.

HUMERAL RETROVERSION, RANGE OF MOTION, AND STRENGTH
ADAPTATIONS IN TENNIS PLAYERS

A Dissertation

Submitted to the John G. Rangos, Sr. School of Health Sciences

Duquesne University

In partial fulfillment of the requirements for
the degree of Doctor of Philosophy

By

Daniel C. Hannah

August 2019

Copyright by
Daniel C. Hannah

2019

HUMERAL RETROVERSION, RANGE OF MOTION, AND STRENGTH
ADAPTATIONS IN TENNIS PLAYERS

By

Daniel C. Hannah

Approved June 6, 2019

Jason S. Scibek, PhD, LAT, ATC
Chair, Department of Athletic Training
Associate Professor of Athletic Training
(Committee Chair)

Christopher R. Carcia, PhD, PT, OCS, SCS
Chair, Department of Physical Therapy
Program Director, Rehabilitation Science
Associate Professor of Physical Therapy
(Committee Member)

Amy L. Phelps, PhD
Assistant Professor of Statistics
(Committee Member)

Fevzi Akinci, PhD
Dean, John G. Rangos, Sr. School of
Health Sciences
Professor of Health Management Systems

ABSTRACT

HUMERAL RETROVERSION, RANGE OF MOTION, AND STRENGTH ADAPTATIONS IN TENNIS PLAYERS

By

Daniel C. Hannah

August 2017

Dissertation supervised by Jason S. Scibek, PhD, LAT, ATC

Purpose: This study aimed to develop an understanding of humeral retroversion (HRV) asymmetries in tennis players and its impact on physical and performance characteristics of the shoulder.

Participants: Healthy tennis players were categorized into 3 groups: younger juniors (n=11, age=14.5±0.5 years), older juniors (n=12, age=17.1±0.9 years), and collegiate (n=16, age=19.6±1.2 years).

Methods: HRV, internal rotation (IR), and external rotation (ER), total arc of motion (TAM), HRV-corrected IR (HRVcIR), and HRV-corrected ER (HRVcER) were measured and calculated bilaterally using a digital inclinometer and ultrasonography. Bilateral differences (Δ) were calculated (dominant minus nondominant) for HRV and ROM variables. Isometric ER:IR strength ratios were measured and calculated for the

dominant limb using hand-held dynamometry. Paired-sample t-tests and one-way ANOVAs were used to analyze limb-to-limb and group comparisons. Pearson correlation coefficients were used to analyze relationships between HRV and both ROM and strength measures.

Results: HRV was significantly greater in the dominant limb in the younger juniors (dominant $62.8^{\circ} \pm 9.1^{\circ}$ vs nondominant $56.3^{\circ} \pm 6.8^{\circ}$, $p=.039$), older juniors (dominant $75.5^{\circ} \pm 11.2^{\circ}$ vs nondominant $68.6^{\circ} \pm 14.2^{\circ}$, $p=.043$), and collegiate players (dominant $71.7^{\circ} \pm 8.5^{\circ}$ vs nondominant $61.2^{\circ} \pm 6.9^{\circ}$, $p=.001$). Significantly less IR was observed in the dominant arms only in older juniors (dominant $36.9^{\circ} \pm 9.9^{\circ}$ vs nondominant $46.3^{\circ} \pm 11.2^{\circ}$, $p<.001$) and collegiate players (dominant $32.4^{\circ} \pm 7.5^{\circ}$ vs nondominant $40.6^{\circ} \pm 5.4^{\circ}$, $p<.001$); however, no differences were observed in IR when corrected for HRV. No significant age-group differences were observed for HRV and ROM variables. $HRV\Delta$ was significantly correlated with $IR\Delta$ ($r=-0.531$, $p=.001$), $ER\Delta$ ($r=0.654$, $p<.001$), $TAM\Delta$ ($r=0.332$, $p=.039$), $HRVcIR\Delta$ ($r=0.735$, $p<.001$), and $HVcER\Delta$ ($r=-0.330$, $p=.040$). No relationships were observed between HRV adaptations and strength ratios.

Conclusion: Tennis players demonstrate increased HRV in the dominant limb, and it appears that this adaptation may occur mostly before the age of 14. ROM asymmetries appear to be significantly influenced by HRV adaptations. Once HRV was accounted for, ROM asymmetries appeared to neutralize. These findings suggest that correcting ROM measures for HRV may provide a more accurate assessment of shoulder motion adaptations.

Clinical Relevance: Considering that tennis players demonstrate asymmetries in HRV, clinicians should be cautious when screening for and implementing interventions for soft tissue motion deficits based on clinical ROM measures.

ACKNOWLEDGEMENT

Throughout this dissertation process I have received a tremendous amount of support and assistance from various individuals for which I am very grateful. I would like to thank my advisor, Dr. Jason Scibek, and committee members Dr. Chris Carcia and Dr. Amy Phelps for their guidance and feedback. I would also like to thank my wife, Ashley Hannah, for willfully and wholeheartedly taking on this journey with me, and for the love and support provided during this endeavor. And while they may not have fully understood their role during this time, I am so grateful for my sons, Drew and Zach. They were able to refocus my perspective, many times over, on what is important in life.

TABLE OF CONTENTS

	Page
ABSTRACT.....	iv
ACKNOWLEDGEMENT	vii
LIST OF TABLES	xii
LIST OF FIGURES	xiii
Chapter 1	1
Introduction.....	1
1.1 Background.....	1
1.2 Objective.....	7
1.3 Overall Hypothesis.....	7
1.4 Dissertation Organization	8
Chapter 2	10
Review of Literature	10
2.1 Background and Epidemiology.....	10
2.2 The Shoulder Complex	11
2.2.1 The Sternoclavicular Joint.....	12
2.2.2 The Acromioclavicular Joint.....	13
2.2.3 The Scapulothoracic Interface.....	13
2.2.4 The Glenohumeral Joint	14
2.2.4.1 Bony Geometry.....	14
2.2.4.1.1 Humeral Retroversion	16
2.2.4.1.2 Glenoid Orientation.....	18

2.2.4.2	Glenoid Labrum	20
2.2.4.3	Glenohumeral Joint Capsule	21
2.2.4.4	Intraarticular Pressure and Concavity Compression.....	22
2.2.4.5	Rotator Cuff.....	24
2.2.4.6	Prime Movers.....	25
2.2.4.7	Scapulohumeral Rhythm.....	26
2.3	Anatomical and Physiological Adaptations of the Upper Extremity in Overhead Athletes	28
2.3.1	Skeletal Adaptations.....	28
2.3.1.1	Wolff's Law	28
2.3.1.2	Bone Strength Adaptations Associated with Asymmetrical Overhead Activity	29
2.3.1.3	Humeral Retroversion Adaptations Associated with Asymmetrical Overhead Activity.....	35
2.3.1.4	Glenoid Retroversion Adaptations Associated with Asymmetrical Overhead Activity.....	44
2.3.2	Soft Tissue Adaptations.....	46
2.3.2.1	Hypermobility Adaptations Associated with Asymmetrical Overhead Activity	46
2.3.2.2	Hypomobility Adaptations Associated with Asymmetrical Overhead Activity	51
2.3.2.3	Shoulder Internal and External Rotation Strength Adaptations Associated with Asymmetrical Overhead Activity.....	61

Chapter 3	66
Methods.....	66
3.1 Participants.....	66
3.2 Procedures.....	67
3.2.1 Range of Motion.....	67
3.2.2 Strength Measures	69
3.2.3 Humeral Retroversion	73
3.3 Data Reduction.....	75
3.4 Data Analysis	76
3.5 Power Analysis	78
Chapter 4	79
Junior and Collegiate Tennis Players Display Similar Bilateral Asymmetries of Humeral Retroversion.....	79
4.1 Dissertation Primary Focus.....	79
4.2 Results.....	79
4.3 Discussion.....	81
Chapter 5	86
Interpreting Soft Tissue Adaptations of the Shoulder After Accounting for Humeral Retroversion Adaptations in Junior and Collegiate Tennis Players.....	86
5.1 Dissertation Secondary Focus.....	86
5.2 Results.....	87
5.3 Discussion.....	91
Chapter 6	103

Relationships Do Not Exist Between Adaptations in Humeral Retroversion and Rotational Strength Ratios of the Shoulder	103
6.1 Dissertation Tertiary Focus.....	103
6.2 Results.....	103
6.3 Discussion.....	104
Chapter 7	108
Conclusion	108
7.1 Future Directions	112
References	115
Appendix 1	139
Appendix 2	146

LIST OF TABLES

	Page
Table 4.1. Participant demographics.....	80
Table 4.2. Mean, standard deviation, minimum, and maximum values for humeral retroversion and bilateral humeral retroversion difference	80
Table 5.1. Dominant and nondominant measures of range of motion variables by age group	88
Table 5.2. Bilateral difference measures of range of motion variables by age group	88
Table 6.1. External and internal rotational strength data for the dominant shoulder.....	104
Table 7.1. Clinical Scenarios	110

LIST OF FIGURES

	Page
Figure 2.1. The critical shoulder angle.	18
Figure 3.1. Subject set-up used for the collection of passive internal rotation range of motion.....	69
Figure 3.2. Subject set-up used for the collection of passive external rotation range of motion.....	69
Figure 3.3. Subject set-up for collecting isometric internal rotation strength of the shoulder in the 30°-30°-30° position.....	71
Figure 3.4. Subject set-up for collecting isometric external rotation strength of the shoulder in the 30°-30°-30° position.....	72
Figure 3.5. Subject set-up for collecting isometric internal rotation strength of the shoulder in the 90°-0° position.....	72
Figure 3.6. Subject set-up for collecting isometric external rotation strength of the shoulder in the 90°-0° position.....	73
Figure 3.7. Subject set-up for collecting humeral retroversion angle using musculoskeletal ultrasound.	75
Figure 3.8. Plot depicting total sample size estimation as a function of effect size.	78
Figure 4.1. Mean dominant and nondominant humeral retroversion measures by age groups.	81
Figure 5.1. Mean dominant and nondominant internal rotation range of motion by age groups.	89

Figure 5.2. Relationship between humeral retroversion difference ($HRV\Delta$) and internal rotation difference ($IR\Delta$).	89
Figure 5.3. Relationship between humeral retroversion difference ($HRV\Delta$) and external rotation difference ($ER\Delta$).	90
Figure 5.4. Relationship between humeral retroversion difference ($HRV\Delta$) and total arc of motion difference ($TAM\Delta$).	90
Figure 5.5. Relationship between humeral retroversion difference ($HRV\Delta$) and humeral retroversion-corrected internal rotation difference ($HRVcIR\Delta$).	91
Figure 5.6. Relationship between humeral retroversion difference ($HRV\Delta$) and humeral retroversion-corrected external rotation difference ($HRVcER\Delta$).	91

Chapter 1

Introduction

1.1 Background

Some of the fastest movements in sport occur during overhead motions of the upper extremity such as throwing and serving.^{1,2} During these movements, the shoulder complex experiences extremely large forces and torques that are necessary to maintain control and stability. However, these same forces and torques have the ability to exceed the integrity of the involved musculoskeletal structures thereby resulting in injury. Unfortunately, there is an increasing trend in the incidence of shoulder and elbow injuries in overhead athletes.³⁻⁶ The simplest explanation for this trend may be that youth are participating in sports more frequently than in years past. Injury risk in the overhead athlete appears to increase with age,⁷⁻⁹ level, volume and intensity of play,⁹⁻¹⁴ and early sports specialization.^{8,15} A majority of the shoulder injuries sustained by overhead athletes are impingement syndromes and rotator cuff pathologies, which suggest chronic overloading of the tissues.^{7,16,17} Chronic shoulder pain has been linked to sport-specific adaptations of the shoulder that include alterations in the bony and soft tissue structures.^{1,18-24} Overall, these sport-specific adaptations result from repeated and extended exposure to overhead athletic activity, which may ultimately lead to overloading the musculoskeletal structures and subsequent injury.

Over the past few decades, the participation in sporting activities has shifted from a recreational-focused activity towards a more competitive emphasis for developing sport-specific skills in order to achieve high levels of success.^{15,25,26} Furthermore, today's

society has glorified the success of elite athletes through fame and fortune. As such, many athletes aspire to achieve elite levels despite the reality that less than 1% of athletes between the ages of 6 and 17 will actually make it to an elite and/or professional status.²⁵ Nonetheless, many young athletes make the transition to intensive, high-volume training, and early sports specialization in hopes of becoming an elite and/or professional athlete.

It is not well understood what constitutes the right amount of training in order to achieve an elite-level status in regards to athletic performance. Ericsson et al²⁷ reported that musicians must practice 10,000 hours over 10 years in order to achieve expertise in their respective genre. This theory was extrapolated to athletic achievements in the now infamous book *Outliers: The Story of Success*.²⁸ In addition to these inferences, there is the timeworn mantra that “practice makes perfect.” While there is a general consensus that the number of hours played positively correlates with the level of achievement in sports, there is inadequate scientific evidence to support the necessity of high amounts of deliberate practice for the development of elite performance.^{26,29} In contrast, the evidence against high intensity, high volume, and early sports specialization is well established with most all medical and healthcare organizations advocating against these practices.²⁹⁻³³ Despite the overall negative connotation, many parents and coaches continue to encourage and promote this concept to young athletes.

The body has the ability to adapt in response to the stresses it experiences during physical activity. Many of these adaptations lead to overall improvements in health and disease prevention including, but not limited to, improved aerobic fitness,³⁴⁻³⁷ body composition,³⁶⁻³⁸ bone health,^{36,37,39} and decreased cardiovascular risk.^{36,37,40} As such, many look to sports, such as tennis, as a means to engage in physical activity for healthy

lifestyles.^{36,37} As mentioned earlier, overhead athletes are also known to develop sports-specific adaptations of the shoulder complex, which include alterations in shoulder motion and strength.^{19,21-24,41-45} Some clinicians and investigators have speculated that these specific adaptations are necessary to achieve enhanced performance.^{2,45-49} However, others have demonstrated that these same adaptations have the potential to progress,^{47,50-53} alter joint biomechanics,^{2,49,54,55} and have been identified as causative factors in the development of injury.^{2,7,21-24,49}

Several investigations have demonstrated that overhead throwing athletes display significant increases in humeral retroversion (HRV) in the dominant arm when compared to the nondominant side.^{45,56-63} The cause of this bony adaptation is thought to be the result of repeated exposures to throwing during the years of skeletal immaturity that impedes the normal derotational (anteversion) growth of the humerus.^{55,64} To our knowledge, only one study has investigated HRV adaptations in tennis players,⁶⁵ despite the similarities between the overhead throwing and serving motions. Several studies have demonstrated that tennis players experience significant bone strength adaptations,⁶⁶⁻⁶⁸ specifically in response to torsional loads placed upon the humerus in the serving arm.⁶⁸ In addition, Taylor et al,⁶⁴ through the use of biomechanical modeling, presented data that suggested torsional loads experienced during the overhead tennis serve are substantial enough to induce HRV changes. Increased measures of HRV shifts the total arc of motion (TAM) to a more externally rotated position, which is thought to explain the commonly observed range of motion (ROM) asymmetries in these athletes.^{45,46,61,69} This osseous adaptation, resulting in an apparent increase of allowable external rotation (ER), is thought to be advantageous for achieving optimal amounts of ER during the late-

cocking phase of the overhead throw for creating maximum ball velocity, while reducing stress on the soft tissue restraints of the glenohumeral joint.^{45,70} On the other hand, there appears to be a safe zone associated with the amount of HRV adaptation as researchers have demonstrated that inadequate or excessive amounts may be linked with injuries to the shoulder and elbow.^{56,57,61-63}

One of the most common adaptations observed in the overhead athlete is a bilateral disparity in the rotational ROM of the glenohumeral joint when measured in 90° of abduction. Altered rotational ROM measurements have been consistently reported in asymmetrical overhead athletes including baseball players,^{45,53,59,71-80} handball players,⁸¹⁻⁸³ softball players,^{76,84} tennis players,^{42,50,74,85-96} volleyball players,^{47,59,97-100} and water-polo players.¹⁰¹ Interestingly, altered mobility patterns have also been observed in the shoulders of swimmers despite the symmetrical movement patterns that occur during swimming.^{14,47,95,102} The differences observed in the dominant arm of overhead athletes have been described to be normal, excessive (hypermobility), and restrictive (hypomobility) adaptations dependent upon the amount and direction of the altered rotational movement.¹⁸

The disparity in the rotational ROM of the overhead throwing athlete is typically observed whereby the dominant shoulder demonstrates increased ER and decreased IR when compared to the nondominant arm.^{72,73,77,81,85,86,92,103,104} This results in a shift in the TAM towards a more externally rotated position, and has been described as the TAM concept.¹⁰⁴ As noted above, this shift in the rotational arc is most likely attributed to increased HRV in the dominant arm; however, soft tissue adaptations in the capsuloligamentous and musculotendinous restraints of the glenohumeral joint may also

play a role.¹⁰⁵ While the shift can be substantial in magnitude, the majority of overhead throwing athletes maintain a TAM that is equivocal when compared bilaterally.¹⁰⁴ Similarly, tennis players are known to demonstrate increased measures of ER and decreased measures of IR.^{42,50,91-93,95,106} However, tennis players commonly demonstrate deficits in IR that exceed the amount of ER gain,^{42,50,91-93,95} which, according to Burkhart et al,⁴⁹ will lead to abnormal kinematics and function of the shoulder joint. In fact, some researchers have demonstrated that tennis players demonstrate no bilateral differences in ER.^{74,90,107} As a result, tennis players often demonstrate significant deficits in the TAM of approximately 9°,^{42,50,74,88,90,92,93} which is larger than the current recommendation of 5° or less for identifying potential injury risk.^{80,105,108} Considering that side-to-side differences in HRV are unknown in tennis players, the relative contributions of bony and soft tissues in the development of these observed rotational ROM adaptations is unknown.

While a shift in the TAM towards a more externally rotated position is considered a normal adaptation, overhead athletes are known to develop maladaptations in the rotational ROM of the shoulder, which have been linked with increased injury risk.¹⁰⁵ These maladaptations are described as either isolated deficits in IR, ER or TAM, or a combination of a directional deficit with a concomitant TAM deficit.¹⁰⁵ It appears that HRV may play an important role in the development of rotational deficits attributed to soft tissue changes.^{62,109,110} Researchers have demonstrated greater measures of HRV in the dominant arm of baseball pitchers presenting with concomitant deficits in IR and TAM as compared to pitchers without rotational deficits.^{62,110} However, when rotational ROM measures are adjusted for side-to-side differences in HRV, IR deficits are substantially reduced while ER deficits become more pronounced.^{62,109} These

observations support the recent findings of Wilk et al⁷⁹ who prospectively determined that professional baseball pitchers with dominant arm ER deficits (dominant side ER less than 5° greater than the nondominant side), not IR deficits, were 2.2 times more likely to succumb to shoulder injury.

It is suggested that rotational shoulder strength should be evaluated as part of the overall injury risk assessment as overhead athletes commonly display a sports-specific adaptation in the dominant arm ER:IR strength ratio.^{19,43,111,112} This alteration in the ER:IR strength ratio is due to significant increases in IR strength with relatively no changes in ER strength.^{43,88,113,114} It appears that rotational motion deficits, particularly those associated with IR deficits, may have deleterious effects on isometric shoulder abduction¹¹⁵ and eccentric ER strength of the shoulder.¹¹⁶ Considering the association between greater measures of humeral retroversion and the effect on developing greater soft tissue adaptations that result in larger IR and TAM deficits,⁶² it is plausible that increased humeral retroversion may have deleterious effects on ER shoulder strength. However, there is evidence to suggest that increased measures of humeral retroversion may have positive implications on rotational strength of the shoulder.¹¹⁷ Nonetheless, given the limited number of studies future investigations are necessary to gain a better understanding of the effects of humeral retroversion on shoulder strength.

In order to mitigate the risk of injury associated with ROM deficits of the overhead athlete's shoulder, it is suggested that clinicians implement appropriate interventions to prevent and treat mobility deficits.^{19,105} The rationale for correcting motion deficits is centered on improving soft tissue restrictions,¹⁰⁵ particularly as HRV can't be modified in the skeletally mature athlete. Current recommendations used to

determine clinically significant findings of shoulder motion deficits in the overhead athlete are based primarily on data collected on baseball players.¹⁰⁵ These include: dominant arm IR deficit $>18^{\circ}$ - 20° with concomitant TAM deficit $>5^{\circ}$ ^{19,105}; dominant arm TAM deficit $>5^{\circ}$ ^{80,105,108}; and dominant arm ER deficit whereby the dominant arm is less than 5° greater than the nondominant arm.⁷⁹ The generalizability of these recommendations may be limited due to known variabilities in normal shoulder motion characteristics among other overhead athlete, as we have noted in tennis players. In addition, it is important to note that these guidelines do not differentiate between the bony and soft tissue adaptations that may contribute to the observed motion deficits. Thus, it is not possible for clinicians to accurately determine the magnitude or direction of soft tissue restrictions without knowing the bilateral differences in HRV.

1.2 Objective

The overall objective for this dissertation is to develop an understanding of HRV in tennis players and its impact on physical and performance characteristics of the shoulder.

1.3 Overall Hypothesis

To test the hypothesis that overhead activity encountered while playing tennis results in an increase in HRV and alterations in shoulder biomechanics. In order to address the overall hypothesis, the following specific aims were evaluated:

Specific Aim 1: To test the hypothesis that tennis players will demonstrate increased HRV in the dominant arm when compared to the nondominant arm for each group of junior and collegiate athletes.

Specific Aim 2: To test the hypothesis that differences will exist between tennis player age groups when comparing side-to-side differences in HRV.

Specific Aim 3: To test the hypothesis that tennis players will demonstrate bilateral differences in passive IR and ER measured at 90° of abduction, TAM, HRVcIR, and HRVcER for each group of junior and collegiate athletes.

Specific Aim 4: To test the hypothesis that differences will exist between tennis player age groups when comparing bilateral differences for IR, ER, TAM, HRV-corrected IR, and HRV-corrected ER.

Specific Aim 5: To test the hypothesis that relationships will exist between the bilateral difference of HRV and each of the following bilateral differences of IR, ER, TAM, HRV-corrected IR, and HRV-corrected ER in tennis players.

Specific Aim 6: To test the hypothesis that a relationship will exist between HRV and the dominant shoulder ER:IR strength ratio in tennis players.

1.4 Dissertation Organization

The rest of this dissertation is organized in the following manner. Chapter Two presents relative background information in the form of a review of the pertinent literature related to this project. Specifically, Chapter Two presents epidemiological data relative to the participation in the game of tennis, a review of the anatomy and biomechanics of the shoulder complex, and anatomical and physiological adaptations that occur in the upper extremity in overhead athletes. Chapter Three discusses the methods used in the execution of this research project, including subject recruitment, procedures and equipment utilized for data collection, and information regarding data reduction and analysis.

We have categorized the six specific aims of this study into three foci. The primary focus of this study was to determine if tennis players demonstrated bilateral differences in HRV, and to compare the extent of difference across the age-continuum of junior and collegiate tennis players. Specific Aims 1 & 2 were grouped together to make up the primary focus. The secondary focus of this study was to differentiate bony and soft tissue adaptations of the shoulder in order to examine the influence that HRV adaptations had on the interpretation of clinical measures of rotational shoulder motion, and to examine these measures across the age-continuum of junior and collegiate tennis players. Specific Aims 3, 4, & 5 were grouped together to make up the secondary focus. The tertiary focus of the study, composed of Specific Aim 6, was to determine if HRV adaptations have an influence on rotational strength of the shoulder in junior and collegiate tennis players. The results of the statistical analyses along with a discussion of our impression of the results for each of the foci are presented in Chapters Four, Five & Six, respectively. Last, Chapter Seven provides a brief conclusion and future directions for research as they pertain to the results of this study.

Chapter 2

Review of Literature

2.1 Background and Epidemiology

Tennis is one of the more popular sports in the world, and is considered to be the most popular international racket sport. In the United States alone, it is estimated that approximately 18 million people participate in the game of tennis.¹¹⁸ The sport has experienced growth over the past few decades,¹¹⁸ which may be attributed to the relatively low cost of participation, and is considered an activity that can be played throughout the lifetime.^{36,119} In addition, there are numerous and well-documented health benefits associated with playing tennis.³⁷

Despite the overall health benefits, tennis players are not immune to injury. The game of tennis is unique in that there are no time constraints in determining when a match ends. As such, a match can last for several hours resulting in the players experiencing hundreds of abrupt, explosive, or repetitious bouts of physical activity.¹²⁰ The physical demands imposed upon the body result in a variety of acute, subacute, and chronic injuries in practically all regions of the body. Although there is a substantial volume of epidemiological data, the incidence and prevalence across all participants is difficult to determine as methodologies and populations studied have varied substantially across studies. Incidence rates for tennis-related injury are reported to range anywhere from 0.04 to 6.05 per 1000 playing hours.^{9,121-123} The prevalence of injury among youth and adolescent players is reported to range from 18.4 to 30 injuries per 100 players.¹²²⁻¹²⁵ In older recreational players, injury prevalence appears to be greater. Jayanthi et al¹²⁶

reported a prevalence of 52.9 injuries per 100 players in a group of recreational tennis players with an average age of 46.9 years.

While there is variability among the literature, there appears to be a consistent pattern with regard to the location and type of injury sustained by tennis players. Injuries mostly occur in the lower extremity (31%–67%), followed by the upper extremity (20%–49%), and trunk (3%–21%).^{9,122,124,126-128} In addition, acute injuries occur more commonly in the lower extremity, whereas chronic/overuse injuries are more commonly in the upper extremity.^{9,16,121,127} When examining injuries based on individual body parts, many have reported the shoulder as having the highest incidence,^{122,123,129} while others have reported it to be among the top two or three.^{7,9} Colberg et al¹²³ conducted a prospective epidemiological study on the incidence and prevalence of musculoskeletal injuries in collegiate tennis players, and observed the shoulder to have the highest incidence of acute injuries (0.4 injuries/1000 playing hours). In a seven-year review of shoulder injuries in collegiate overhead athletes, Laudner et al¹⁷ found that sub-acromial impingement syndrome and rotator cuff tendinopathies were reported in tennis players at significantly higher rates than any other type of injury to the shoulder.

2.2 The Shoulder Complex

The shoulder complex is comprised of the clavicle, humerus, and scapula. When linked together with the axial skeleton, this complex is interconnected via three diarthrodial joints and one physiological interface. The shoulder complex allows for the greatest ROM of any joint or joint complex in the human body. This unique characteristic is essential in fulfilling its primary purpose of manipulating the hand in space to execute a variety of tasks.¹³⁰ These tasks range anywhere from basic activities of daily living to

more complex movements like those performed during occupational and athletic activities. While there are numerous advantages for the shoulder complex having extensive mobility, stability is compromised as an inherent consequence of allowing such a large ROM. In order to maintain stability, the shoulder complex relies heavily upon the interdependent relationship among its static (e.g., joint capsule and ligaments) and dynamic (e.g., muscles) stabilizers. Consequently, large forces and torques are generated about the anatomical structures of the shoulder complex. Overtime, these structures may experience excessive loads that may ultimately result in various forms of adaptation and/or injury. Therefore, a thorough understanding of the shoulder complex anatomy is warranted, and a brief review is presented here with emphasis placed on the structures of the glenohumeral joint.

2.2.1 The Sternoclavicular Joint

The sternoclavicular joint is the only direct bony articulation between the upper extremity and the axial skeleton. This saddle joint, connecting the medial end of the clavicle to the manubrium, is very stable despite poor congruency between the two bones.¹³¹ Stability of the joint is maintained passively with the interclavicular ligament, the sternoclavicular ligaments, and the costoclavicular ligaments along with dynamic support from the sternocleidomastoid and subclavius muscles.^{131,132} In addition, the joint is divided into two separate joint spaces by the presence of an intra-articular fibrocartilaginous disk that contributes to stability by limiting excessive displacement and improving congruency between the clavicle and manubrium.¹³¹⁻¹³³ While the joint is classified as a saddle joint, the function is described relative to a spheroidal joint, which is vital for the vast amount of mobility available to the shoulder complex.¹³¹

2.2.2 The Acromioclavicular Joint

The acromioclavicular joint links the distal end of the clavicle to the acromion process of the scapula. The joint is classified as a plane joint; however, the mobility of the scapula at this joint is described as having three degrees of rotational freedom.^{134,135} An articular disc is commonly present in the joint, but is known to vary in shape and size. The articular disc undergoes rapid degenerative changes beginning in the second decade of life, and the absence of the disc is suggested to play a role in early development of osteoarthritis.¹³¹ Passive joint stability is provided by the acromioclavicular and coracoclavicular ligaments. Furthermore, the fascial fibers of the deltoid and trapezius blend with the superior fibers of the acromioclavicular ligament adding additional support to the stability of the joint.^{131,132,136} The acromioclavicular ligament serves as the primary restraint against posterior displacement and posterior axial rotation of the clavicle.^{131,132,136,137} The coracoclavicular ligaments are often reported to serve as the primary suspensory ligaments from which the scapula is suspended.^{131,132} In regards to their role in acromioclavicular joint stability, these ligaments serve as the primary restraints against superior displacement of the clavicle.^{131,132,136}

2.2.3 The Scapulothoracic Interface

The scapulothoracic interface is where the anterior aspect of the scapula approximates with the posterior thorax. This interface is not classified as a true joint as there are specific characteristics that are lacking (i.e., bony articulation, joint capsule, and capsular ligaments). The scapula is primarily stabilized to the thorax by six muscles that originate from the axial skeleton: the levator scapulae, pectoralis minor, rhomboid major, rhomboid minor, serratus anterior, and trapezius. In addition to the stability created by

muscular force, some stabilization may be provided by the vacuum-like pressure effects created within the scapulothoracic and subscapular bursae. These bursae are located between the posterior thoracic wall and the serratus anterior (i.e., scapulothoracic space), and between the serratus anterior and the subscapularis (i.e., subscapular space).¹³⁸ These spaces allow for the gliding motions that occur at the scapulothoracic interface. Last, motion of the scapula on the thoracic wall is limited by the constraints of the sternoclavicular and acromioclavicular joints.

2.2.4 The Glenohumeral Joint

The glenohumeral joint is the most mobile joint in the human body. This joint is classified as a spheroidal joint having three degrees of freedom that include flexion/extension, abduction/adduction, and IR/ER. While the joint allows for vast mobility, it is inherently unstable by design. The humeral head is stabilized against the glenoid during various movements by numerous static and dynamic mechanisms. The static mechanisms that assist in maintaining stability include the bony geometry, glenoid labrum, capsular and ligamentous structures, and negative intraarticular pressure and concavity compression. The dynamic mechanisms include the rotator cuff, primary movers, and scapulohumeral rhythm. However, it is important to note that these mechanisms act in concert whereby no one structure stabilizes the joint alone throughout the ROM.

2.2.4.1 Bony Geometry

Originally, it was speculated that stability of the glenohumeral joint was jeopardized due to a lack of congruency or shallowness between the two articulating surfaces. This was based on the thought that the glenoid was relatively flat and much

smaller than the larger and more spherically shaped humeral head.¹³⁹ However, it is not that the articulating surfaces of the humeral head and glenoid lack gross congruency. Studies have demonstrated that the radii of the mating articulating surfaces are within 1-3 mm,¹³⁹⁻¹⁴¹ and have differences of less than 1% in sphericity.¹⁴⁰ Nonetheless, the small differences that exist between the surfaces results in varying amounts of contact throughout the ROM. The contact area has been demonstrated to increase as the joint is abducted with the largest amount of contact area occurring in the mid-range of elevation.^{142,143} In addition, an improvement in joint congruency and decreased joint contact pressures have been observed as the joint is abducted.¹⁴² Warner et al¹⁴² expressed that this “made sense teleologically” as joint stability is known to be at its greatest risk in positions of abduction, and the greatest loads applied to the glenohumeral joint during overhead throwing occur in an abducted position.

Rather than joint congruency, glenohumeral joint stability is compromised mostly due to the disproportionate size of the larger hemispherical humeral head to the smaller, ellipsoidal-shaped glenoid. The proportionality between the two articulation surfaces is commonly described using the analogy of a golf ball sitting atop of a golf ball tee. Soslowsky et al¹⁴⁰ revealed that the humeral head articular surface area is an average of 3.12 to 2.9 times larger than the glenoid for males and females, respectively. This disproportionality results in only 30% of the humeral head being in contact with the glenoid at any given position of glenohumeral motion.^{140,143}

Other bony parameters of the humerus and scapula have been investigated for their influence on glenohumeral joint stability. Specifically, HRV and glenoid orientation

measures are known to affect glenohumeral joint stability,¹⁴⁴⁻¹⁴⁹ injury risk,^{56,57,61,63,150-157} and surgical outcomes.¹⁵⁸⁻¹⁶¹

2.2.4.1.1 Humeral Retroversion

The phenomenon of twisted growth about the long axis of the humerus has been observed by anatomists since the middle of the 18th century.¹⁶² In the field of anthropology, the term humeral torsion is used to describe the orientation of the humeral head relative to the distal mediolateral axis of the humerus.¹⁶³ This reference measure is based upon the primitive orientation of the humeral head that is described as being directed posteriorly, and measures of a more medially facing humeral head are indicated by larger degrees of humeral torsion.^{162,163} However, in the clinical and sports medicine fields, the term HRV is used whereby the default orientation of the humeral head is directed medially.¹⁶³ Therefore, measures of a more posteriorly facing humeral head are indicated by an increasing degree of HRV. It is important to note that these measures are relatively the inverse of each other and can be viewed as complementary (or supplementary depending on the location of the 0° reference position) angles of measure. In other words, a smaller humeral torsion measure corresponds to a larger HRV measure, and vice versa. In order to improve clarity, the term HRV will be used throughout this treatise.

The amount of HRV that one develops appears to be influenced by a combination of evolutionary, developmental, and functional factors.¹⁶²⁻¹⁶⁷ The evolutionary shift of the scapular position from a more lateral to posterior orientation on the thorax appears to have necessitated a shift in the orientation of the humeral head. As the scapula was shifted posteriorly, the glenoid fossa was consequently oriented in a more lateral

direction. Therefore, the orientation of the humeral head shifted from a more posterior position to a more medially facing position to maintain its articulation with the scapula. According to Larsen et al,¹⁶³ the adaptation in HRV was necessary to maintain functional motion of the elbow (i.e., flexion/extension) in the sagittal plane. In regard to developmental and functional factors, Krahl and colleagues^{162,166,167} acknowledged the influences that muscular forces and function have on the development of HRV. Their findings have since been substantiated in the sports medicine literature whereby overhead throwing athletes have been shown to demonstrate significant bilateral asymmetries in HRV measures.^{45,46,51,58,60,76,99,168-172} The influence that functional activities have on HRV adaptations will be discussed in detail later in this chapter (see Section 2.3.1.3).

The average measure of HRV is approximately 30° ,¹⁷³⁻¹⁷⁵ and has considerable within- and between-subject variability by as much as 38° ⁶⁰ and 90° ,^{163,173} respectively. Being that HRV is influenced by the evolutionary positioning of the scapula on the thorax, it is logical to assume a relationship exists between the amount of HRV and the planar orientation of the scapula. Therefore, it should be of no surprise that the scapula normally rests on the posterior thorax in a plane that is angled approximately 30° to 45° anterior to the coronal plane of the body.¹⁷⁶ In view of that, several reports have embraced the influential role that the scapula and its alignment with the humeral head has in maintaining stability of the glenohumeral joint.¹⁷⁷⁻¹⁸¹ Interestingly, it does appear that HRV has an impact on glenohumeral joint stability. Decreased measures of HRV have been demonstrated in individuals that have sustained first-time and recurrent anterior dislocations of the glenohumeral joint.^{155,157,182} Furthermore, other studies have reported success using rotational osteotomies to restore normal measures of HRV (e.g., average

postoperative retroversion of 32° ¹⁶¹) in patients who experienced recurrent dislocations and had small HRV angles (e.g., average preoperative retroversion of 12° ¹⁶¹).^{159,161}

2.2.4.1.2 Glenoid Orientation

The orientation of the glenoid describes its geometrical relationship with respect to the body of the scapula. There are numerous mechanisms by which the orientation of the glenoid can be captured and defined. The following measures are of particular interest to this dissertation: glenoid inclination, glenoid version, and the critical shoulder angle. Glenoid inclination and version are two of the most common measures used to quantify the orientation of the glenoid. Glenoid inclination describes the amount of upward or downward tilt of the glenoid as measured in the coronal plane of the scapula.^{183,184} Glenoid version represents the amount of anterior or posterior tilt measured in the transverse plane of the scapula.^{183,185} An additional metric, the critical shoulder angle (Figure 2.1), was recently defined as a radiological parameter that quantifies the angle created between the inclination plane of the glenoid and the amount of lateral extension of the acromion (i.e., acromion index¹⁸⁶).¹⁵²

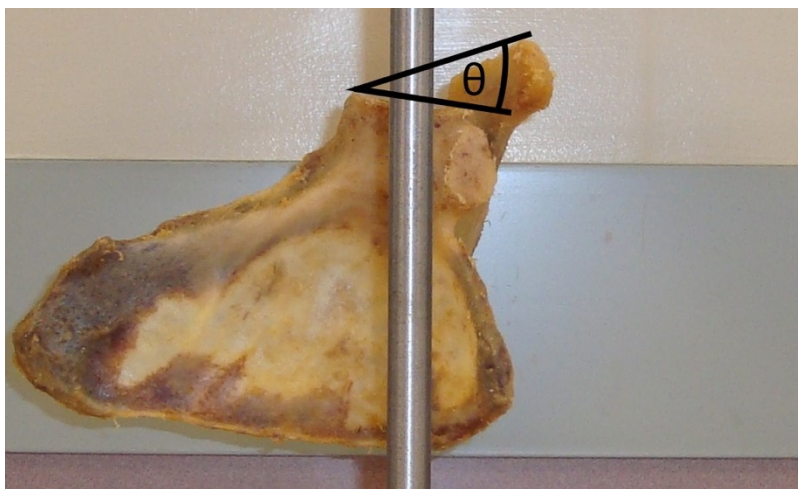


Figure 2.1. The critical shoulder angle. The angle (θ) is formed between one vector that connects the most superior aspect of the glenoid with the most inferior aspect, and the second vector extending from the most inferior aspect of the glenoid to the most lateral projection of the acromion process. The coracoid process has been removed to improve visualization.

On average, the glenoid faces slightly superiorly with approximately 4-5° of inclination,^{150,183} and slightly posteriorly of approximately 1° of retroversion.^{175,183} While not to the same degree as HRV, both glenoid inclination and version demonstrate considerable variability with measures varying by as much as 23°¹⁸³ and 22°,¹⁷⁵ respectively. The critical shoulder angle has been reported to have an average measure of approximately 33°,^{152,187} and has been found to vary by as much as 25° in patient populations.¹⁵² Similar to HRV, it appears that the orientation of the glenoid may be affected by human growth and developmental factors,^{183,188,189} and functional activity.^{45,151,190,191} The influence that functional activities have on glenoid orientation adaptations will be discussed in detail later in this chapter (see Section 2.3.1.4).

According to the literature, the orientation of the glenoid appears to play a pivotal role in effecting the health and stability of the glenohumeral joint. Numerous investigations have demonstrated that altered measures of glenoid inclination and/or retroversion are found in patients or cadaveric specimens with rotator cuff tears.^{150,152-154,156,184,192-197} It has been theorized that greater measures of glenoid inclination promote superior translation of the humeral head, which may lead to rotator cuff disease via compression of the tendon against the undersurface of the acromion.^{194,198} However, this theory has been challenged by in-vivo studies that found no evidence to suggest glenoid inclination is responsible for superior translation of the humeral head during arm elevation tasks.^{192,195} More recently, studies investigating the effects of glenoid inclination-dependent changes of the critical shoulder angle revealed alterations in the joint reaction forces of the glenohumeral joint.¹⁴⁴⁻¹⁴⁷ Researchers have demonstrated that increasing the inclination angle of the glenoid requires greater activity of the rotator cuff

to maintain stability of the joint.¹⁴⁶ Consequently, greater loads are then placed on the supraspinatus, which may overload the tendon resulting in degenerative changes overtime.¹⁴⁵

Glenoid inclination and version have also been linked to acute and recurrent luxations of the glenohumeral joint further suggesting their role in maintaining joint stability. The evidence suggests that individuals with posterior instability have a greater incidence of larger measures of glenoid retroversion.^{148,199} Others have demonstrated that individuals who sustained anterior glenohumeral joint dislocations have a more anterior/inferior facing glenoid as opposed to healthy shoulders that have a more posterior/superior oriented glenoid.¹⁴⁹

2.2.4.2 Glenoid Labrum

The glenoid labrum is a triangular-shaped fibrocartilaginous structure that is attached to the peripheral rim of the bony glenoid fossa. According to Cooper et al,²⁰⁰ the anterior to anterosuperior region of the labrum is loosely connected to the glenoid rim and is considered to be comparable to the meniscus of the knee. In addition, the labrum blends superiorly with the tendon of the long head of the biceps brachii as it anchors into the supraglenoid tubercle. Inferiorly, the labrum is a more fibrous, rigid extension of the glenoid rim.²⁰⁰

Accounting for approximately 50% of the depth of the glenoid socket, the labrum acts as a chock block thereby limiting the amount of translational movement of the humeral head on the glenoid.¹³⁹ Biomechanical studies have demonstrated that excising the labrum reduces the resistance against translation of humerus on the glenoid by 20-65%.^{201,202} The labrum further contributes to the stability of the joint by improving joint

congruency, creating a suction effect, and adding to the overall articulating surface area of the glenoid.²⁰³ The labrum also functions as an intermediary, connecting the capsuloligamentous structures of the glenohumeral joint to the bony glenoid.

2.2.4.3 Glenohumeral Joint Capsule

The articulating surfaces of the glenohumeral joint are enclosed by a thin, cylindrical sleeve of fibrous connective tissue.²⁰⁴ Commonly referred to as the articular capsule, it attaches proximally to the scapula around the neck of the glenoid with some fibers blending into the glenoid labrum.^{18,132,204} The capsule attaches to the humerus about the anatomical neck just distal to the peripheral rim of the articular surface of the humeral head.²⁰⁴

In order to allow for a large ROM, the capsule is loose and redundant. This is evident when considering the volume within the capsule is approximately twice the size of the humeral head,¹³² and the amount of available joint laxity allows for approximately 2-2.5 cm of joint distraction. Therefore, the amount of stabilization provided by the capsule in the mid-ranges of glenohumeral motion is minimal. Only at the end-ranges of motions does the capsule begin to increase its contribution to joint stabilization and/or restricting joint motion.

The capsule is reinforced by thickened bands of collagenous tissue in the anterior and inferior regions. The bands are referred to collectively as the glenohumeral ligaments. In the anterosuperior region, the capsule is reinforced by the superior glenohumeral ligament. In addition, the extracapsular coracohumeral ligament originating from the coracoid process, blends with the fibers of the superior glenohumeral ligament at their insertions into the humerus. These structures limit inferior displacement of the

humerus with the arm adducted, while limiting ER when the humerus is adducted.²⁰⁵ The middle glenohumeral ligament provides anterosuperior stability and restrains ER between 0° and 90° abduction.²⁰⁵ The hammock-like inferior glenohumeral ligament complex is comprised of an anterior band, a posterior band, and an axillary pouch interposed between the two bands. This complex is the primary capsuloligamentous stabilizer of the abducted humerus. O'Brien et al²⁰⁶ elaborated on the distinct functional importance of each band and the axillary pouch. At 90° of abduction, it was observed that the anterior band becomes more prominent with increasing ER. Along with the inferior pouch, these structures “cradled” the humeral head anteriorly acting as the primary static restraint to anterior motion. A reciprocal observation was made for the posterior band and axillary pouch during IR in the same abducted position.²⁰⁶

2.2.4.4 Intraarticular Pressure and Concavity Compression

The intraarticular pressure within the joint cavity as well as the amount of force that compresses the humeral head into the glenoid cavity contribute to the stability of the glenohumeral joint. The intraarticular surface of the glenohumeral joint capsule is lined with synovial tissue. A small amount of synovium is produced to provide nutrients and lubrication to the articular surfaces of the glenoid and humerus. Concurrently, the interaction between the synovium and the tissues confined within the sealed joint cavity create a vacuum-like effect, which contributes to the stabilization of the joint by sucking the humerus into the glenoid.²⁰⁷ The negative pressure, which has been reported to average between -32 mmHg²⁰⁸ and -67.8 mmHg,²⁰⁹ limits the amount of distraction and translation of the humeral head on the glenoid. Habermeyer et al²⁰⁸ demonstrated that negative intraarticular pressure exerts a stabilizing force ranging from 68 N to 225 N for

traction loads applied to the humerus that ranged from 0 N to 300 N. The researchers acknowledge these forces are likely lower in-vivo as other factors (e.g., friction) would reduce the traction forces applied to the joint. Nonetheless, several cadaveric studies have demonstrated the effect that intraarticular pressure has on stabilizing the shoulder by comparing the amount of translational motion at the joint before and after venting the joint capsule.²⁰⁸⁻²¹² Alexander et al²¹¹ reported that translations in all directions increased by as much as 50.8% when testing the joint in 30° of abduction. Additionally, Habermeyer et al²⁰⁸ demonstrated that the labrum acts as a gasket thereby sealing the humeral head to the glenoid. Any disruptive lesions to the labrum eliminated the sealing mechanism thereby resulting in decreased stability.²⁰⁸

Concavity compression describes the stabilization effect that results from compressing a convex surface onto another concave surface. This concavity compression force limits translational movement of the convex surface (i.e., humeral head) on the concave surface (i.e., glenoid fossa). The rotator cuff, long head of the biceps brachii, deltoid, and potentially all other muscles that cross the glenohumeral joint (depending on the position of the humerus in respect to the glenoid) have the ability to increase stability by compressing the humeral head into the glenoid fossa. Likewise, these muscles have the ability to create instability. Research has demonstrated that decreasing the estimated physiological rotator cuff force from 150% to 50% results in a significant increase in superior translation of the humeral head.²¹² Additionally, Alexander et al²¹¹ demonstrated that loading the long head biceps tendon with 20 N improves stability of the joint by decreasing anterior translational movements of the humeral head by 42.6% and inferior translation by 73.3%. These studies demonstrate the importance of a synergistic working

relationship amongst the musculature of the shoulder, particularly when movement occurs in the mid-ranges of motion during which the capsuloligamentous structures are lax.²¹³

2.2.4.5 Rotator Cuff

The rotator cuff is comprised of the supraspinatus, infraspinatus, teres minor, and subscapularis, which all originate from the scapular body and insert onto the greater and lesser tubercles of the humerus. The tendons of the rotator cuff form a glove-like structure surrounding the humeral head with fibers of the tendons blending into the glenohumeral joint capsule providing reinforcement. Individually, each rotator cuff muscle has its own independent action and role. The supraspinatus creates abduction of the humerus and reinforces the joint against superior forces.^{132,207,214} The infraspinatus and teres minor externally rotate the humerus and protect against posterior forces.^{132,207,214} The subscapularis acts to internally rotate the humerus and resists anterior forces.^{132,207,214} However, when viewed collectively these muscles function dynamically to stabilize and “steer” the humeral head during active movements of the shoulder.^{132,207,214}

Dynamically compressing the humeral head into the glenoid during active arm movements contributes to the concavity compression stabilization effect. Studies have demonstrated with electromyography that the rotator cuff is active during arm elevation tasks.^{215,216} The activity of the rotator cuff provides stabilization to the joint by preventing the humeral head from translating superiorly due to the large shear forces created by the deltoid, particularly during the early ranges of elevation.²¹⁷ In support, numerous cadaveric studies have demonstrated that the rotator cuff is effective at preventing

translational movements of the humeral head when simulated shear forces are applied to the joint.²¹⁸⁻²²² Interestingly, it appears that this force couple between the deltoid and the rotator cuff can be maintained even when the supraspinatus has been compromised.^{214,223-225} This suggests that the transverse force couple (i.e., subscapularis, infraspinatus, and teres minor) is sufficient in compressing the humeral head against the glenoid thereby creating a stable fulcrum for humeral elevation.

2.2.4.6 Prime Movers

Eleven muscles cross the glenohumeral joint and contribute to movement of the shoulder complex, which include: the rotator cuff, teres major, deltoid, pectoralis major, latissimus dorsi, triceps brachii, biceps brachii, and coracobrachialis. These muscles are typically grouped together based on their role in creating gross motion of the shoulder complex in the three cardinal planes. However, it is rare for motions of the shoulder complex to occur strictly in the planes during activities of daily living, occupational tasks, or athletic activity. Therefore, the role that each of these muscles play is dependent on the movement of the shoulder complex and the muscle's line of pull relative to the axis of rotation at the joints involved. Most often the rotator cuff is not included in a listing of prime movers as they are traditionally viewed as stabilizing muscles. However, studies have demonstrated that these muscles markedly contribute to gross movements of the shoulder complex. Specifically, the supraspinatus has been demonstrated to have a larger moment arm than the deltoid through the first 50° of abduction,²²⁶ and contributes approximately 50% of the maximum isokinetic abduction torque.²²⁷ Additionally, the infraspinatus and teres minor have been demonstrated to be the primary external rotators of the glenohumeral joint.²²⁸⁻²³¹ Last, while not considered prime movers of the shoulder,

the scapular stabilizing muscles of the scapulothoracic interface play an integral role in shoulder complex motion (see also Section 2.2.3).

2.2.4.7 Scapulohumeral Rhythm

The mechanical interactions between the scapula and humerus play an integral role in stabilizing and facilitating optimal function of the shoulder and upper extremity.¹⁷⁷ The term scapulohumeral rhythm has been coined to describe the synchronous movements that occur between the scapula and humerus during arm elevation. Inman et al²¹⁷ were the first to quantify the corresponding movement between these bones during normal humeral elevation. They proposed an overall 2:1 ratio indicating that for every 2° of glenohumeral elevation there is a corresponding 1° of scapular upward rotation. Since, other investigations have demonstrated a large degree of variability in scapulohumeral rhythm with ratios ranging from less than 1:1 to 4.2:1.²³²⁻²³⁶ Nonetheless, there does appear to be a coordinated effort between the scapula and humerus in maintaining stability of the shoulder complex.

The role of the scapula has received a lot of attention for its role in maintaining scapulohumeral rhythm and normal shoulder function.^{21,22,177,178,180,181,237,238} It is suggested that the primary role of the scapula is to serve as a stable base of support for the glenohumeral joint.^{177,238} In doing so, the scapular stabilizers must manipulate the scapula on the thoracic wall to maintain congruency between the glenoid and humeral head during upper extremity activities. In order to optimize the concavity compression effect, the angulation between glenoid and humeral head must fall within a “safe zone,” whereby the humerus must be positioned within 30° anterior or posterior of the plane of the scapular body.²³⁹

In addition to manipulating the scapula, it is theorized that the scapular stabilizers must dynamically secure the scapula against the thoracic wall in order to execute the transference of forces via the kinetic chain.¹⁷⁷ Currently, scapular stability is indicated by the presence of a “normal” movement pattern of the scapula during humeral elevation tasks.^{237,238,240} In healthy populations the scapula has demonstrated a common pattern of upward rotation, posterior tilt, and high variability of IR/ER during elevation tasks in the frontal, sagittal, and scapular planes.^{135,241} It is commonly accepted that the presence of scapular instability, indicating muscular imbalances or weakness of the scapular stabilizers,^{181,238} is manifested in the form of scapular dyskinesis. Scapular dyskinesis is used to describe aberrant movement patterns of the scapula as demonstrated by scapular winging or dysrhythmia when an individual performs the dynamic scapular dyskinesis test.²⁴²⁻²⁴⁵ It is widely accepted that individuals without scapular stability are predisposed to a number of shoulder pathologies.²³⁸ Further, scapular dyskinesis has been linked to several shoulder pathologies including, but not limited to, impingement, rotator cuff tears, labral pathology, acromioclavicular separations, and multidirectional instability of the glenohumeral joint.²³⁸ However, there does not appear to be a substantial link between scapular kinematics in individuals with and without subacromial impingement.^{246,247} Moreover, a growing number of studies have demonstrated that the presence of scapular dyskinesis is more common than not in healthy populations.^{97,243,248-251} Therefore, the presence of scapular dyskinesis as an indicator of scapular instability is confounded, and the concept of what constitutes “normal” scapular stability has recently come under scrutiny.²⁵²

2.3 Anatomical and Physiological Adaptations of the Upper Extremity in Overhead Athletes

The functional demands of sports like baseball, softball, swimming, tennis, and volleyball require a delicate balance of mobility and stability of the overhead athlete's shoulder.^{18,45} This delicate balance between mobility and stability is maintained via a concerted effort between dynamic muscular activity and passive restraint of the bony, capsular, and ligamentous tissues.^{24,45} These tissues are repeatedly exposed to extremely large forces and torques that are generated throughout the extremes of shoulder motion.^{1,2,49,55,176,253-255} Overtime, these athletes are thought to undergo various anatomical and physiological adaptations that manifest in various forms of altered shoulder mobility and muscular performance.^{18,21,24,49} In addition, researchers have debated whether these adaptations compromise the stability of the shoulder joint thereby increasing the risk of injury.^{18,21,24,49} This section of the literature review will address the various skeletal and soft tissue adaptations that have been observed in overhead athletes and the implications associated with injury risk.

2.3.1 Skeletal Adaptations

2.3.1.1 Wolff's Law

Bone is a dynamic tissue that is formed and remodeled throughout life in response to the mechanical loads under which it is placed. This adaptability was popularized by the early works of Julius Wolff (1836-1932) who theorized that a bone's gross shape and its adaptations are the result of the tissue's response to mechanical stimuli.²⁵⁶ Wolff proposed that when bone is subjected to larger loads, the tissue will respond by remodeling in a manner by which the bony structure will be able to better withstand the

incurred forces.²⁵⁷ Likewise, when bone is loaded less, the tissue will undergo a catabolic response.²⁵⁷ In other words, bone will remodel by a means to be able to withstand only those loads to which it is subjected. Unfortunately, the entirety of Wolff's writings has not withstood the test of time as new evidence has been discovered.²⁵⁶ Nonetheless, the term "Wolff's law" has become more of a catch-all term used today to describe the response of bony tissue to mechanical stimulation.²⁵⁶

Today, it is now understood that mechanotransduction plays a crucial role in the maintenance and remodeling of bony tissue, which is a process through which mechanical loads are converted to biochemical signals and cellular signaling.²⁵⁸ Mechanotransduction involves a four-step process of mechanocoupling, biochemical coupling, signal transmission, and the effector cell response.²⁵⁸ In mature bone, a load applied to bone results in hydrostatic pressure changes and affects interstitial fluid flow. These changes are thought to be the primary stimuli to affect the activity of the osteocytes. Ultimately, osteocytes and their progenitors appear to be strain-sensitive in that they have the ability to transduce mechanical signals induced by mechanical loads into cues that results in the remodeling of bony tissue.^{259,260} The duration, magnitude, and rate of mechanical loading all appear to have an influential impact on the overall structural composition of bone.²⁵⁸ In addition, there is evidence to suggest that cyclic loading may be one of the most influential stimuli to the bone remodeling process.^{258,259}

2.3.1.2 Bone Strength Adaptations Associated with Asymmetrical

Overhead Activity

For over a century, investigators have realized that bone has the ability to respond to loads imparted upon it through a process of adaptation. However, the quantification of

these adaptations to exercise were not noted until the 1960s and 1970s. While studies examining bone strength characteristics (e.g., bone mineral content, cortical thickness, moments of inertia, etc.) in sedentary versus active populations may provide insight into bone's adaptability, asymmetrical overhead athletes provide a unique perspective that allows for control of various factors (e.g., nutritional, genetic, environmental, etc.) that may contribute to differences among groups. Jones et al⁶⁶ were the first to report notable asymmetrical differences in humeral cortical thickness measures in male and female professional tennis players. Cortical thickness in the dominant arm of male players was found to be 34.9% greater than the nondominant, and the dominant arm in females was 28.4% greater than the nondominant.⁶⁶ Since this landmark study, numerous investigations have confirmed the significant side-to-side differences in bony hypertrophy due to asymmetrical exercise in the upper extremities of overhead athletes.^{67,68,261-283} These bony asymmetries are reported to occur throughout the entire length of the respective bones studied.⁶⁷ While substantial bony adaptations in racket-sport athletes (e.g., tennis and squash) appear to occur in humerus, ulna, radius and metacarpals,^{274,276,279} it appears that bony adaptations in overhead throwers are limited to the humerus.²⁸³

It was earlier posited that passive loading through weight bearing was one of the major contributors of mechanical loading to bony tissue, thus leading to bony adaptations. However, this theory does not explain the substantial side-to-side bony asymmetries found in the arms of overhead athletes as the upper extremities are non-weight bearing. Within the past two decades, evidence has suggested that muscular forces are likely the major contributors of the applied strain to bone regardless of weight-

bearing status, which subsequently leads to adaptations in bone strength.²⁸⁴ While some studies have reported strong relationships between muscle size/strength and bone strength,^{269,275,285,286} others have revealed weaker relationships.^{265,267} Ireland et al²⁷⁵ reported strong relationships ($r = .73 - .86$) between muscle size and cortical bone cross-sectional area in both the dominant and nondominant arms of elite youth tennis players. In contrast, Daly et al²⁶⁵ reported fair relationships between side-to-side differences in muscle area and side-to-side differences in bony geometry measures (bone mineral content, cortical area, and moment of inertia) of adolescent female tennis players, which only accounted for 11.8% to 15.9% of the variance of the differences in bony geometry measures. When evaluating these studies, it appears that factors in addition to muscular forces likely contribute to the development of bone mass and shape as seen in the overhead athlete.^{265,267,275}

The adaptations in bone strength that occur in overhead athletes are suggested to have a greater response to torsional forces rather than bending and compressive forces.^{67,68,261,262,274,275,278,283} As noted earlier, baseball players demonstrate substantial humeral adaptations in bone strength, yet these same adaptations do not appear in the radius and ulna as found in racket sport athletes. Warden et al²⁸³ revealed significant side-to-side differences in the humerus of baseball players when compared to a group of non-throwers. The baseball players demonstrated 23.7% greater difference in bone mineral content, 23.8% and 21% greater differences in cortical area and thickness, and 30.2% greater difference in the polar moment of inertia.²⁸³ In contrast, no significant differences were found in the radius and ulna.²⁸³ Similarly, Bogenschutz et al²⁶² revealed significant side-to-side differences in the humerus of softball players. The softball players

demonstrated 14.7% greater difference in bone mineral content, 15.9% and 18.1% greater differences in cortical area and thickness, and 18.2% greater difference in the polar moment of inertia.²⁶² Interestingly, significant differences were revealed in bone strength measures between fast-pitch pitchers and position players.²⁶² Position players exhibited twice as much adaptation than pitchers indicating that throwing mechanics influenced the magnitude of adaptations in bone strength.²⁶² The differences that contributed to the disparity are most likely due to differences in the throwing mechanics between the windmill fast-pitch and overhead throw, which ultimately affected the torsional stresses that were applied to the humerus.²⁶² The windmill fast-pitch can be described as a circumduction movement of the shoulder in a plane that is nearly parallel to the frontal plane. During the execution of the throw the elbow is maintained near a fully extended position creating a relatively minimal amount of torsional stress to the humerus.²⁸⁷ In comparison, the overhead throw occurs with the humerus abducted and the elbow flexed, while the rotational movement occurs about the long axis of the humerus. The overhead throw is well known to create substantial torsional stress about the humerus,^{2,55} which may explain the differences revealed between softball pitchers and overhead throwers.

While the demonstrated effects of torsional forces appear quite clearly in overhead throwers, the cause of bone strength adaptations in the dominant arms of racket sport athletes may not appear as well-defined. Traditionally, the bony adaptations observed in tennis players were speculated to be the result of the impacts that occurs between the racket and ball during tennis play.^{66,279} However, recent studies have provided insight that adds additional support to the theory that torsional forces have a major effect on bone strength adaptations found in racket sport athletes.^{64,68} Ireland et al⁶⁸

presented a unique case study of a tennis player that used his dominant arm for service strokes, and ground strokes with the nondominant arm. The service arm humerus was found to have 22% to 27% greater measures of bone mass, total and cortical cross-sectional area when compared bilaterally. The most pronounced difference was the 47% greater difference in polar moment of inertia of the serving arm, which represents a measure of torsional stiffness. Asymmetries of the ulna were in favor of the ground strokes arm; however, the asymmetries of the radius were reported to be comparable to what would be observed in side-to-side asymmetries of non-tennis players. The observations of the case were also compared to a 12-subject control group of traditional unilateral tennis players. The comparisons between the case and control group revealed similar humeral side-to-side asymmetries in the serving arm, yet slightly less pronounced in the case subject. The ulnar asymmetries of the ground strokes arm were similar, yet less pronounced than the control group; however, the asymmetries of the radius were notably smaller than the control group. Thus, the investigators suggested these findings provide substantial evidence, with consideration of the inherent limitations of a case study, supporting the theory that bone strength adaptations in tennis players are mostly influenced by torsional forces experienced during the tennis serve.⁶⁸ In addition, the findings presented in the case study by Ireland et al⁶⁸ substantiate earlier findings of a finite element model by Taylor et al.⁶⁴ The biomechanical model analysis consisted of data collected from high-speed video analysis of tennis serve, a musculoskeletal analysis, a finite element based density growth analysis, and an x-ray based bone density analysis.⁶⁴ The model was found to accurately predict bone strength adaptations in the humerus in response to loads experienced during the tennis serve. The torsional forces

created during maximal shoulder ER were found to be responsible for the bone density adaptations observed in the humerus. In contrast, ball impact was predicted to have a limited effect on bone density changes of humerus. The findings by Ireland et al⁶⁸ and Taylor et al⁶⁴ support the theory that bony adaptations in the humerus of racket sport athletes are primarily effected by torsional forces experienced during the overhead service motion as opposed to the forces experienced during contact between the racket and ball.

Overall, there is substantial evidence that supports the theory that asymmetrical overhead athletic activity induces bone strength adaptations in response to the demands placed upon the skeletal system. In particular, overhead throwing and racket sport athletes clearly demonstrate bone strength adaptations in the humerus primarily in response to large torsional loads. These adaptations of the humerus are perceived to be a positive adaptation in overhead throwing and racket sport athletes.^{64,67,68,262,271,274-277,279,283} It is theorized that these same torsional forces may lead to adaptations in HRV, particularly when young athletes are exposed to these forces while the proximal humeral physis is open.^{6,70,77,288} Despite the overwhelming evidence that tennis players demonstrate significant torsional adaptations in bone strength,^{67,68,261,274,275,278} and modeling evidence supporting twisted bone growth (increases in HRV),⁶⁴ there are currently no studies that have reported HRV measures in tennis or other racket sport athletes. Therefore, investigations are warranted in determining the extent of HRV adaptations in this particular group of overhead athletes.

2.3.1.3 Humeral Retroversion Adaptations Associated with Asymmetrical Overhead Activity

In addition to bone's ability to improve its strength characteristics in response to the loads it experiences, there is a growing body of evidence that suggests asymmetrical overhead activity can affect normal growth patterns of HRV. Over the past two decades, several studies have reported increased measures of HRV in the dominant arms of baseball^{45,46,51,53,57,58,60,61,69,71,75,76,109,169,172,190,289-292} handball,¹⁷⁰ softball,⁶⁰ swimming,⁶⁰ and volleyball⁹⁹ athletes. While it is common to find this general trend in the overall population,^{162,175} the magnitude of bilateral difference is much larger among overhead athletes.^{45,58,60,75,76} To date, the consequences of this apparent adaptation in overhead athletes are unclear. Some investigators have speculated that increased HRV is a healthy adaptation in that it allows for a more externally rotated position of the forearm without jeopardizing the stabilizing tissues of the glenohumeral joint.^{21,45,57,61,169,170} However, others have demonstrated a link with injury to the shoulder and elbow in baseball players that demonstrated a lack of or excessive degree of HRV, respectively.^{56,57,61-63}

Earlier studies investigating the variability of HRV in man speculated that the final amount of HRV is a result of primary (hereditary) and secondary (ontogenetic) factors.^{162,163,166,167} The primary factors were discussed earlier in this chapter (see Section 2.1.4.1.1). The secondary factors that affect the final amount of HRV are the opposing muscular forces and functional activities undertaken during the growing years prior to skeletal maturity.^{162,163,166,167} These factors result in torsional loads applied about the long axis of the humerus that have the ability to influence the overall degree of HRV.^{162,163,166,167}

During the years of skeletal growth, the humerus de-rotates from a position of marked retroversion (average of 78° in fetal specimens¹⁷³) to an average measure of approximately 30° .¹⁷³⁻¹⁷⁵ During childhood, the proximal humerus is composed of three primary ossification centers: the humeral head, the greater tuberosity, and the lesser tuberosity. These ossification centers unite to form a single proximal humeral epiphysis between 5 and 7 years of age. The proximal physis is the primary location of longitudinal^{6,288} and torsional growth^{166,289} of the humerus, contributing to 80% of the overall growth of the humerus with 90% of the growth occurring after age 11.^{6,288,293} In the general population, the derotational process of the humerus occurs most rapidly up to the age of 8 years.¹⁶⁵ It then continues at a slower pace until the proximal humeral physis closes at skeletal maturity,¹⁶⁵ which occurs approximately between the ages of 14 and 17 years in females and 16 and 18 years in males.^{6,288} However, it appears that exposure to asymmetrical overhead activities during these years has the potential to inhibit this normal derotational process thereby resulting in marked bilateral asymmetries in HRV.

Studies investigating HRV adaptations in adult overhead athletes consistently report increased measures of HRV in the dominant arm, with average differences ranging from 6.4° to 17.7° .^{45,46,56,60,61,69,76,99,109,170,190,289,294,295} The magnitude of bilateral differences observed in these athletes are substantially larger than the bilateral differences reported in the general population, which typically range from 1° to 4° .^{45,60,174,175} While studies consistently demonstrate a pattern of increased HRV in the dominant arm, there is substantial within-subject variability that likely indicates several confounding factors including age, genetic variation, measurement differences, participation history, and overhead mechanics.⁷⁰

Studies investigating youth overhead athletes have attempted to provide some understanding as to when and how changes occur in the dominant arms that results in bilateral asymmetries in HRV. Thus far, these investigations have included only youth baseball players, and all have demonstrated bilateral asymmetries with increased measures of HRV occurring in the dominant arm.^{51,57,58,60,71,75,172,290,296} Unfortunately, there does not appear to be a consensus as to when these adaptations become evident as these studies have utilized different age ranges and grouping categories in their investigations. Thus, making comparisons is difficult. In a study of youth baseball players enrolled in grades three through eight (ages ranging 9-14 years), Yamamoto et al¹⁷² revealed significant bilateral differences in HRV in 5th graders (average ages were not provided for each grade level) but not in older or younger groups. Although, subject numbers were small in third (n = 1) and fourth graders (n=4), which may have affected their results.¹⁷² Utilizing a similar grouping design, Kurokawa et al²⁹⁰ evaluated HRV measures in youth baseball players enrolled in first through sixth grades. Significant bilateral asymmetries were not revealed until the fourth grade (aged 10-11 years) and above.²⁹⁰ In another study of youth baseball players ranging in ages from 9 to 17 years, bilateral asymmetries in HRV did not become significant until 11 to 12 years of age.²⁹⁶ The findings of these studies suggest that significant bilateral differences in HRV become evident around age 11.^{172,290,296} This coincides with the onset of rapid longitudinal growth of the humerus that occurs at the proximal humeral physis;²⁹³ the predominant site of HRV growth and/or adaptation.^{166,289} In contrast, other studies have demonstrated that bilateral asymmetries are evident in youth baseball players as young as 8 years of age.^{51,71,75}

HRV adaptations in the dominant arm of overhead athletes appear to be in response to the large opposing torsional stresses placed about the long-axis of the humerus while executing the overhead throw (baseball, handball, soft) serve (tennis), or hit (volleyball). During the late-cocking phase of these overhead motions, the distal end of the humerus experiences an ER torque caused by the inertial forces of the forearm, hand, and/or the ball or racket held in the hand.^{55,64} Concurrently, the internal rotators of the shoulder impart an IR torque to the proximal end of the humerus in preparation to transition to the acceleration phase of the overhead movement.^{55,64} Biomechanical studies investigating the kinetics of the overhead throw⁵⁵ and tennis serve⁶⁴ demonstrate these opposing torsional loads imparted about the long axis of the humerus are consistent with the development of HRV. It is suggested that the observed increase in HRV in the dominant arms of overhead athletes is not accentuated by the overhead throw/serve, but instead these torsional forces act to retard the normal derotational (anteverted) growth that occurs during normal skeletal growth.^{55,64} In support of this theory, studies investigating youth overhead athletes have demonstrated that HRV of the nondominant arm decreases with age while HRV in the dominant arm appears to remain constant.^{290,296}

Studies examining sex differences in HRV typically report males having greater measures than females in the general population.^{162,173,175} However, it is less well known how overhead athletic activity affects HRV adaptations in females compared to males as there are a limited number of studies that have investigated these measures in female athletes. Whiteley et al⁶⁰ compared HRV measures across multiple overhead sports (baseball, softball, swimming, and non-overhead athletes) and multiple age levels (adolescents and adults) for both male and female athletes. All athletes regardless of

sport, age level, or sex demonstrated significant bilateral differences with the average difference in favor of increased HRV in the dominant limb. In regard to sex, no significant differences were demonstrated when comparing the amount of side-to-side differences in HRV among all overhead throwing athletes (males = 11.8°, female = 12.3°), adult overhead throwing athletes (male = 12.0°, female = 13.7°), and adolescent overhead throwing athletes (male = 11.2°, female = 11.7°). In contrast, Hibberd et al⁷⁶ reported a significant difference in the amount of side-to-side difference in HRV when comparing intercollegiate baseball and softball players (baseball = 14.1°, softball = 7.9°). In another study examining elite male and female swimmers, Holt et al¹⁰² demonstrated no significant bilateral differences in HRV in the group of females (average difference = 1.0°). Interestingly, no significant sex differences were detected when comparing ipsilateral HRV measures (average difference: dominant = 0.3°, nondominant = 5.7°) despite significant side-to-side differences being detected in male swimmers (average difference = 6.4°).¹⁰² When examining these studies collectively, the contradictory findings limit the generalizability of the effects of asymmetrical overhead activity on HRV adaptations in female athletes. In addition, including swimming athletes in the generalizability of HRV adaptations should be done with caution as swimming requires symmetrical overhead activity of the dominant and nondominant limbs. Thus, more studies are needed that exam HRV measures in female overhead athletes to aid in our understanding of the potential adaptations caused by asymmetrical overhead activity.

HRV appears to be a contributing factor in the rotational ROM adaptations commonly observed in overhead throwing athletes. These athletes typically present with the dominant shoulder demonstrating decreased measures of IR and increased measures

of ER.^{72,73,77,81,85,86,92,103,104} These alterations typically correspond with each other thereby resulting in a TAM that is equal bilaterally, but the arc of motion has shifted to a more externally rotated position on the dominant side.^{24,104} While there is evidence to suggest soft tissue adaptations contribute to these observed alterations in rotational motion,^{20,49,50,297} researchers have speculated that increased HRV measures observed in the dominant shoulders of overhead throwing athletes is the primary cause of rotational asymmetries in these athletes.^{45,169,170} However, the relative contributions of bony and soft tissue adaptations in the observed motion patterns of overhead athletes remains unclear.

Several investigations have attempted to evaluate the influence of HRV on ROM measures; however, the ability to interpret this relationship has proved to be challenging.⁶² Investigators have utilized correlation analyses to examine the relationships between the side-to-side difference in HRV to the amount of IR and ER available in the dominant arms of overhead athletes. When considering the TAM concept,^{24,104} it is plausible to assume that greater differences in HRV will lead to correspondingly lesser measures of IR and greater measures of ER. However, studies have revealed inconsistent relationships ranging from non-significant findings to significantly weak to moderate relationships for both IR and ER.^{46,62,69,75,169,172,295,298} These findings indicate substantial variability in the contributions that HRV has in rotational motion adaptations in the overhead athlete. Nonetheless, the majority of these studies have identified significant relationships, thus HRV appears to contribute significantly to the commonly observed rotational ROM asymmetries in overhead athletes. Specifically, there appears to be a stronger relationship with the degree of HRV

and measures of glenohumeral IR and horizontal adduction, but not with ER.^{45,46,78,109,169,298} For example, Hibberd et al⁵¹ demonstrated that HRV has a significant influence on IR asymmetries in adolescent baseball players. The adolescent baseball players demonstrated significant age-related increases in IR deficits; however, the IR asymmetries remained unchanged across age groups after accounting for HRV.⁵¹ These results indicate HRV adaptations accounted for the age-related increases in IR deficits.⁵¹

The risk of injury associated with HRV adaptations in the overhead athlete is currently not well defined. Several investigators have suggested that increased measures of HRV in the dominant arm of overhead athletes may be a healthy adaptation.^{45,57,61,170,171} Increased HRV positions the forearm in a more externally rotated position relative to the proximal humerus. Thus, overhead athletes with greater measures of HRV are able to position the forearm in what is perceived to be optimal amounts of ER during the cocking phase of the overhead throw/serve/hit.^{45,170} However, those with lesser measures of HRV would require hyperexternal rotation at the glenohumeral joint to achieve the same relative position of the forearm. Hyperexternal rotation of the glenohumeral joint results in overstretching the anterior capsuloligamentous structures that may lead to instability and pain,¹⁷⁰ and has been demonstrated to exacerbate internal impingement forces upon the rotator cuff tendons and posterosuperior labrum.⁵⁴ In addition, others have suggested that hyperexternal rotation may cause excessive twisting and shear forces on the rotator cuff, long head of the biceps brachii, and the superior glenoid labrum (via the peel-back mechanism).^{49,61,299} In support of these proposed injury mechanisms associated with the development of shoulder pathology, studies have linked shoulder injuries to professional baseball and handball players demonstrating smaller

measures of HRV as compared to uninjured players.^{63,170} As such, this adaptation possibly serves as a protective mechanism against injury to the stabilizing structures of the glenohumeral joint.

In contrast to the health benefits that increased HRV may have in overhead athletes, this adaptation may have deleterious effects to the posterior structures of the glenohumeral joint. In order to appreciate these consequences, we must preface with the potential performance enhancing characteristics associated with increased HRV. Being that increased HRV shifts the TAM to a more externally rotated position, overhead athletes are able to position optimally the forearm during the cocking phase of the overhead motion. This, in effect, increases the arc of motion over which forces are applied to the arm during the acceleration phase of the overhead motion. As such, the forces are imparted over a longer period of time thereby increasing angular velocity.^{2,300} Studies have demonstrated that greater measures of ER correspond to higher throwing velocities in pitchers.^{2,300} Consequently, higher distraction forces are imparted on the humerus during the deceleration phase.^{70,110,295} During the deceleration phase, the posterior rotator cuff and capsule are responsible for dissipating the energy created during the acceleration phase.^{47,49,297} Due to a decrease in the available range of IR, overhead athletes with increased HRV are likely placing additional stress to the posterior structures due to a compressed deceleration phase.^{62,110,295} As such, there appears to be a compounding effect between achieving higher throwing velocities and having a compressed period to decelerate the arm. This effect may lead to posterior capsular thickening and/or decreased mobility of the posterior shoulder,^{62,110,295} which both have been linked in the development of shoulder pathology.^{18,21,49}

Several studies have evaluated the importance of the kinetic chain in the proper execution of the overhead throw or serve.^{72,300-302} Theoretically, faulty executions of proximal segments can have injurious effects distally.^{61,303} For example, sufficient IR of the shoulder during the deceleration phase of the overhead throw has been described as a protective mechanism against injury to the distal segments.^{301,302} As such, IR deficits of the shoulder may manifest in the form of injuries at the elbow.³⁰³ Similarly, greater measures of ER during the cocking phase of the overhead throw has been associated with increased valgus moments at the elbow resulting in increased tensile forces at the medial elbow and increased compressive forces laterally.^{2,55,300,304} Again, resulting in an increased risk of injury to the elbow. These alterations in the mechanics of the overhead throw correspond with the rotational alterations of the shoulder that are associated with increased HRV. Interestingly, researchers have demonstrated a link between increased HRV and elbow pathology.^{56,63} In a study of collegiate baseball pitchers, Myers et al⁵⁶ found those with a history of elbow pain demonstrated a greater side-to-side HRV difference (mean difference = 7.2°) than those without an injury history. Similarly, Noonan et al⁶³ reported professional baseball pitchers who sustained an injury to the elbow demonstrated 5° greater HRV in the dominant arm than those without injury.

In summary, there is substantial evidence demonstrating increased HRV measures in the dominant arms of overhead athletes. The development of this bony adaptation is theorized to be the result of repeated exposures to large torsion forces, particularly during the cocking phase of the overhead throw/serve/hit. It appears that exposure to the torsional forces retards the normal derotational growth of the humerus during the years of skeletal immaturity. The significance of this adaptation is unclear as there seems to be

both benefits and consequences. Paradoxically, there may be a “sweet spot” of the right amount of HRV.^{45,51,61-63,109} It appears that an insufficient amount of HRV increases the risk of shoulder injury, whereas an excessive amount of HRV may put the athlete at risk for elbow injury. To date, no studies have reported HRV measures in tennis players despite the similarities between the overhead serve and throw, and the similarities in rotational ROM adaptations of the shoulder. As such, future investigations are warranted to determine if tennis players experience similar adaptations in HRV.

2.3.1.4 Glenoid Retroversion Adaptations Associated with Asymmetrical Overhead Activity

Considering the tremendous forces created at the glenohumeral joint during the overhead throw,^{2,55,300} investigators have theorized that overhead athletes may undergo osseous adaptations of the glenoid.^{45,191} Similar to the development of HRV adaptations, the normal anteversion growth of the glenoid¹⁸⁸ may be hindered due to repeated exposures to these forces during the years of skeletal growth. While substantially smaller in magnitude compared to HRV adaptations, researchers have revealed increased measures of glenoid retroversion (approximately 3.4°) in the dominant arms of professional baseball players when compared to the nondominant arm.^{45,151,190} However, there is conflicting evidence when comparing the dominant arms of throwers to the dominant and nondominant arms of non-throwing populations.^{45,191} Researchers speculate that the adaptation in glenoid orientation occurs in response to the compressive loads experienced during the late cocking phase between the greater tubercle of the humerus and the posterosuperior glenoid.⁴⁵ As a result, this adaptation may contribute to greater measures of ER while also protecting against pathological internal impingement

and superior labral anterior-to-posterior (SLAP) lesions.^{45,151,190} In support of these claims, Sweitzer et al¹⁵¹ reported that professional baseball pitchers without a history of SLAP repair displayed an average of 4.4° greater retroversion in the dominant arm while pitchers with a history of SLAP repair demonstrated no significant differences. In contrast, Drakos et al¹⁹¹ speculated that glenoid adaptations might not be protective. In addition to revealing increased retroversion measures, these investigators demonstrated that the adaptations of the glenoid are more morphologically complex whereby significant increases in glenoid depth were observed. As such, the authors speculated that the posterior glenoid rim becomes more prominent thereby increasing the probability of contact between the undersurface of the rotator cuff in the glenoid rim.¹⁹¹ However, the generalizability of this study is limited due to the inclusion of only symptomatic professional baseball players.¹⁹¹

Considering the direct relationship between the humerus and the glenoid, it is logical to concomitantly examine these structures for adaptations that may be associated with asymmetrical overhead activity. Wyland et al¹⁹⁰ reported that humeral and glenoid retroversion adaptations occur proportionately in the dominant arms of professional baseball pitchers. The investigators revealed a significant positive relationship between HRV and glenoid retroversion resulting in a 2.3:1 “thrower’s ratio.”¹⁹⁰ This relationship was not observed in the nondominant arm,¹⁹⁰ which agrees with the majority of studies that have examined the relationship in the general population.^{175,305} These findings suggest that during the years of skeletal growth overhead throwing induces a coupled adaptation in humeral and glenoid retroversion.¹⁹⁰

2.3.2 Soft Tissue Adaptations

2.3.2.1 Hypermobility Adaptations Associated with Asymmetrical Overhead Activity

In order to meet the demands of the functional activities of the overhead athlete, the shoulder must be able to move through an extreme range of rotational motion. Observed increases in ER in the dominant arm of overhead athletes when assessed in 90° of abduction is thought to be an adaptation in response to the demands of the activity. Kinematic analyses of the overhead throw have revealed that the shoulder may experience ER measures as high as 210° during the late cocking phase.³⁰⁶ While not to the same extent, but still to an extreme degree, tennis players have demonstrated average peak measures of 172° of ER during the corresponding phase of the tennis serve.³⁰⁷ In all likelihood, these extreme measures of ER are not fully endured at the glenohumeral joint. It is likely that movement at the scapulothoracic interface and limitations of the biomechanical models used to analyze data account for some of the motion.^{235,308} Nonetheless, overhead athletes are thought to require a sufficient amount of laxity at the glenohumeral joint to permit these excessive measures of rotational motion. Concurrently, the shoulder must be stable enough to endure these extreme motions without yielding to injury. Wilk and colleagues have described this conundrum, particularly pertaining to overhead throwers, as the “throwers paradox” and have coined the term “throwers laxity” to describe the hypermobility that is thought to be observed during clinical examination.^{24,309} This laxity is described as an acquired, atraumatic adaptation due to stretching of the anterior and inferior capsuloligamentous tissues when the shoulder is repetitively placed in extreme positions of the ER during the late cocking

phase of the overhead throwing/serving motion.³¹⁰⁻³¹⁶ However, there is contradictory evidence that negates this theory of acquired laxity.^{49,72,317-321}

While the literature appears much more consistent regarding increased measures of ER in the dominant shoulder of overhead athletes, the evidence is much more ambiguous regarding the presence of acquired laxity. Some of the earlier investigations utilized manual examination techniques to quantify the amount of glenohumeral translation in the shoulders of overhead throwing athletes. In a study of 76 collegiate athletes, Lintner et al³²² revealed that only 32% of the athletes demonstrated side-to-side translational asymmetries of at least one grade in one direction. Interestingly, of the ones found with bilateral differences, 19 of 24 subjects demonstrated a higher degree of laxity in the nondominant shoulder. Bigliani et al⁷² reported that 61% of pitchers and 47% of position players at the professional level demonstrated a positive sulcus sign in the dominant shoulder. No significant bilateral differences were observed in either the pitchers or position players, which likely indicates the presence of increased congenital laxity in those players. In a study of 25 professional baseball pitchers, Crockett et al⁴⁵ reported no significant differences in glenohumeral laxity measures when comparing the dominant to nondominant sides. However, the authors cautioned against the interpretability of their findings due to a lack of statistical power. Overall, the results of these studies are limited in their interpretability, as intrarater and interrater reliability measures are routinely reported to be poor for manual glenohumeral translation tests.^{317,322,323} In an effort to reduce subjectivity, Sethi et al³²⁴ utilized an electromagnetic tracking system while performing manual tests to quantify the amount of anteroposterior glenohumeral translation in the shoulders of 57 college and professional baseball players.

When examining the data by playing position, pitchers demonstrated significantly greater ER and increased measures of anteroposterior translation in the dominant arm when compared bilaterally. In contrast, the position players demonstrated no significant differences in ER and anteroposterior translation when compared bilaterally. Interestingly, a significant relationship was revealed between the bilateral differences in ER and anteroposterior translation when examined across all players.

Due to the limitations associated with manual translation tests, other investigators have incorporated more reliable and accurate instrumented arthrometers to quantify glenohumeral translational motion in the shoulders of overhead athletes.^{18,317-321} These devices (e.g., Telos, Weiterstadt, Germany and LigMaster, SportsTech, Charlottesville, VA) have allowed researchers to quantify translational motion according to the specified forces applied by the device to the glenohumeral joint. When tested in overhead functional positions (i.e., abduction and ER of the shoulder), researchers have demonstrated that baseball players^{317,319} and swimmers³¹⁸ have equal laxity bilaterally in both anterior³¹⁷⁻³¹⁹ and posterior directions,^{318,319} and when compared to non-overhead athletes.³¹⁸ Crawford and Sauers³²⁰ demonstrated that anterior laxity was significantly reduced in 90° of ER when compared to the neutral rotation position in a group of 22 asymptomatic high school baseball pitchers, which suggests that the integrity of the anteroinferior capsule is intact in the throwing shoulder. In contrast to the findings of Sethi et al,³²⁴ Crawford and Sauers³²⁰ reported no significant differences between the throwing and non-throwing shoulders for total anteroposterior translation, and Borsa et al³¹⁹ were unable to demonstrate a significant relationship between measures of glenohumeral ER and translational motions (anterior and posterior).

Researchers have also utilized arthrometers (i.e., LigMaster) to evaluate joint stiffness in the shoulders of high school and professional baseball pitchers in an attempt to determine the effectiveness of the soft tissues in resisting anteriorly and posteriorly directed translational forces.^{320,321} Crawford and Sauers³²⁰ revealed no significant differences in anterior and posterior glenohumeral stiffness when compared bilaterally in a position of 90° abduction and neutral rotation. However, a significant increase in stiffness against anteriorly directed forces and a concomitant decrease in anterior laxity was displayed in the position of 90° of ER when compared to the neutral position. Similarly, Borsa et al³²¹ demonstrated no significant bilateral differences in anterior and posterior joint stiffness when assessed in 90° of abduction and 60° of ER. No differences between anterior and posterior stiffness were demonstrated in the neutral rotation position³²⁰; however, anterior joint stiffness was significantly greater than posterior stiffness in 60° of ER.³²¹ In consideration of these findings, it appears that the anteroinferior glenohumeral ligament complex provides greater stability in the overhead throwing position as previously described.^{18,206,207}

The consequences of acquired laxity has been a debated topic since the late 1980s with FW Jobe and colleagues first theorizing the concept of an acquired microinstability in the dominant shoulders of overhead throwing athletes.³¹⁰⁻³¹³ It was speculated that acquired laxity in the anteroinferior aspect of joint capsule creates a subtle instability that would allow the humeral head to make abnormal contact with the coracoacromial arch thereby resulting in secondary impingement symptoms.³¹⁰ Thus, acquired laxity was considered the primary pathology and reason for development of shoulder pain in the overhead thrower.³¹⁰⁻³¹³ However, considering the limited success of throwing athletes

fully returning to play following capsulolabral reconstruction suggests the concept of acquired laxity is not comprehensive.^{311,313} CM Jobe et al³¹⁵ expanded on FW Jobe's theory of instability and suggested that repeated exposure to extreme measures of ER can lead to a spectrum of injuries. Specifically, CM Jobe et al³¹⁵ speculated that acquired microinstability of the anteroinferior aspect of the capsule would aggravate posterosuperior impingement and lead to pathological changes in the rotator cuff tendons and labrum. However, several studies have provided evidence that internal impingement may become pathologic without signs of increased laxity. Walch et al³¹⁶ were the first to describe pathological posterosuperior internal impingement in a group of overhead throwers and noted no signs of anterior instability. Sonnery-Cottet et al³²⁵ found similar findings in their study of 25 tennis players. Halbrecht et al³²⁶ disagreed that anterior instability exacerbated internal impingement, and instead concluded that instability would mitigate the effects as the anteriorly subluxed humeral head would result in less contact with the posterosuperior glenoid. Later, Burkhart et al^{49,237,299,327} strongly opposed the concept of microinstability and proposed their theory on the role of posteroinferior capsular contracture in the disabled throwing shoulder. It was suggested that tightness in the posteroinferior capsule would shift the humeral head posterosuperiorly during the late cocking phase of the overhead throw/serve that would allow for greater measures of ER. In what is described as a pathological cascade, the excessive ER shears the biceps anchor resulting in a posterior type II SLAP lesion. Collectively, the posteroinferior contracture of the capsule along with the SLAP lesion is speculated to result in relative redundancy in the anteroinferior capsule resulting in a pseudolaxity as opposed to FW Jobe's³¹⁰⁻³¹³ theory of acquired microinstability.

Overall, there are no quantitative experimental studies that have objectively determined the presence of acquired laxity resulting in a concomitant increase in ER.¹⁸ The only evidence that substantiates this claim are those studies that involve pathological changes in the anteroinferior capsular tissues. Warner et al³²⁸ reported increased measures of ER in symptomatic patients with instability. Other researchers using cadaveric models have demonstrated a corresponding increase in ER from non-destructive ER induced stretching of the anteroinferior capsule.^{54,329} However, these studies are limited in their applicability to overhead athletes as the theory of acquired laxity is a result of atraumatic stretching of the anteroinferior capsular structure of the glenohumeral joint. Despite the lack of objective evidence to confirm the theory of acquired laxity in the overhead athlete, researchers and clinicians continue to publish clinical commentaries and reports on the role that microinstability has in the development of pathology in these athletes.³³⁰⁻³³³ As such, the overall evidence for acquired laxity is confounded and further objective studies are needed to examine joint laxity in the overhead athlete.

2.3.2.2 Hypomobility Adaptations Associated with Asymmetrical Overhead Activity

It is well known that overhead athletes commonly demonstrate altered ranges of motion in the dominant shoulder when compared to the nondominant side and to non-overhead athletes. However, large asymmetries in shoulder motion between the dominant and nondominant sides appear to be problematic as recent studies have noted the risk of injury in these athletes to increase by as much as 1.9- to 9-fold.^{79,80,84} Specifically, glenohumeral IR deficits,^{49,334} horizontal adduction deficits,³³⁴ TAM deficits^{80,335}, and ER deficits,⁷⁹ have all been identified as potential risk factors for injury development in the

overhead athlete. Currently, the direct cause of these hypomobility adaptations are not well known. However, it is most likely that adaptations in bone, capsule, and muscle tissue all contribute in varying degrees to the observed hypomobility adaptations in these athletes.^{59,105,336}

Over the past several years, posterior shoulder immobility has received a lot of attention in the sports medicine community. Burkhart et al^{49,237,327} introduced the concept of glenohumeral internal rotation deficit (GIRD) as the key contributor to shoulder pathology in the overhead athlete. They theorized that overhead throwing athletes develop a contracture of the posterior capsule that leads to alterations in glenohumeral kinematics, which ultimately leads to pathological conditions in the shoulder. The authors noted that most all throwers demonstrate some degree of GIRD; however, they suggested from their clinical observations that a relative side-to-side difference of 25° less IR in the dominant arm was considered a threshold for “symptomatic GIRD.” Since, several researchers have linked GIRD with injury to the shoulder^{80,96,334} and elbow^{303,337} with some researchers demonstrating a link with as little as 11° of GIRD with injury to the upper extremity.³³⁴

In addition to measuring GIRD, researchers have utilized the measurement of passive humeral horizontal adduction (HAD) to quantify posterior shoulder immobility in overhead athletes.^{73,101,298,334} Researchers have revealed that HAD is interrelated to GIRD when assessed in individuals with known shoulder pathology.^{334,338} These studies reported that for every 4-5° of GIRD there was a corresponding 1-cm change in HAD.^{334,338} Myers et al³³⁴ was the first to demonstrate that baseball players diagnosed with internal impingement displayed significantly more GIRD and less HAD as

compared to a group of healthy throwers. Similarly, Vad et al⁹⁶ found tennis players with a history of shoulder pain demonstrated significantly larger deficits in IR and horizontal adduction when compared to healthy players. These findings support the theory by Burkhart et al^{49,237,327} in that posterior immobility may likely play a contributing role in the development of shoulder pathology in the overhead athlete.

Wilk et al¹⁰⁴ were the first to describe the TAM concept. The authors advocated that while overhead throwing athletes commonly display substantial decreases in IR with concomitant increases in ER in the dominant shoulder, the combined measures of IR and ER (i.e., TAM) should remain equal when compared bilaterally. Since this concept was proposed, others have provided evidence that the shift in the ROM of the shoulder is a result of HRV adaptations in the dominant side.^{45,46,61,69} Thus, any resulting deficits in the TAM in the dominant shoulder are suggested to be caused by adaptations in the soft tissues surrounding the joint. In professional baseball pitchers, TAM deficits of the dominant shoulder are considered acceptable when the magnitude of difference is 5° or less when compared to the nondominant side.^{80,105} Researchers have demonstrated prospectively that professional baseball pitchers with TAM deficits greater than 5° are approximately 2.5 times more likely to sustain an injury to the shoulder⁸⁰ or elbow.³³⁵ TAM deficits may also contribute to injuries of the elbow as Garrison et al³³⁷ found significantly greater deficits in high school and collegiate baseball players who sustained an ulnar collateral ligament tear when compared to a group of healthy players. As such, this concept may provide clinicians with a means to detect potentially deleterious alterations in the rotational ROM of the shoulder.^{21,105}

Most recently, researchers have introduced the concept of ER deficiency as a risk factor for shoulder injury in overhead athletes.^{79,105} Wilk et al⁷⁹ defined ER deficiency in professional baseball pitchers as a difference of less than 5° for dominant arm ER when compared to the nondominant side. In other words, it is expected to find baseball players with 5° greater ER in the throwing shoulder, and any differences less than 5° more in the dominant shoulder may impart abnormal stresses to the shoulder that increases the risk of injury.^{79,105} In fact, Wilk et al⁷⁹ revealed that professional pitchers demonstrating ER deficiencies were 2.2 times more likely to sustain a shoulder injury that required time on the disabled list and 4.0 times more likely to experience shoulder surgery. These findings are in stark contrast to the common findings of posterior shoulder immobility (i.e., glenohumeral IR deficit and horizontal adduction deficit). The authors speculated that increased awareness of posterior shoulder immobility has prompted recovery routines to include stretches that address posterior immobility, and more conservative monitoring of pitch counts and rest between outings at the professional level have impacted the outcomes of their study.⁷⁹

While GIRD has received the most attention regarding the overhead athlete's shoulder, the latest evidence has highlighted the importance of also including bilateral differences in the TAM and ER when screening the overhead athlete's shoulder. Traditionally, symptomatic GIRD was viewed as its own entity; however, this may be misleading as it does not provide a complete picture of the ROM profile of the shoulder. Tokish et al²⁹⁴ found that GIRD was present in 35% to 43% of asymptomatic professional baseball pitchers when using three different definitions of GIRD from the literature. In addition, researchers have consistently noted that overhead throwing athletes have

approximately 11° more of HRV in the dominant shoulder.^{46,51,58,60,61} This osseous change in and of itself “predetermines” a given amount of GIRD. However, researchers do not consider the amount of GIRD observed due to increased HRV to be pathologic as HRV is thought to simply shift the TAM to a more externally rotated position.^{21,24} In addition, the observed increases in HRV are typically smaller than the 30° to 50° of GIRD reported in symptomatic athletes, and the amount of HRV will not change once the physes are closed.³³⁶ In light of this, Manske et al¹⁰⁵ recently proposed the concept that overhead athletes may present with two different types of GIRD. Anatomical GIRD (*a*-GIRD) was suggested to describe the normally observed loss of IR while maintaining an adequate amount of ER and a TAM within 5° of the nondominant side. The second type suggested was pathological GIRD (*p*-GIRD), which is used to describe any observed GIRD greater than 18°-20° with a concurrent loss of TAM or an increase in ER deficiency. The authors¹⁰⁵ suggested this would create a more complete picture of the rotational ROM profile in the overhead athlete’s shoulder rather than using the prior suggested thresholds for GIRD of a 20° side-to-side difference in IR.²¹

The cause of hypomobility measures of the shoulder in overhead athletes has garnered much attention over the past several years; however, there appears to be no consensus as to which tissues are primarily responsible. While HRV adaptations are attributed to a shift in the TAM to a more externally rotated position, deficits in either IR or ER with concomitant deficits in the TAM are most likely the result of soft tissue adaptations. Soft tissue adaptations that occur in response to imparted stresses resulting in GIRD and TAM deficits are most commonly thought to be induced by microtrauma-induced scarring of the posterior glenohumeral capsule resulting in contracture of the

tissue,^{21,49,338,339} and/or increases in posterior rotator cuff stiffness.^{18,298,336,340} These adaptations in the posterior capsule and rotator cuff occur in response to the extreme loads endured during the deceleration phase of the overhead throwing/serving motion.^{49,336,341-343}

Currently, there are no *in vivo* studies that have directly determined the existence of soft tissue shortening/contracture in the dominant shoulders of overhead athletes. However, there are several studies that have provided clinical evidence to support a link between soft tissue adaptations and the disabled shoulder due to ROM deficits. Burkhart et al⁴⁹ reported posterior capsular thickening during arthroscopic evaluation of overhead throwing athletes undergoing repair of type II SLAP lesions. Others have utilized ultrasonography to demonstrate increased posterior capsular thickness in the dominant shoulders of overhead throwing athletes.^{71,339,344} Takenaga et al³⁴⁴ demonstrated that both posterior and posteroinferior regions of the glenohumeral capsule were significantly thicker and stiffer in the throwing shoulder of college baseball players. Researchers have speculated that capsular adaptations are likely seen more so in throwers between the ages of 25 and 40 years old.³³⁶ However, Astolfi et al⁷¹ recently reported increased posterior capsule thickness measures in the dominant shoulders of youth baseball players ranging in age from 8 to 12 years old. Interestingly, studies have consistently demonstrated moderate negative relationships between measures of posterior capsule thickness and IR in college baseball players.^{339,344,345} In addition, posterior capsular thickness appears to correspond with increased measures of HRV. These findings suggest that adaptations in the posterior capsular structures may play a pivotal role in the development of rotational deficits in the shoulders of overhead throwing athletes. Furthermore, the findings of

Thomas et al²⁹⁵ suggest that overhead athletes with increased measures of HRV are placing proportionately larger stresses on the posterior capsule during the deceleration and follow through phase of the overhead throwing motion. In support of this claim, professional baseball pitchers with significant GIRD and TAM deficits display greater bilateral differences and absolute dominant side measures of HRV as compared with pitchers without GIRD.⁶² As such, HRV adaptations may play an important role in the development of rotational motion deficits caused by soft tissue adaptations.

Several researchers have used cadaveric models to examine the effects of posterior capsular tightness via surgically induced capsular plication.³⁴⁶⁻³⁵¹ Harryman et al³⁴⁶ revealed significant shifts of the humeral head on the glenoid in a superior direction during flexion and anteriorly during horizontal abduction. Others have examined humeral head displacements in positions of abduction and ER to simulate the late-cocking phase of the overhead throw.^{347,349,351,352} These studies consistently reported significant increases in posterior translations^{349,351} or nonsignificant trends of posterosuperior displacement of the humeral head.^{347,352} However, it should be noted that these studies stretched the anterior capsular to mimic the supposed laxity observed in overhead throwing athletes prior to examining the effects of induced posterior capsular tightness. As noted earlier, there is clinical evidence that suggests overhead athletes do not exhibit anterior capsular laxity.³¹⁷⁻³²⁰ Therefore, the findings of these studies utilizing cadaveric models may be limited because of inducing anterior capsular laxity. In regard to the effects of posterior capsular plication on glenohumeral rotational motion, researchers have revealed significant decreases in IR^{349,351} and TAM.³⁴⁹ Last, Gates et al³⁵¹ demonstrated that posterior translational movement was significantly decreased in the

posterior direction after capsular plication while Grossman et al³⁴⁷ reported no differences in anterior, posterior, superior, and inferior directions. However, it should be noted that the amount of GIRD induced by Grossman et al³⁴⁷ was less than what is commonly found clinically in asymptomatic throwing shoulders, which may explain the differences observed between the two studies.

Researchers have utilized instrumented arthrometers to examine glenohumeral translations in the dominant arms of overhead athletes. As noted by others,¹⁸ if glenohumeral rotational motion is limited by contracture of the posterior capsule then it is plausible to suggest that translation of the glenohumeral joint would be reduced when compared bilaterally. Interestingly, studies utilizing instrumented arthrometry have been unable to confirm this theory. In a study of professional baseball pitchers, Borsa et al³¹⁹ demonstrated no significant side-to-side differences in posterior translation of the glenohumeral joint in the position of 90° abduction and 60° ER, yet IR ROM was significantly reduced by 9.7° in the dominant shoulder. In fact, the average posterior translation measurements in both the dominant and nondominant sides were more than twice the amount of translation detected in the anterior direction. In a similar study of high school baseball pitchers, Crawford and Sauers³²⁰ reported no significant side-to-side differences in posterior laxity or stiffness when tested in the position of 90° abduction and neutral rotation.

As noted earlier, muscular stiffness of the rotator cuff has been suggested as a possible mechanism for the development of posterior shoulder tightness. Several studies have demonstrated that both IR and TAM are significantly reduced after acute bouts of baseball pitching^{341,342} and tennis play.³⁴³ IR appears to be the most affected with

professional baseball pitchers experiencing deficits as large as 15%,³⁴¹ and tennis players demonstrating deficits as large as 41%.³⁴³ The stark contrast in differences between baseball players and tennis players are most likely attributed to differences in design. Researchers examining the effects in baseball players focused on average pitching outings (average pitch-counts were between 50 and 72)^{341,342} whereas the study involving tennis players examined the effects during prolonged tennis play (3-hour tennis match; approximately 250 serves and 547 ground strokes).³⁴³

Decreases in rotational motion of the glenohumeral joint following acute episodes of activity have been attributed to the repetitive eccentric muscle activity experienced during the deceleration and follow-through phases of the overhead throwing and serving motion.^{49,336,341-343} Repetitive eccentric muscle activity has been demonstrated to increase muscular stiffness, referred to as thixotrophy, which is known to affect joint mobility.^{336,353,354} These changes in muscular stiffness are not related to neurological changes, but instead are related to the actual physical damage that occurs to the sarcomere.³³⁶ Researchers have demonstrated actual “sarcomere popping” due to excessive strain imparted on the muscle tissue during eccentric muscle activity.^{353,355} This damage stimulates the release of chemical mediators as part of the normal healing process, which also results in muscle shortening.^{336,353} Researchers have noted that these acute decreases in shoulder motion, while likely a normal physiological process, may predispose these athletes for potential injury if they continue to play with these deficits.^{341,343} Therefore, it is suggested that normal ROM measures are restored prior to the next bout of overhead activity.

In summary, regardless of which tissues are attributing to motion deficits of the shoulder, there is strong evidence demonstrating hypomobility in the dominant shoulder of overhead athletes. The literature supports the concept that significant deficits in shoulder mobility increase the risk of sustaining an injury to the upper extremity. Most studies investigating the effects of hypomobility on the risk of injury mostly involve baseball players. There are far fewer studies that have examined this in tennis players,^{92,93,95,96,356} and of these studies only a few have linked deficits in IR of the shoulder with shoulder injuries/pain in tennis players.^{92,96}

The current recommendations for shoulder rotational mobility in the overhead athlete are based on normal rotational ROM measures in baseball players.^{21,79,80,105,335,336} While similarities exist between the overhead movement patterns of baseball and tennis players, there is evidence to suggest unique mobility patterns in the rotational ranges of motion in the dominant arms of tennis players. Researchers consistently report that tennis players of all levels demonstrate significant deficits in dominant shoulder IR.^{42,50,74,88,90-93,95,107} In addition, studies consistently indicate that IR measures decrease with years of experience and age.^{50,92,106,357} In contrast, there are inconsistencies with regard to ER measures in tennis players. Most studies report that tennis players demonstrate significantly greater measures of ER in the dominant arm^{42,50,91-93,95,106}; however, others have been unable to demonstrate side-to-side differences.^{74,90,107} Similarly, there are inconsistencies regarding changes in ER with years of experience and age.^{50,357} In a cross-sectional study, Kibler et al⁵⁰ reported that ER appears to increase with age; however, Roetert et al³⁵⁷ revealed no significant increases in ER in a longitudinal study that tracked tennis players from the age of 14 to 17 years old. Do these data indicate

tennis players develop IR or ER deficits? Burkhart et al⁴⁹ suggested that IR deficits that exceed ER gains are indicative of pathological GIRD. Interestingly, researchers consistently report these findings in tennis players. However, as mentioned earlier in this section, recent observations have led to a newly proposed method of determining differences between *a*-GIRD and *p*-GIRD by incorporating both IR and TAM measures.¹⁰⁵ Most studies reporting ROM measures for overhead throwing athletes show equivocal TAM measures that are typically within 5° when compared bilaterally.^{45,74,75,77,84,108} However, the majority of studies reporting TAM measures in tennis players report an approximate deficit of 9° in the dominant shoulder,^{42,50,74,88,90,92,93} and some reporting deficits even as large as 20° or more.^{50,95} Manske et al¹⁰⁵ suggested that HRV measurements should be incorporated into the ROM screening of overhead athletes in order to determine the direction and magnitude of the rotational deficit, which is consistent with the suggestions others.^{58,109} However, no studies have 1) determined if bilateral differences in HRV exist in tennis players, and 2) incorporated HRV measures to assist in interpreting shoulder mobility measures in tennis players. As such, future investigations are warranted.

2.3.2.3 Shoulder Internal and External Rotation Strength Adaptations

Associated with Asymmetrical Overhead Activity

Strength profiles of the internal and external rotators of the shoulder in the overhead athlete are well established and demonstrate a sports-specific strength pattern in the dominant arms. Tennis players consistently demonstrate significantly greater measures of IR strength across all age-groups and performance levels,^{42,43,88,112-114,358} yet some studies report no significant bilateral differences in strength of the external

rotators.^{43,88,113,114} Further, Cools et al⁴² revealed that normalized ER strength measures in tennis players remain unchanged when examined across the age continuum from 10 to 20 years of age.

Investigators have suggested that injury risk should not be based on IR or ER strength alone.^{19,43,111} Rather, rotational shoulder strength should be examined based on the external rotator to internal rotator (ER:IR) strength ratio.^{19,43,111} The recommended minimum threshold for distinguishing a healthy muscular balance is an isokinetic ER:IR ratio of 0.66 (210°/s and 300°/s) or an isometric ER:IR ratio of 0.75, with an overall dominant-sided increase of 10% when compared to the nondominant side.^{19,42,43} Recently, Cools et al¹¹² reported normative data for eccentric and isometric strength measures in the overhead athlete using handheld dynamometry. The isometric ER:IR strength ratios for the dominant shoulder in tennis players varied from 0.62 to 0.97, which are slightly higher than the normally recommended values. These differences were likely due to differences in populations studied, and differences in testing position and protocols.¹¹²

Investigators have attempted to determine if rotational motion deficits of the shoulder have an impact on shoulder strength in the dominant shoulders of overhead athletes. Laudner et al³⁵⁹ were unable to demonstrate a relationship between ER strength and glenohumeral IR or horizontal adduction in the dominant shoulders of professional baseball players. The researchers hypothesized that pitchers with weaker ER strength would have greater stresses imposed upon the posterior structures, thus leading to greater adaptations in posterior shoulder tightness. Interestingly, Laudner et al³⁵⁹ utilized absolute measures of posterior shoulder tightness measures rather than side-to-side

differences, which may have compromised their results. When taking side-to-side differences in rotational motion of the shoulder into consideration, others have been able to demonstrate significant effects on shoulder strength.^{115,116} In a study of 193 professional baseball pitchers, Amin et al¹²⁷ reported significant decreases in isometric shoulder abduction strength in pitchers with GIRD ($GIRD \geq 25^\circ$ and TAM deficit $> 5^\circ$) as compared to pitchers without GIRD. Similarly, Guney et al¹¹⁶ reported that adolescent overhead athletes with GIRD ($GIRD \geq 18^\circ$) have significantly lower isokinetic eccentric ER to concentric IR strength ratios when tested at $90^\circ/s$ compared to adolescent overhead athletes without GIRD. The difference in strength ratios were attributed to significantly lower measures of eccentric ER strength observed in those with GIRD. In consideration of these studies, it appears that rotational motion deficits of the glenohumeral joint may have deleterious effects on shoulder strength.

Considering that shoulder strength may be impacted by rotational deficits caused by soft tissue adaptations, it is plausible to consider that HRV adaptations may also contribute to alterations in strength measures. Researchers have demonstrated that overhead athletes with greater measures of HRV are more susceptible to developing GIRD and TAM deficits.⁶² It is thought that increased measures of HRV results in a smaller arc of motion over which the humerus can internally rotate during the deceleration and follow through phases of the overhead throwing motion.^{62,70} Consequently, larger forces must be exerted by the posterior shoulder musculature to decelerate the upper extremity. In addition, varying degrees of HRV may place the rotator cuff musculature at different lengths for any given relative position of the forearm in the range of IR and ER. Furthermore, given that HRV adaptations occur predominantly

at the proximal humeral physis,^{166,289} it is conceivable that larger degrees of HRV could create a disparity in the lengths of the rotator cuff musculature compared to the larger primary movers that insert distal to the proximal physis. These changes in muscular lengths have the potential to affect muscular force production via alterations in the length-tension relationship of the muscle.

To our knowledge, only one study has investigated the impact that HRV may have on shoulder strength. Rhi and So¹¹⁷ compared differences in HRV and isokinetic concentric strength of the dominant shoulders of adolescent baseball players when players were grouped based on years of playing experience (greater than or less than 10 years). Players with more than 10 years of playing experience displayed significantly greater measures of HRV, and demonstrated significantly greater measures of IR and ER strength. Fair to moderate significant correlations were revealed between HRV and both IR and ER strength for the players with more than 10 years of playing experience. In contrast, no significant relationships were demonstrated for the less experienced players. It should be noted that the average age difference between groups was approximately 3 years, which likely explains the significant differences in strength measures as absolute torque values were utilized in all analyses.

In summary, it is well established that overhead athletes demonstrate significant side-to-side differences in rotational strength measures of the shoulder in favor of the dominant arm. However, there is evidence to suggest that adaptations in rotational strength are direction-dependent resulting in muscular imbalances between the internal and external rotators of the shoulder. These imbalances have been linked with injury; therefore, researchers have investigated potential mechanism that may influence these

imbalances. Currently, there is evidence that suggests posterior shoulder tightness may have deleterious effects on shoulder strength while other research suggests increased HRV may have positive implications on strength. However, given the limited number of studies available additional investigations are warranted.

Chapter 3

Methods

3.1 Participants

Forty junior and collegiate tennis players consented to participate in this study; however, one participant was excluded from the study after failing the screening process. As a result, data collected on the remaining thirty-nine participants were included in the final analyses. Junior tennis players were required to be enrolled as a 9th – 12th grade high school student, current member of an area high school team or tennis club/association, and tennis was considered to be the primary sport. Collegiate tennis players were current members of a college or university sponsored tennis team competing in the National Collegiate Athletic Association or National Association of Intercollegiate Athletics. Subjects were recruited via electronic and/or hardcopy flyers that were sent to the coaching staff for distribution, onsite recruitment by the research team, and by word-of-mouth.

Participants were required to meet the following inclusion criteria: 1) between the ages of 14 and 25, and 2) free from any shoulder injury in the 6 weeks prior to testing. Participants were excluded from the study if they met any of the following criteria: 1) any elbow or shoulder surgery within the 6 months previous to testing, 2) any current shoulder or elbow pain that limited play, and 3) the presence of any neurological condition that affected muscular strength and consequent upper extremity ROM. In addition, all participants were screened using the American Shoulder and Elbow Surgeons shoulder assessment form to aid in determining healthy shoulder status

(Appendix 1).³⁶⁰ Tennis players were divided into three age groups: the junior tennis players were divided into two groups consisting of 14-15 year-olds⁵¹ (Younger Juniors) and 16-18 year-old⁵¹ (Older Juniors), and the third group consisted of subjects that were currently participating on intercollegiate tennis teams (Collegiate). All participants were required to read and sign an informed consent form approved by the Duquesne University Institutional Review Board (Appendix 2). For participants under the age of 18, parental or guardian written consent was obtained in addition to athlete assent.

3.2 Procedures

After obtaining written consent, participant demographic data were collected including height, weight, age, and sex. The dominant arm was recorded as the hand that was used to grasp a tennis racket during ground strokes and service. All data were collected prior to any stretching, warm-up, or playing activities. The order of testing was prescribed whereby the ROM measurements occurred first, followed by the collection of strength data, and the assessment of HRV occurred last. The rotational direction of motion and strength tested, as well as the order of limb tested, was randomized to aid in preventing any potential testing bias. This study was field-based; therefore, data were collected at various tennis centers and universities in the Pittsburgh, PA regional area or the Augusta, GA regional area.

3.2.1 Range of Motion

The assessment of rotational ROM of the shoulder was assessed with each subject positioned supine on a treatment table in 90° of shoulder abduction and elbow flexion. A digital inclinometer (Baseline® Digital Inclinometer, 12-1057, Fabrication Enterprises, White Plains, NY) was used for measures of both IR and ER. According to the

manufacturer, the digital inclinometer is accurate to within 0.1° . Rotational ROM assessment techniques utilizing digital inclinometers have been reported to have excellent measures of intrarater reliability with ICCs ranging from .94 to .988, and acceptable measures of precision with SEMs ranging 1.2° to 3.0° .^{76,361,362} Pilot data captured during a previous study revealed excellent measures of intrarater reliability and precision for the primary investigator for measuring IR ($ICC_{3,1} = .908$; $SEM = 2.3^{\circ}$) and ER ($ICC_{3,1} = .974$; $SEM = 2.1^{\circ}$) with a digital inclinometer.

To assess passive IR (Figure 3.1) and ER (Figure 3.2), the examiner used one hand to apply a posteriorly directed force to the anterior aspect of the shoulder girdle to stabilize the scapula. Care was taken to avoid excessive pressure to the humeral head that could potentially alter normal glenohumeral arthrokinematics. A towel roll was placed between the table and subject's arm when necessary to maintain alignment of the humerus in the coronal plane. The humerus was passively rotated with the examiner's other hand. A custom-made grip was attached to the digital inclinometer that allowed the examiner to maintain alignment of the digital inclinometer while grasping the subject's forearm. Once the respective end-ROM was achieved, the angular orientation of the forearm was recorded. The recorded angle indicated the amount of passive rotational motion of the glenohumeral joint achieved from the beginning reference position. The reference position was defined whereby the forearm was vertically oriented. The end-range was indicated by a firm end-feel of the motion and any noticeable increase in an anteriorly-directed pressure by the subject's shoulder girdle into the stabilizing hand of the examiner. Prior to assessing ROM, each subject underwent a familiarization routine involving 2-3 repetitions of progressively increasing arcs of passive IR and ER. ROM

measurements for glenohumeral IR and ER were collected across three test trials, respectively.



Figure 3.1. Subject set-up used for the collection of passive internal rotation range of motion.



Figure 3.2. Subject set-up used for the collection of passive external rotation range of motion.

3.2.2 Strength Measures

The assessment of IR and ER isometric strength of the shoulder was measured with a handheld dynamometer (ergoFET 300, Hogan Health Industries[®], West Jordan, UT) that has a manufacturer's reported accuracy of $\pm 2\%$. The handheld dynamometer is

a clinician-friendly and practical means of assessing strength, particularly in field-based studies, where access to an isokinetic dynamometer is not feasible. While the isokinetic dynamometer is considered the gold-standard method for assessing strength,³⁶³ studies have demonstrated support for concurrent validity with handheld dynamometry for assessing shoulder strength.³⁶³⁻³⁶⁷ The testing of shoulder IR and ER strength with handheld dynamometry has revealed moderate to excellent measures of intrarater reliability with ICCs ranging from .57 to .99,^{112,361,368-371} and tolerable measures of precision with SEMs ranging from 2.59 N to 9.48 N.^{361,367} For this investigation, we assessed shoulder strength using two different testing positions. First, strength was assessed with the subject seated using the 30°-30°-30° position described by Riemann et al.³⁷⁰ This position has demonstrated moderate to excellent measures of intrarater reliability with ICCs ranging from .570 to .921.³⁷⁰ The primary investigator for the present study has demonstrated excellent intrarater reliability and acceptable precision utilizing this technique for collecting IR (ICC_{3,1} = .963; SEM = 10.55 N) and ER (ICC_{3,1} = .967; SEM = 5.63 N) strength measures. For the second technique, the subject was positioned supine with the shoulder positioned in 90° of shoulder abduction and the arm in neutral rotation (90°-0°). Several studies have demonstrated good to excellent intrarater reliability measures utilizing this technique with reported ICCs ranging from .82 to .96.^{112,368,371}

To assess strength in the 30°-30°-30° position (Figure 3.3), the subject sat erect on a treatment table with the thighs fully supported, the lower legs hanging off the edge of the table, and the uninvolved hand resting on the proximal thigh. A bolster was placed between the upper arm and trunk to maintain a glenohumeral position of 30° abduction

and flexion. Also, the bolster aided to limit accessory motion of the upper arm as the subject was directed to squeeze the bolster between the arm and trunk during testing. The forearm was positioned in neutral pronation/supination and aligned parallel with the sagittal plane (30° of ER) while the elbow was held in 90° of flexion. For the $90^\circ-0^\circ$ position (Figure 3.4), the subject was positioned supine on the treatment table with the shoulder elevated 90° in the frontal plane. The forearm was positioned perpendicular to the surface of the treatment table. Again, the forearm was placed in 0° of pronation/supination with the elbow maintained in 90° of flexion. The subject rested his/her uninvolved hand on the abdomen during the assessment. In an effort to limit accessory movement of the upper arm, the tester provided a stabilizing force at the distal aspect of the humerus. Specifically, the tester stabilized on the medial aspect during IR testing, and on the lateral aspect during ER testing.



Figure 3.3. Subject set-up for collecting isometric internal rotation strength of the shoulder in the $30^\circ-30^\circ-30^\circ$ position.



Figure 3.4. Subject set-up for collecting isometric external rotation strength of the shoulder in the 30°-30°-30° position.



Figure 3.5. Subject set-up for collecting isometric internal rotation strength of the shoulder in the 90°-0° position.



Figure 3.6. Subject set-up for collecting isometric external rotation strength of the shoulder in the 90°-0° position.

For all strength measurements, the participants were asked to generate a maximal effort using a “make” test against the unyielding resistance provided by the investigator, which is commonly used in handheld dynamometry studies.^{112,251,361,370,372} The handheld dynamometer was placed over the volar aspect of the distal radioulnar joint when testing IR, and the dorsal aspect for ER. For each trial, the subject was directed to build up their intensity to a maximum effort over a 2-second period and maintain a maximum effort for an additional 3 seconds. The peak force (kgf) exerted by the subject against the handheld dynamometer was recorded for each trial. Three trials, with 30-second rest periods, were performed for each testing position, respectively. Prior to collecting strength data, all subjects were instructed on proper performance of the tests, and performed 2-3 sub-maximal practice trials to increase familiarity with the testing procedures.

3.2.3 Humeral Retroversion

HRV was measured indirectly using musculoskeletal ultrasound. This method of assessment has been demonstrated to have excellent measures of intrarater reliability with

ICCs ranging from .907 to .997, and tolerable measures of precision with SEMs ranging from 0.8° to 5.0° when utilizing a two-person technique.^{62,99,168,373} Myers et al¹⁶⁸ validated the two-person ultrasound technique against the gold-standard method of computerized tomography ($r = .797$, $r^2 = .635$, $p = .001$). For this investigation, we used a one-person ultrasound technique. In a previous study,³⁷⁴ the primary investigator demonstrated excellent reliability (ICC_{3,1} = .992; SEM = 0.8°) with the one-person ultrasound technique. In addition, one-person technique was validated against the two-person ultrasound technique and demonstrated a significant linear relationship between the two techniques ($r^2 = 0.928$, $F_{1,28} = 361.753$, $p < .001$).³⁷⁴

HRV was measured with each subject positioned supine on a treatment table with the involved shoulder abducted to 70° and the elbow flexed to 90° (Figure 3.5). With one hand, the primary investigator positioned and maintained the subject's forearm in a vertical position. Vertical alignment of the ulna was verified with a plumb-line that was secured to the subject's wrist with a hook and loop cinch strap. The plumb-line was allowed to freely hang thereby creating a vertical reference line. While maintaining the forearm in vertical alignment, the investigator used his other hand to manipulate the ultrasound probe (8-13 MHz linear probe, GE Venue 40, GE Healthcare, Milwaukee, WI or 13-6 MHz linear probe, Fujifilm Sonosite M-Turbo, Bothell, WA) on the proximal aspect of the humerus. The ultrasound probe was tilted about the long axis of the humerus to achieve a transverse sectional (short-axis) view of the lesser and greater tubercles. A transparent film with printed horizontal gridlines spaced 0.5 cm apart was affixed to the US unit's display to aid in verifying parallel alignment of ultrasound probe's head with the tubercles. Once the desired position of the ultrasound probe was achieved, the angular

orientation was measured by an attached digital inclinometer (Baseline® Digital Inclinometer, 12-1057, Fabrication Enterprises, White Plains, NY) with respect to the vertical. Positive values were recorded when the probe was tilted laterally from vertical, and negative values were recorded when the probe was tilted medially from vertical. For both extremities, measurements of HRV were collected over three trials.



Figure 3.7. Subject set-up for collecting humeral retroversion angle using musculoskeletal ultrasound.

3.3 Data Reduction

HRV data were converted by subtracting the recorded angle from 90° in order to create positive values for all measures of HRV. Therefore, larger angles represented greater values of HRV, and smaller values indicated lesser values of HRV.

Three-trial means were calculated for measures of IR and ER ROM, HRV, and strength measures. Using the nondominant side as a baseline, bilateral differences were calculated for ROM and HRV measures to represent any sport-specific adaptations that may have resulted due to tennis play. Strength measures were collected from the dominant side only. The following variables were calculated using the respective three-

trial means to aid in determining the impact of HRV on interpreting ROM measures: TAM, TAM difference (TAM Δ), glenohumeral IR difference (IR Δ), glenohumeral ER difference (ER Δ), HRV difference (HRV Δ), HRV-corrected IR (HRVcIR), HRV-corrected ER (HRVcER), HRVcIR difference (HRVcIR Δ), and HRVcER difference (HRVcER Δ). The TAM was calculated as the sum of ipsilateral glenohumeral IR and ER for both the dominant and nondominant sides. HRVcIR and HRVcER were calculated for both the dominant and nondominant sides, and were defined as the available ROM from anatomical neutral, respectively. Anatomical neutral corresponds to the starting position of the forearm whereby the tubercles of the humerus would be aligned parallel with respect to the horizontal.¹⁰⁹ Therefore, HRVcIR was calculated using the equation IR - (90 - HRV), and HRVcER was calculated using the equation ER + (90 - HRV). All difference measures were calculated by subtracting the nondominant measurement from the dominant measurement for each respective variable. Thus, all difference measures resulting in positive integers indicated dominant-sided gains, whereas negative integers represented dominant-sided deficits.

Strength data were converted into strength ratios and were calculated for both testing positions (30°-30°-30°ER:IR and 90°-0°ER:IR) by dividing the averaged peak force of the external rotators by the averaged peak force of the internal rotators, respectively.

3.4 Data Analysis

To address the primary focus of this study, paired-sample t-tests were utilized to determine differences in HRV between dominant and nondominant sides for each age group of tennis players (Specific Aim 1). In addition, a one-way analysis of variance

(ANOVA) was used to compare HRV Δ between age groups (Specific Aim 2). Post hoc comparison procedures were conducted when appropriate using Bonferroni adjustments. For this focus of the study, the level of significance was set at $p < .05$.

For the secondary focus of this study, side-to-side differences were evaluated for IR, ER, TAM, HRVcIR, and HRVcER using multiple paired-sample t-tests (Specific Aim 3). Similarly, multiple one-way ANOVAs will be used to assess differences in IR Δ , ER Δ , TAM Δ , HRVcIR Δ , and HVcER Δ between age groups (Specific Aim 4). All post hoc comparison procedures were conducted when appropriate using Bonferroni adjustments. Pearson correlation coefficient analyses were used to determine if relationships exist between HRV Δ and each of the following: clinical measures used to indicate rotational motion adaptations of the shoulder (IR Δ , ER Δ , and TAM Δ), and clinical measures corrected for HRV Δ (HRVcIR Δ and HRVcER Δ) (Specific Aim 5). These secondary analyses enabled us to determine if playing tennis results in side-to-side differences between these ROM variables, and how age and HRV may affect these ROM variables. For Specific Aims 3 & 4, the level of significance was lowered to $p < .01$ using a Bonferonni correction for multiple statistical testing on five potentially dependent variables on the same set of subjects. However, the alpha-level for the correlation analyses remained at $p < .05$.

Finally, to address the tertiary focus of this study (Specific Aim 6), the relationship between HRV and ER:IR strength ratios, the level of significance was set at $p < .05$. Four Pearson correlation coefficients were used to analyze for relationships between dominant HRV and both 30°-30°-30°ER:IR and 90°-0°ER:IR, and between HRV Δ and both 30°-30°-30°ER:IR and 90°-0°ER:IR.

3.5 Power Analysis

An *a priori* power analysis was conducted for both a paired-sample t-test and a one-way ANOVA with three groups utilizing G*Power (G*Power v3.1.9.2, Düsseldorf, Germany). Effect sizes ($d = 1.10$ and $f = 0.55$) were calculated utilizing data from the literature for humeral retroversion measures in overhead athletes.⁶⁰ For the paired-sample t-test, an estimated sample size of 10 subjects was calculated using an alpha of .05, a desired power of .80, and two tails. Using the same alpha-level and desired level of power, an estimated sample size of 36 subjects was calculated for the one-way ANOVA with three groups.

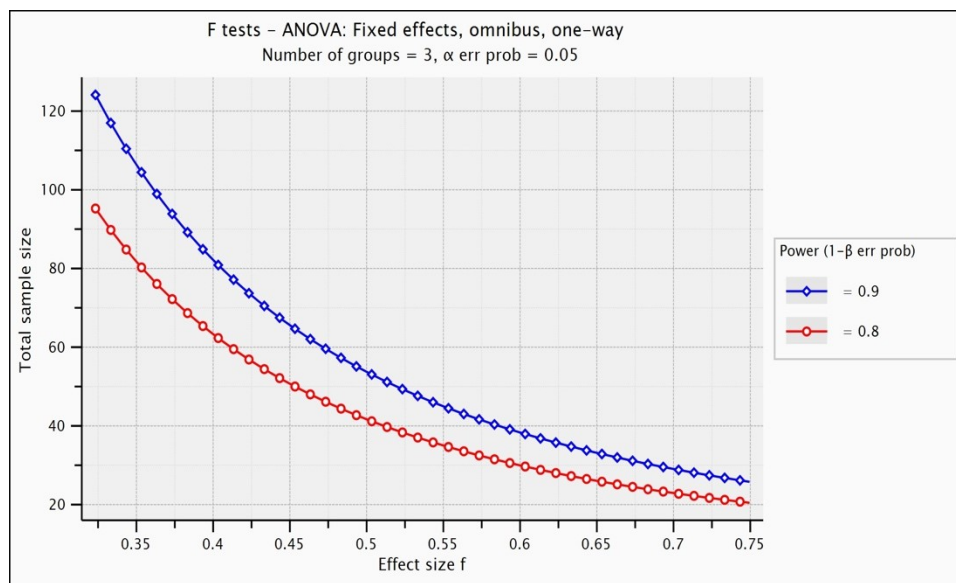


Figure 3.8. Plot depicting total sample size estimation as a function of effect size.

Chapter 4

Junior and Collegiate Tennis Players Display Similar Bilateral Asymmetries of Humeral Retroversion

4.1 Dissertation Primary Focus

The primary focus of this study was to determine if tennis players demonstrate bilateral differences in HRV, and whether these differences would be similar across three different age groups of junior and collegiate players.

Specific Aim 1: To test the hypothesis that tennis players will demonstrate increased HRV in the dominant arm when compared to the nondominant arm for each group of junior and collegiate athletes.

Finding: Tennis players demonstrated increased measures of HRV in their dominant arm across all age groups.

Specific Aim 2: To test the hypothesis that differences will exist between tennis player age groups when comparing side-to-side differences in HRV.

Finding: The magnitude of $HRV\Delta$ was similar across all three age groups of tennis players.

4.2 Results

Forty individuals consented to participate in this study; however, one participant was excluded from the study after failing the screening process. As a result, data collected on the remaining thirty-nine participants were included in the final analyses. Demographic data for the three age groups of tennis players are presented in Table 4.1.

Table 4.1. Participant demographics

Variable	Younger Juniors	Older Juniors	Collegiate
Sex	3 females, 8 males	9 females, 3 males	9 females, 7 males
Age, y*	14.5 ± 0.5	17.1 ± 0.9	19.6 ± 1.2
Height, cm*	171.9 ± 7.9	168.1 ± 8.3	169.9 ± 9.4
Mass, kg*	59.1 ± 8.2	60.9 ± 9.6	69.3 ± 10.0
Onset age of playing, y*	6.3 ± 1.9	7.8 ± 3.4	6.7 ± 1.7
Playing experience, y*	8.2 ± 2.1	9.3 ± 3.4	12.9 ± 1.9

* Values expressed as mean ± standard deviation.

The mean, standard deviation, minimum, and maximum values for the measured variables are presented in Table 4.2. For all age groups, significantly greater measures of HRV were observed in the dominant arm compared to the nondominant arm (younger juniors: $t_{10} = 2.370, p = .039, d = .715$; older juniors: $t_{11} = 2.282, p = .043, d = .659$; collegiate: $t_{15} = 4.042, p = .001, d = 1.011$) (Figure 4.1). However, no significant differences were detected in $HRV\Delta$ when compared across all three groups ($F_{2,36} = .683, p = .511, \eta^2 = .037$).

Table 4.2. Mean, standard deviation, minimum, and maximum values for humeral retroversion and bilateral humeral retroversion difference

Variable	Younger Juniors (n = 11)				Older Juniors (n = 12)				Collegiate (n = 16)			
	\bar{x}	SD	min.	max.	\bar{x}	SD	min.	max.	\bar{x}	SD	min.	max.
DHRV, °	62.9	9.1	52.3	74.9	75.5	11.2	61.0	94.2	71.7	8.5	57.8	89.0
NDHRV, °	56.3	6.8	44.7	67.4	68.6	14.2	45.8	93.7	61.2	6.9	45.6	74.6
$HRV\Delta$, °	6.5	9.2	-8.9	18.7	6.9	10.4	-10.5	24.4	10.5	10.4	-3.7	32.8

DHRV, dominant humeral retroversion; NDHRV, nondominant humeral retroversion; $HRV\Delta$, bilateral humeral retroversion difference.

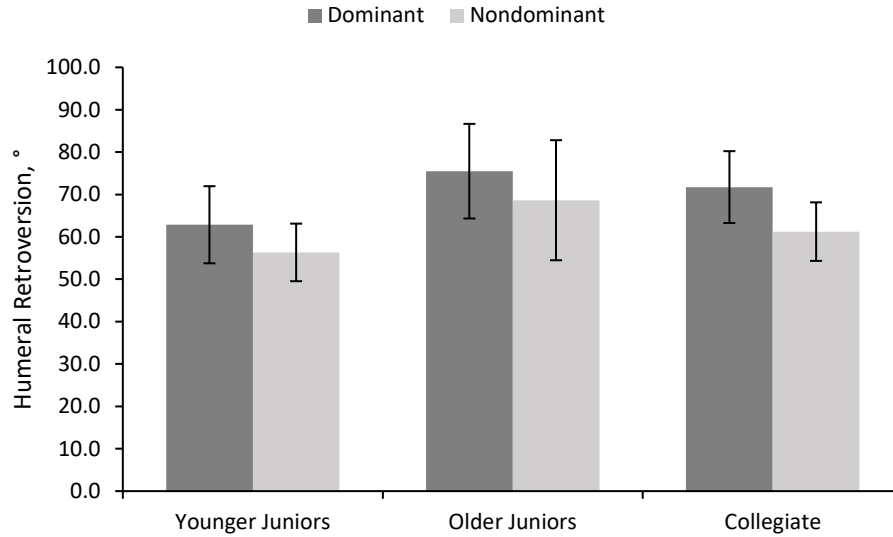


Figure 4.1. Mean dominant and nondominant humeral retroversion measures by age groups.

4.3 Discussion

The purpose of this investigation was to determine if tennis players displayed significant bilateral differences in HRV, and whether the magnitude of $HRV\Delta$ was similar across three age groups of junior and collegiate tennis players. We were able to confirm our first hypothesis as our results revealed significant bilateral differences in HRV in all three groups. The average bilateral difference in HRV ranged across groups from 6.5° to 10.5° . Contrary to our second hypothesis, our results revealed the magnitude of $HRV\Delta$ was similar across all three groups.

We observed significantly greater measures of HRV (approximately 8° overall) in the dominant arm when compared to the nondominant arm in tennis players. The differences observed in our study are much greater than the nominal 1° to 4° of difference observed in the general population.^{45,60,175} Our results are consistent with numerous investigations that have reported significantly greater measures of HRV in the dominant arms of adolescent, collegiate, and professional overhead throwing athletes.^{45,51,60,75,109}

Despite similarities between the overhead throw and tennis serve, there is a paucity in the

literature regarding HRV adaptations in tennis players. To our knowledge, only one other study has reported bilateral asymmetries in HRV in tennis players.⁶⁵ Unfortunately, the researchers did not report specific statistics on the tennis players as data were aggregated with data from baseball and softball players.⁶⁵ Thus, making comparisons with our study difficult.

When comparing HRV Δ across age groups, our data revealed no significant differences, which indicated that HRV Δ was similar between the ages of 14 and 25. These results are consistent with other studies that have investigated HRV adaptations across the age-spectrum of youth and young adults participating in asymmetrical overhead sports. Struminger et al⁶⁵ reported no significant differences in HRV Δ measures when comparing youth (11-14 year-olds) to collegiate overhead athletes, which included baseball, softball, and tennis athletes. In a study using youth and adolescent baseball players, Hibberd et al⁵¹ found no differences in HRV Δ between two different age groups (14-16 years and 16-18 years) of high-school-aged players. In further support, Oyama et al⁵³ observed no significant changes in HRV of the dominant limb over a 1-year period of time in a group of high school baseball players. Our findings provide further support that the majority of torsional growth and/or adaptation takes place prior to the teenage years.

Torsional and longitudinal growth of the humerus mostly occurs at the proximal humeral physis.¹⁶⁵ The derotational growth of the humerus takes place most rapidly before the age of 8 years, and this process continues at a slower pace until skeletal maturity, which occurs around the age of 16 years.¹⁶⁵ While the degree of HRV is largely the result of genetic predisposition, secondary factors (e.g., muscular forces and functional activities) incurred during the years of skeletal growth have been implicated to

have the ability to alter the final degree of HRV.¹⁶³ It is theorized that the opposing torsional forces that occur during the late cocking phase of the overhead throw/serve are substantial enough to inhibit the normal antetorsional growth of the humerus in the skeletally immature, and is manifested as significantly greater measures of HRV of the dominant limb. The age at which significant bilateral differences in HRV becomes evident in overhead athletes is around age 11,²⁹⁶ and has been observed in youth baseball players as young as 8 years old.^{51,75} Considering the majority of the participants (69%) in the current study played no other overhead throwing/serving sports than tennis, provides support that the torsional forces experienced during tennis are substantial enough to affect the normal derotational growth of the humerus. Our results uphold the findings by Taylor et al⁶⁴ who, through biomechanical simulations, found the torsional forces experienced during the tennis serve to be sufficient to affect torsional growth of the humerus.

In contrast, there are no long-term longitudinal studies that provide conclusive evidence that overhead throwing/serving is the cause for the observed increase in dominant limb HRV and/or the large degree of $HRV\Delta$ in overhead athletes. Rather, there may be a natural amount of $HRV\Delta$ in any given person resulting in an inherent culling as individuals age whereby those with greater HRV in the dominant limb remain in his/her sport.⁶⁰ Despite that no significant differences were detected across age groups in $HRV\Delta$, the collegiate group displayed approximately 4° more of side-to-side difference in HRV than both groups of junior players. Thus, future longitudinal studies are warranted that will provide more conclusive evidence regarding the effect that overhead activities have on the development of bilateral asymmetries in HRV.

Numerous investigations have discussed the influential role that HRV adaptations have in the interpretation of clinical measures of shoulder ROM in overhead throwing athletes,^{51,60,75,105,109} and our results revealed that tennis players are no exception. While all three groups demonstrated a pattern of increased HRV in the dominant limb, there was substantial variability in the amount $HRV\Delta$ (range = 43°) in the overall sample with values ranging from one subject with a difference of -10.5° (the nondominant limb displayed greater HRV than the dominant) to another with a 32.8° difference (dominant HRV greater than nondominant HRV). These findings are not unique to tennis players as other researchers have reported considerable variability within and between individuals by as much as 38° ⁶⁰ and 90° ,^{163,173} respectively. However, this does provide further evidence that simple, clinical goniometric measures of rotational shoulder motion are inadequate for clinicians to accurately differentiate between the bony and soft tissue adaptations that may contribute to motion deficits in the dominant arm of overhead athletes. Unfortunately, most clinicians are unable to prescribe directionally-accurate ROM exercises as HRV measures via diagnostic imaging are unattainable. Therefore, future studies are warranted that investigate new clinical-friendly methods that aid clinicians in identifying soft tissue contributions to motion deficits so that appropriate interventions can be prescribed to mitigate injury risk.

We identified a few limitations to consider when interpreting the results of this study. First, we utilized a cross-sectional design for this study. Therefore, we were unable to definitively determine that the observed differences in HRV were in response to the torsional forces experienced while participating in tennis. Second, we decided to combine both male and female data in our sample of junior and collegiate tennis players. Others

have demonstrated that both male and female overhead throwing athletes display significant side-to-side differences in HRV and the amount of HRV Δ is not affected by sex.⁶⁰ For the purposes of this study, we were most interested in determining if tennis players demonstrate bilateral differences in HRV as seen in overhead throwing athletes. Finally, we did not include ROM measurements as part of the primary focus of the overall study, which limits our ability to examine the effects that HRV Δ measures have on interpreting clinical measures of rotational shoulder motion when screening for and implementing interventions to mitigate injury risk.

Chapter 5

Interpreting Soft Tissue Adaptations of the Shoulder After Accounting for Humeral Retroversion Adaptations in Junior and Collegiate Tennis Players

5.1 Dissertation Secondary Focus

The secondary focus of this study was to determine if playing tennis results in bilateral asymmetries in the rotational ROM of the shoulder, and to determine if differences were affected by player age and HRV adaptations of the dominant arm.

Specific Aim 3: To test the hypothesis that tennis players will demonstrate bilateral differences in passive IR and ER measured at 90° of abduction, TAM, HRVcIR, and HRVcER for each group of junior and collegiate athletes.

Finding: The older juniors and collegiate age groups demonstrated significant decreases in passive IR of the dominant arm. All other variables tested did not result in significant bilateral differences.

Specific Aim 4: To test the hypothesis that differences exist between tennis player age groups when comparing IR Δ , ER Δ , TAM Δ , HRVcIR Δ , and HVcER Δ .

Finding: The magnitude of IR Δ , ER Δ , TAM Δ , HRVcIR Δ , and HVcER Δ were similar when compared across all three age groups.

Specific Aim 5: To test the hypothesis that relationships exist between HRV Δ and each of the following: IR Δ , ER Δ , TAM Δ , HRVcIR Δ , and HVcER Δ in tennis players.

Finding: Fair to good significant relationships were observed between HRV Δ and measures of IR Δ , ER Δ , TAM Δ , HRVcIR Δ , and HVcER Δ .

5.2 Results

Descriptive statistics for dominant and nondominant variables of IR, ER, TAM, HRVc IR, and HRVcER are presented in Table 5.1. For Specific Aim 3, older juniors and collegiate tennis players demonstrated significantly less IR in the dominant shoulder compared to the nondominant shoulder ($t_{11} = -4.914, p < .001, d = 1.419$; $t_{15} = -4.652, p < .001, d = 1.163$, respectively), while the younger juniors failed to reach statistical significance ($t_{10} = -3.112, p = .011, d = 0.938$) (Figure 5.1). No significant differences were revealed for any of the remaining side-to-side comparisons of ER, TAM, HRVcIR, and HVcER for any of the age groups (Table 5.1). For Specific Aim 4, there were no significant differences between age groups for any of the difference variables: IR Δ ($F_{2,36} = .375, p = .690, \eta^2 = .020$), ER Δ ($F_{2,36} = .384, p = .684, \eta^2 = .021$), TAM Δ ($F_{2,36} = .480, p = .623, \eta^2 = .026$), HRVcIR Δ ($F_{2,36} = 1.149, p = .328, \eta^2 = .060$), and HVcER Δ ($F_{2,36} = .046, p = .955, \eta^2 = .003$) (Table 5.2). For Specific Aim 5, all correlations were computed with aggregate data from all three age groups considering the results of the ANOVAs above. HRV Δ was significantly correlated with IR Δ ($r = -0.531, p = .001$) (Figure 5.2), ER Δ ($r = 0.654, p < .001$) (Figure 5.3), TAM Δ ($r = 0.332, p = .039$) (Figure 5.4), HRVcIR Δ ($r = 0.735, p < .001$) (Figure 5.5), and HVcER Δ ($r = -0.330, p = .040$) (Figure 5.6).

Table 5.1. Dominant and nondominant measures of range of motion variables by age group

Variable	Younger Juniors (n = 11)			Older Juniors (n = 12)			Collegiate (n = 16)		
	Dominant	Nondominant	<i>p</i>	Dominant	Nondominant	<i>p</i>	Dominant	Nondominant	<i>p</i>
Internal rotation, °	39.8 ± 7.9	46.7 ± 6.9	.011	36.9 ± 9.9	46.3 ± 11.2	< .001	32.4 ± 7.5	40.6 ± 5.4	< .001
External rotation, °	145.2 ± 14.3	140.5 ± 14.7	.221	139.0 ± 14.1	133.3 ± 15.3	.060	134.5 ± 14.2	126.2 ± 14.2	.012
TAM, °	184.9 ± 17.0	187.2 ± 17.0	.580	175.9 ± 16.2	179.7 ± 18.4	.182	166.9 ± 14.7	166.8 ± 14.9	.974
HRVcIR, °	12.6 ± 6.5	13.0 ± 4.4	.845	22.4 ± 8.3	25.0 ± 10.2	.415	14.1 ± 8.0	11.8 ± 7.0	.272
HRVcER, °	172.3 ± 13.0	174.1 ± 14.7	.594	153.5 ± 13.6	154.7 ± 14.2	.523	152.7 ± 15.7	155.0 ± 11.8	.335

Values expressed as mean ± standard deviation. TAM, total arc of motion; HRVcIR, humeral retroversion-corrected internal rotation; HRVcER, humeral retroversion-corrected external rotation.

Table 5.2. Bilateral difference measures of range of motion variables by age group

Variable	Younger Juniors	Older Juniors	Collegiate	<i>p</i>
Internal rotation difference, °	-6.9 ± 7.4	-9.4 ± 6.7	-8.2 ± 7.0	.690
External rotation difference, °	4.7 ± 11.9	5.6 ± 9.3	8.3 ± 11.6	.684
Total arc of motion difference, °	-2.2 ± 12.7	-3.8 ± 9.3	0.1 ± 9.8	.623
Humeral retroversion-corrected internal rotation difference, °	-0.4 ± 6.2	-2.6 ± 10.7	2.3 ± 8.2	.328
Humeral retroversion-corrected external rotation difference, °	-1.8 ± 11.0	-1.2 ± 6.3	-2.3 ± 9.0	.955

Values expressed as mean ± standard deviation.

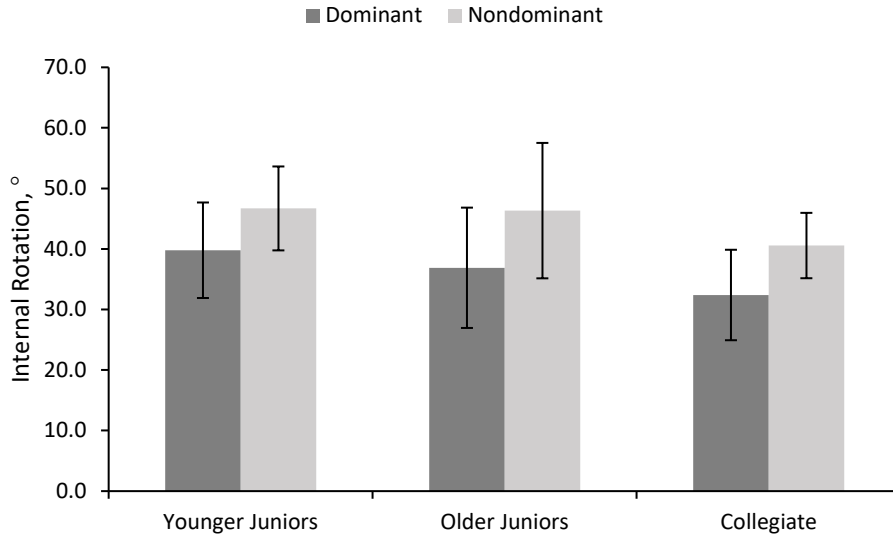


Figure 5.1. Mean dominant and nondominant internal rotation range of motion by age groups.

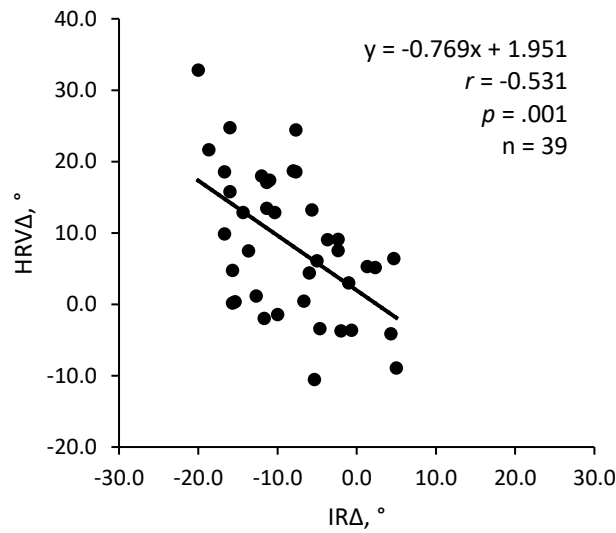


Figure 5.2. Relationship between humeral retroversion difference (HRV Δ) and internal rotation difference (IRA Δ).

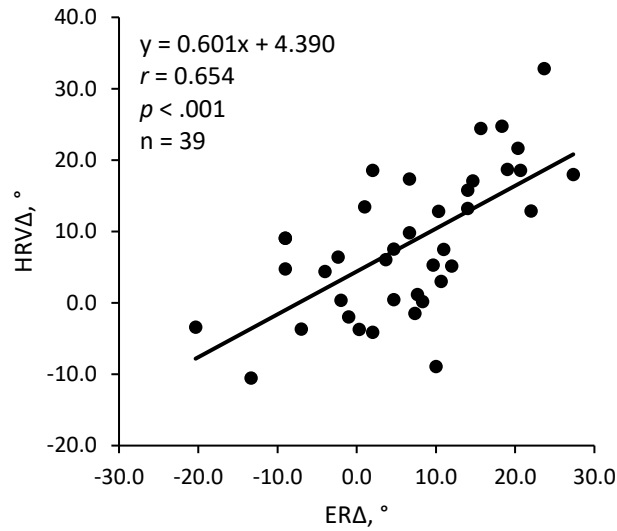


Figure 5.3. Relationship between humeral retroversion difference (HRV Δ) and external rotation difference (ER Δ).

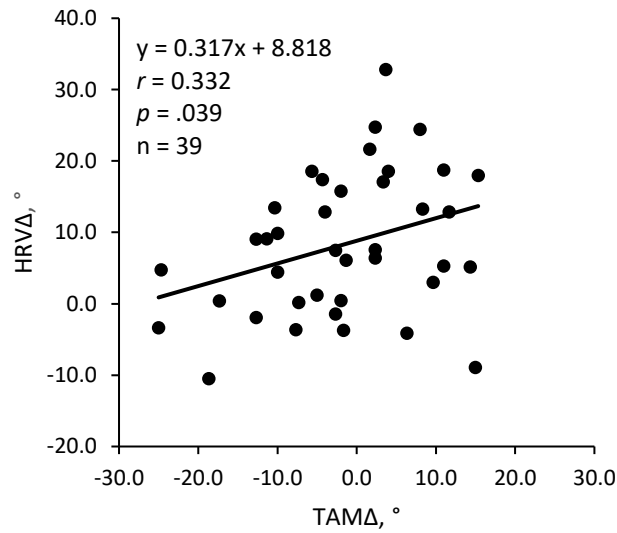


Figure 5.4. Relationship between humeral retroversion difference (HRV Δ) and total arc of motion difference (TAM Δ).

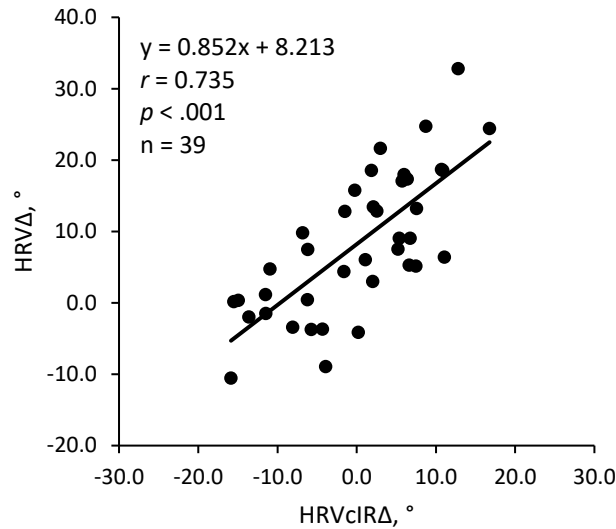


Figure 5.5. Relationship between humeral retroversion difference (HRVΔ) and humeral retroversion-corrected internal rotation difference (HRVcIRA).

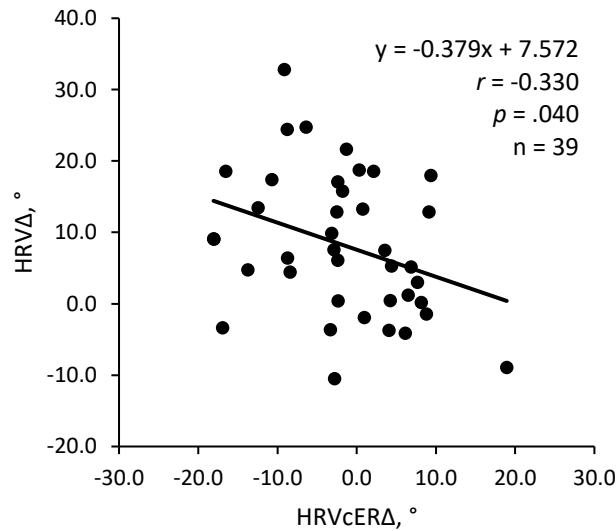


Figure 5.6. Relationship between humeral retroversion difference (HRVΔ) and humeral retroversion-corrected external rotation difference (HRVcERA).

5.3 Discussion

Many investigations have previously reported that overhead throwing athletes often present with bony and soft tissue adaptations of the dominant arm. These adaptations are commonly observed as changes in the rotational ROM of the dominant shoulder

whereby the dominant side displays decreased IR and increased ER when compared to the nondominant side. This shift in the TAM to a more externally rotated position is considered normal in this population as long as the TAM is maintained within 5° of the nondominant side, and it is presumed that HRV is the sole contributor to this observed asymmetry.¹⁰⁴ However, if a TAM deficit is identified then soft tissue restrictions are considered to be the cause and clinical interventions are recommended to improve motion in an attempt to mitigate injury risk. Unfortunately, without knowledge of the bilateral difference in HRV, clinicians are unable to accurately determine the direction and magnitude of soft tissue restrictions for clinical management. The purpose of this investigation was to determine if tennis players displayed bilateral asymmetries in the rotational ROM of the shoulder, and how HRV adaptations of the dominant arm related to and affected the interpretation of clinical ROM measurements. Additionally, we sought to determine if the aforementioned variables were affected by player age.

Similar to other studies involving tennis players,^{42,50,106} we observed significantly decreased IR measures of the dominant side when compared to the nondominant side for the older junior (-9.4°) and collegiate groups (-8.2°). While no significant bilateral difference in IR was revealed for the younger junior group ($p = .011$), we found the IR deficit (-6.9°) in this group to be notable considering the observed large effect size ($d = 0.938$) and no significant differences when comparing $IR\Delta$ across all three groups. For ER measures, our data revealed no significant bilateral differences in any of the groups. Although, the collegiate group trended towards significance ($p = 0.012$, $d = .710$) with an average dominant-sided ER gain of 8.3°, which was proportional to the group's -8.2° IR deficit. The cause for non-significant findings were most likely due to the larger

variability in the ER measures as compared to the smaller variability observed with IR measures. However, in support of our findings other researchers have reported no significant ER bilateral differences in tennis players.^{74,90,107} This finding is in contrast to the more commonly reported pattern of increased ER in tennis players,^{42,50,91-93,95,106} which is also consistently observed in overhead throwing athletes. For TAM measures, there were no significant bilateral differences observed in any of the groups even though both the older junior and collegiate groups displayed significant IR deficits without significant ER gains. In support of our results, a recent study by Nutt et al¹⁰⁶ reported no significant bilateral differences in the TAM in elite tennis players ranging from 11 to 24 years of age. However, it is often reported that tennis players display a TAM deficit of approximately 9°. ^{42,50,74,88,90,92,93} Although it is unclear why these differences exist between studies, it may be explained by differences in the populations studied including, but not limited to, age of the players, years of experience, injury history, and participation in injury prevention/stretching programs. Unfortunately, we did not collect data about our subject's injury history nor information about injury prevention/stretching habits. Regardless, our overall sample of tennis players would not be considered at an increased risk of injury as the ROM measures did not exceed current recommendations used to identify motion deficits.¹⁰⁵

We observed no significant differences when comparing $IR\Delta$, $ER\Delta$, and $TAM\Delta$ across the three groups of tennis players, which indicated no progression of a dominant-sided motion deficit or gain with increased age. In support of our findings, Moreno-Perez et al⁹² reported no significant relationships between years of tennis play or professional play and bilateral difference measures of shoulder motion in professional tennis players.

In contrast, Gillet et al³⁵⁸ demonstrated a significant loss of dominant-sided IR and TAM with an increase in biological age in prepubertal (aged 7 to 13 years) tennis players. However, despite no differences observed in the bilateral difference measures across age groups, there appeared to be a general trend in our data whereby IR, ER, and TAM decreased in both limbs with increased age. Unfortunately, we did not assess this statistically as it was not part of our study. Nonetheless, this observation is supported by others that have observed a bilateral loss of rotational shoulder motion with increased age.⁹² Moreno-Perez et al⁹² revealed negative relationships between rotational ROM measures (IR and ER) and years of tennis practice, years of professional play, and player's age for both the dominant and nondominant shoulders. It is unclear why these changes occur bilaterally, although some plausible reasons may be due to innate inflexibility or training/maturation adaptations of muscular tissue.

While evidence indicates that not all ROM changes are the result of significant soft tissue adaptations,^{49,72,317-321} it has been suggested, particularly with IR motion loss and shoulder pathology, that exposure to asymmetric overhead activity can lead to hypomobility of the posterior shoulder.⁶² It has also been noted that due to greater stresses created about the shoulder in older athletes that the potential exists for greater changes in shoulder mobility to occur with increased age.³³⁶ Considering that there were no significant differences in the bilateral difference measures and HRV data across the age groups in the current study of healthy tennis players, an asymmetrical progression of a motion deficit may indicate maladaptation of the soft tissues of the shoulder, which has been linked with injury in tennis players.⁹⁶ Therefore, it may be beneficial to screen for

these motion changes and/or implement injury prevention programs to mitigate the risk of motion deficits that have been associated with tennis play and injury.

To account for the effect that HRV has on rotational shoulder motion, we corrected the clinical ROM measures by adjusting for the HRV Δ .¹⁰⁹ The adjustment was done to reset the reference position for measuring IR and ER motion based on the positioning of the bicipital tubercles rather than the forearm, which gives a more accurate determination of the available IR and ER motion at the glenohumeral joint. Once the IR measures were corrected for HRV Δ , the observed significant IR deficits in the older junior and collegiate groups were no longer present. In all three groups, the average IR difference became less apparent once these measures were corrected for HRV Δ . These findings suggest that what was perceived clinically as a dominant-sided IR deficit was mostly a reflection of the bilateral difference in HRV rather than a limitation attributed mostly to the soft tissues about the glenohumeral joint. In support of our findings, others have reported similar results in baseball players.^{51,109,375} However, this is in contrast to earlier studies that suggested the primary reason for observed IR deficits in overhead throwers was mostly due to a contracture of the soft tissue structures of the posterior shoulder.⁴⁹

As noted above, there were no significant ER bilateral differences observed in our groups; however, once ER motion was corrected for HRV Δ we observed a similar pattern whereby the nominal dominant-sided ER gains appeared to neutralize. Again, earlier studies based on clinical ROM suggested that overhead athletes displayed increased ER due to an acquired laxity of the glenohumeral joint due to extreme ER motion experienced during the overhead throw/serve.^{24,309} As such, the possibility of overhead

athletes displaying tightness in the anterior soft tissue structures leading to ER deficits were mostly overlooked. However, earlier work by Crockett et al⁴⁵ highlighting the impact of HRV on ER ROM, and more recent studies that have accounted for HRV Δ in ROM measures of baseball players, have revealed not only the neutralizing effect on IR measures but a reciprocation of what was an apparent ER gain to a true ER deficits.^{109,375} Further, a study by Wilk et al⁷⁹ revealed that professional baseball pitchers who demonstrated an ER rotation deficit (clinically determined ROM measures of $< 5^\circ$ ER gain on the dominant side) were 2.2 times more likely to experience a shoulder injury. Given that our sample of healthy junior and collegiate tennis players displayed no significant rotational deficits after accounting for HRV Δ , it would be valuable to clinicians to replicate this study in a sample of symptomatic players.

In an attempt to further expand our understanding of the effects that HRV may have on the interpretation of clinical ROM measures in tennis players, we also included correlation analyses to determine if any significant relationships exist. Not surprisingly, HRV Δ was significantly correlated with both IR Δ ($r = -0.531$) and ER Δ ($r = 0.654$). These relationships support the opinion that greater bilateral differences in HRV (dominant HRV $>$ nondominant HRV) significantly contribute to the commonly observed TAM shift of the dominant limb to a more externally rotated position. However, the relationships were moderate, suggesting that other factors likely contributed to the observed ROM asymmetries in our sample of tennis players. These factors are likely to be a combination from both intrinsic and extrinsic origins. The method used in this study to determine HRV is an indirect technique that is based on the assumption that the ulna is projected perpendicular to the epicondylar axis of the elbow when the elbow is positioned

in 90° of flexion (carrying angle at 90°).¹⁶⁸ However, Hernigou et al³⁷⁶ reported the average carrying angle at 90° is not projected perpendicular and has considerable variability with an average angle of $11.1^\circ \pm 5.2^\circ$ (range 7° to 19°). Other factors may have been associated with subject positioning and any related measurement error between the ROM and HRV measures. For example, the alignment of the forearm for these measures was performed with two different methods. For ROM measures an inclinometer was compressed against the medial aspect of the distal ulna whereas a plumb line was used to align the ulna for the HRV measurements; therefore, soft tissue approximation may have introduced error between measurements.³⁷⁴ Nonetheless, these explanations for the amount unexplained variability between HRV Δ and ROM may be limited via simple correlation analyses, and the amount of unexplained variability suggests that a multivariate approach may be the more appropriate analysis to better explain the relationship between HRV Δ and ROM changes in the overhead throwing/striking athlete. For example, multivariate analyses have been used to gain a better understanding about what factors (e.g., amount of elbow flexion during late cocking, the timing of maximal shoulder external rotation, lower extremity positioning, and pelvic and trunk orientation) influence the loads created about the shoulder and elbow during overhead throwing movements, which have been associated with injury risk.² Considering these loads are transmitted to the same bony and soft tissues that affect ROM at the shoulder, it is suggested that future studies utilize multivariate approaches that examine these same factors. These types of analyses may enable researchers to gain a better understanding of the relationship between HRV adaptations and the commonly observed ROM adaptations in overhead throwing/striking athletes.

In a study including collegiate baseball players, Myers et al¹⁰⁹ similarly reported a significant negative relationship between HRV Δ and IR Δ ($r = -0.66$), but no significant relationship between HRV Δ and ER Δ . While similar differences were reported for average ER Δ , our HRV Δ (approximately 8° across all 3 groups) was much smaller than the 17.7° reported by Myers et al.¹⁰⁹ This may indicate differences in bony and soft tissue adaptations between these two types of athletes as it relates to the differences in demands of the functional activities of the two sports. While the overhead throw and serve have similarities, there are inherent differences that require differences in the allowable motion of the shoulder. Kinematic analyses have revealed that the shoulder experiences average maximum ER measures up to 210° during the overhead throw and 172° during the tennis serve.^{306,307} Further, the overhead throw experiences much larger angular velocities at the shoulder as compared to the overhead serve.^{254,307} These differences in the two overhead activities inherently create dissimilar forces about the shoulder, which possibly lead to differences in bony and soft tissue adaptations.

Considering that HRV-corrected ROM measures provide a truer reflection of the available motion at the glenohumeral joint, we decided to include HRV-corrected measures in our correlation analyses. We observed fair to good relationships between HRV Δ and both HRVcIR Δ ($r = 0.735$) and HRVcER Δ ($r = -0.330$). These relationships suggest that individuals with larger HRV Δ (dominant HRV > nondominant HRV) correspond with larger amounts of true IR gains and ER deficits, and vice versa with smaller HRV Δ . To our knowledge, we are the first to analyze these relationships in overhead athletes, thus making comparisons difficult. Interestingly, the observed relationship between HRV Δ and HRVcIR Δ conflicts with research that suggests greater

measures of dominant-sided HRV have deleterious effects on posterior shoulder mobility.⁶² Noonan et al⁶² reported that professional baseball pitchers who displayed GIRD (IR deficit $\geq 15^\circ$ with concomitant TAM deficit $\geq 10^\circ$) had greater measures of dominant-sided HRV than those without GIRD. Granted, this argument is limited as larger HRV Δ measures don't necessarily indicate large measures of dominant-side HRV when compared to smaller HRV Δ measures, because the bilateral difference measure is based on the nondominant side HRV measure. Nonetheless, our data indicated that for every degree increase in HRV Δ , specifically those with larger HRV Δ ($>8^\circ$ more on the dominant side), there was a corresponding 0.85° increase in true IR gain. The relationship also indicated that for every degree decrease in HRV Δ , specifically those with smaller HRV Δ ($<8^\circ$ more on the dominant side), there was a corresponding 0.85° increase in true IR deficit. What these data may suggest is that there is an optimal amount HRV adaptation in tennis players in regards to the effects that it may have on posterior shoulder mobility. In support, many studies have also suggested that there may be an optimal amount of HRV adaptation in overhead throwing athletes.^{45,60} The same may be argued for anterior shoulder mobility considering the significant negative relationship between HRV Δ and HRVcER Δ . However, with the strength of the relationship being considered fair, there are likely other factors than HRV Δ that play a larger role in the development of true ER gains or deficits. Nonetheless, these relationships do suggest that HRV-corrected ROM measures of the shoulder are moderately influenced by the amount of HRV adaptation in the dominant limb. We recommend future investigations include tennis players with demonstrated GIRD and concomitant TAM deficits to further investigate the relationship between HRV adaptations and ROM deficits.

Over the past several years, GIRD has received a lot of attention as it has been linked with shoulder pain and injury in overhead athletes.^{80,91,92,96,334} However, utilizing GIRD as an independent screening tool for at-risk players is misleading as it does not provide a complete ROM profile of the shoulder. Consistent with our findings, researchers have consistently reported that overhead throwing/striking athletes have increased measures of HRV in the dominant arm. This bony adaptation presets a given amount of GIRD, which should be considered nonpathologic. According to the TAM concept,¹⁰⁴ a dominant-sided shift of the TAM to a more externally rotated position is considered to be a reflection of the HRV Δ if the TAM is maintained within 5° of the nondominant side. Unfortunately, this concept does not allow for a clinician to distinguish between the bony and soft tissue adaptations that may occur in the overhead athlete, particularly those that display a substantial loss of dominant-sided motion. Instead, this concept relies upon the assumption that any deficits observed in the dominant side are indicative of soft tissue restrictions of the posterior shoulder. According to the observed relationship between HRV Δ and HRVcIR Δ in the present study, individuals with larger HRV Δ (>8° more on the dominant side) had IR gains. Attempts by a clinician to improve posterior shoulder mobility in these players could have detrimental effects on the soft tissues of the posterior shoulder. These players may actually have benefited from interventions that improve ER ROM instead considering the significant negative relationship between HRV Δ and HRVcER Δ . In contrast, those with smaller HRV Δ (<8° more on the dominant side) had IR deficits and ER gains, which would require interventions directed to improving internal rotation. Considering our findings, we caution clinicians from implementing intervention strategies to improve IR

based solely on the TAM concept, and strongly recommend that clinicians incorporate HRV measures into their shoulder ROM screenings. In support, recent studies have questioned the utility of the TAM concept. Reuther et al³⁷⁵ demonstrated that TAM Δ is moderately correlated with both HRVcIR Δ and HRVcER Δ , and indicated their findings suggested difficulty in determining the direction of soft tissue deficits with basic clinical goniometry. Further, there is a growing body of evidence that implicates deficits in ER, which represents a shift in the paradigm.^{79,109,375} Unfortunately, we realize that most clinicians are unable to attain measures of HRV, which means they will be unable to accurately determine the direction and magnitude of rotational ROM deficits of the shoulder in order to implement appropriate treatment strategies and monitoring. Researchers have attempted to create clinical methods of measuring HRV via palpation techniques; however, the clinical utility of these methods are questionable due to less than optimal measures of reliability and validity^{373,377}; therefore, the development and investigation of additional clinically-based measures of HRV are warranted. In light of the limitations associated with GIRD, TAM, and difficulties of attaining HRV measurements, Manske et al¹⁰⁵ proposed two different concepts of GIRD to aid clinicians in shoulder ROM screening for overhead athletes. The authors used *a*-GIRD to describe those who present with a nominal loss of IR, but maintain sufficient ER and a TAM within 5° of the nondominant side. The second type, coined *p*-GIRD, is used to identify at-risk players who present with an IR deficit $\geq 18^\circ$ - 20° with a concomitant TAM deficit $>5^\circ$ or ER deficit ($<5^\circ$ ER gain on the dominant side). Unfortunately, we're unable to determine the usefulness of *p*-GIRD as a screening tool as our overall sample of junior and collegiate tennis players did not meet the criteria for *p*-GIRD.

We have identified some limitations that require consideration. First, this study only included healthy junior and collegiate tennis players. We neither included a control group of nonoverhead athletes nor did we include a group of overhead throwing athletes. For the current study, we utilized the tennis players' nondominant limb to serve as the control for the natural growth and development of HRV rather than a group of non-overhead throwers/strikers. Including a group of overhead throwing athletes would have allowed comparisons to determine if bony and soft tissue adaptations are similar between the two types of overhead athletes. Further, by including only healthy tennis players we are unable to generalize the results to an injured population. Next, we utilized a cross-sectional study design and did not control for the onset age of playing tennis, the total years of participation, and participation in other overhead sports. Therefore, some participants may have exhibited greater adaptations in the bony and soft tissues than others due to differences in playing experience. Last, we determined the estimated sample size based on the primary focus of our study. However, the small sample sizes in each group may have affected the ability to detect differences in this secondary focus due to a lack of statistical power for some of the variables. For example, the variability associated with the ER measurements were much larger than the variability associated with IR. We conducted a power analyses *a priori* for external rotation measures that yielded estimated sample sizes of more than 1,000 subjects, which was determined to be unreasonable for this study in consideration of the primary focus. However, we reduced the *p*-value for the statistical analyses for this secondary focus in an attempt to reduce type I errors that are likely to occur due to multiple statistical testing using different potentially dependent variables on the same set of subjects.

Chapter 6

Relationships Do Not Exist Between Adaptations in Humeral Retroversion and Rotational Strength Ratios of the Shoulder

6.1 Dissertation Tertiary Focus

The tertiary focus of this study was to determine if there were observable relationships between the amount of HRV adaption (dominant HRV and HRV Δ) and the strength output of the external and internal rotators of the dominant shoulder (30°-30°-30°ER:IR and 90°-0°ER:IR).

Specific Aim 6: To test the hypothesis that a relationship exists between HRV and the dominant shoulder ER:IR strength ratio in tennis players.

Finding: No significant relationships were observed.

6.2 Results

Descriptive statistics for dominant-sided strength measures are presented in Table

6.1. No significant relationships were observed between any of the paired variables:

dominant HRV and 30°-30°-30°ER:IR ($r = 0.159, p = .332$), dominant HRV and 90°-0°ER:IR ($r = -0.167, p = .309$), HRV Δ and 30°-30°-30°ER:IR ($r = 0.048, p = .774$), and HRV Δ and 90°-0°ER:IR ($r = -0.192, p = .242$).

Table 6.1. External and internal rotational strength data for the dominant shoulder

Variable	30°-30°-30°			90°-0°		
	ER*	IR*	ER:IR	ER*	IR*	ER:IR
Younger juniors	0.15 ± 0.03	0.26 ± 0.06	0.58 ± 0.09	0.21 ± 0.05	0.22 ± 0.06	0.98 ± 0.16
Older juniors	0.16 ± 0.03	0.23 ± 0.05	0.68 ± 0.10	0.21 ± 0.04	0.20 ± 0.05	1.03 ± 0.16
Collegiate	0.18 ± 0.03	0.27 ± 0.06	0.68 ± 0.11	0.24 ± 0.06	0.24 ± 0.05	1.00 ± 0.20
Overall	0.16 ± 0.03	0.26 ± 0.05	0.65 ± 0.11	0.22 ± 0.05	0.22 ± 0.05	1.00 ± 0.17

Values expressed as mean ± standard deviation. 30°-30°-30°, participant was positioned in 30° flexion, 30° abduction, and 30° of external rotation; 90°-0°, participant was positioned in 90° abduction and 0° external/internal rotation; ER, external rotation; IR, internal rotation; younger juniors (n = 11), older juniors (n = 12), collegiate (n = 16), and overall (n = 39).

* Strength data are normalized to mass.

6.3 Discussion

Healthy tennis players are known to consistently demonstrate significant side-to-side differences in rotational strength of the shoulder in favor of the dominant arm.^{42,43,88,112-114,358} However, inconsistencies are reported in the literature regarding strength adaptations of the external rotators as some studies have reported significant increases in the dominant side,¹¹² while others have revealed no bilateral differences.^{43,88,113,114} The reported differences in ER strength may likely be a result of muscular imbalances between the internal and external rotators of the shoulder, which have been linked with injury in tennis players.^{378,379} Unfortunately, it is not clear what potential mechanisms may influence these muscular imbalances.

The purpose of our tertiary focus was to determine if relationships exist between absolute values of HRV and ER:IR strength ratios, and HRVΔ and ER:IR strength ratios. We observed no significant relationships between any of the paired variables of HRV and ER:IR strength ratios. Unfortunately, it is difficult to make direct comparisons with the literature as we are the first to examine these particular relationships. To our knowledge, only one other study has explored the relationship between HRV and rotational strength of the shoulder. Rhi and So¹¹⁷ reported significant fair to moderate positive correlations between dominant side HRV and both IR (180°/s and 300°/s) and ER (300°/s) absolute

isokinetic strength measures in a group of elite baseball players with more than 10 years of playing experience. Their results suggest that greater measures of HRV may have beneficial implications on rotational shoulder strength; unfortunately, they did not provide a rationale. While our results are indifferent, their findings may provide some insight as to why we did not observe significant relationships, particularly concerning the relationship with the absolute values of HRV. Based on both IR and ER strength having positive relationships with HRV,¹¹⁷ it is plausible that utilizing ER:IR ratios negated any detectable relationships that may have existed between HRV and directional-specific strength of the shoulder rotators. In addition, the ER:IR ratios may also have obscured any observable relationships with HRV Δ and strength.

We decided to utilize strength ratios in our study in consideration that injury risk is suggested to be best determined via ER:IR strength ratios rather than direction-specific measures. The ER:IR ratios varied between 0.58 to 1.03 with lower ratios observed in the 30°-30°-30° position as compared to the 90°-0° position. Our results are consistent with what others have reported in studies that utilized similar subject populations, testing positions, and protocols.^{42,112}

Considering that HRV may be positively correlated with both IR and ER strength and may not have been observable utilizing strength ratios, we decided to take a further look into our data. For the group of collegiate tennis players, significant moderate to good relationships were revealed between dominant HRV and mass-normalized IR strength for both testing positions: 30°-30°-30° ($r = 0.563, p = .023$) and 90°-0° ($r = 0.550, p = .027$), and between HRV Δ and mass-normalized IR and ER strength in both positions: IR at 30°-30°-30° ($r = 0.556, p = .025$) and 90°-0° ($r = 0.687, p = .003$), and ER at 30°-30°-30°

($r = 0.682, p = .004$) and 90° - 0° ($r = 0.543, p = .030$). These observed relationships are in concordance with those reported by Rhi and So,¹¹⁷ which indicate that greater adaptations of HRV correspond with greater measures of rotational shoulder strength. Granted, there are differences between study methods; however, it should not detract from the overall observation that HRV adaptations appear to have some type of beneficial association with rotational shoulder strength. Significant relationships were not observed in the two junior tennis player groups; therefore, it may be speculated that there is not enough strength development achieved in the younger-aged players to become noticeable, despite that humeral adaptations have already become apparent. It is interesting that significant correlations using absolute measures of HRV were observed only for IR strength, whereas correlations with $HRV\Delta$ were observed for both IR and ER strength measures. Part of the premise for our investigation was that HRV growth occurs predominantly in the proximal humeral physis, which lies between the insertion of the intrinsic rotator cuff (i.e., subscapularis) and extrinsic primary movers (i.e., latissimus dorsi, pectoralis major, teres major). This may create a disparity in the relative insertions points of the IR muscles resulting in alterations in muscle torque development about the shoulder. However, it appears that $HRV\Delta$ may be a more robust approach than absolute measures of HRV of the dominant arm. This is not surprising as $HRV\Delta$ is viewed as a way to demarcate the extent of the torsional adaptation in response to the loads experienced in the dominant arm during asymmetrical overhead sporting activities. Therefore, those with greater $HRV\Delta$ may have experienced greater loads over time that led to larger adaptations in rotational shoulder strength. Bearing in mind that HRV adaptations may potentially have

positive implications on rotational strength of the shoulder, future investigations are warranted.

We acknowledge a few limitations that warrant discussion. Our sample of tennis players included participants across a rather large age-continuum of young adolescents and young adults. Several studies have demonstrated age-related sport-specific adaptations in shoulder strength in tennis players.^{42,43,112} In a study of 10- to 20-year-old elite tennis players, Cools et al⁴² reported that normalized IR strength significantly increased with age while ER strength remained unchanged. Our sample may have limited our ability to observe any relationships between HRV and rotational shoulder strength, especially considering age-related adaptations of the shoulder that may occur within and/or between the groups of adolescents and young adults. A second limitation is that we utilized handheld dynamometry to measure shoulder rotational strength. While handheld dynamometry can be argued as being more clinically applicable, field-based clinical measurements typically do not reach the same level of accuracy, reliability, and validity as laboratory-based measurements. Nonetheless, we took steps to limit bias in testing by utilizing one tester for all measurements, and we used established, reliable, and validated testing positions described in the literature.^{112,370} Further, as mentioned in the methods section of this dissertation, reliability and precision were established prior to testing for the investigator.

Chapter 7

Conclusion

Participation in tennis subjects one's shoulder to a high volume of repetitive movements, which comes with an increased risk of shoulder injury and/or pain.¹²⁷ The overhead tennis serve requires high forces and torques to be transmitted across the shoulder to produce the rapid upper extremity movements necessary for optimal performance.^{253,255,307} Overtime, these unilateral high forces and torques are thought to lead to asymmetrical musculoskeletal adaptations that manifest in the form of HRV adaptations, altered ranges of shoulder motion, and strength adaptations. However, an excessive or insufficient amount of adaptation may lead to an increased risk of injury. As such, researchers and clinicians have developed ROM screening recommendations that may be used to identify pathological mobility and aid in the implementation of therapeutic interventions to mitigate the risk of injury.^{104,105} A key component that is recommended for clinicians to incorporate into ROM screenings is to include HRV measures considering the substantial effects it has on interpreting shoulder rotational ROM measures. However, to our knowledge there are no studies that have investigated the effects that HRV measures have on the interpretation of ROM measures of tennis players. Likewise, there is a paucity in the literature regarding the effects of HRV adaptations on rotational strength of the shoulder in the overhead throwing/striking athlete (e.g., baseball, softball, handball, and volleyball). Therefore, the overall objective for this study was to develop an understanding of HRV measures in tennis players and its impact on ROM and strength measures of the shoulder.

Consistent with previous studies that have investigated overhead throwing/striking athletes, our results revealed significantly greater measures of HRV in the dominant arms of tennis players. Although it cannot be stated with absolute certainty, when we combine our results along with the body of evidence regarding HRV adaptations in overhead athletes it seems plausible that participating in tennis may affect the normal anteversion growth of the dominant-sided humerus. Considering the large amount of side-to-side variability in HRV measures in the overall population,¹⁷⁵ there may be another plausible explanation for our findings and others. There may be a natural self-selection process by which individuals with greater measures of HRV stay in the sport as they have an anatomical advantage over those with less than optimal HRV measures who eventually leave the sport due to poor performance or injury.⁶⁰ It appears that HRV adaptations take place at an early age and most likely prior the age of 14 as we observed no significant differences in $HRV\Delta$ across the three age groups of junior and collegiate tennis players. Considering that tennis players demonstrate this adaptation, clinicians should be cautious when screening for and implementing interventions for motion deficits based on simple clinical measures. We observed significant IR deficits; however, these deficits were neutralized once the goniometric measures were corrected for $HRV\Delta$. Likewise, the observed nominal amount of ER gains were offset after adjusting for $HRV\Delta$. These findings suggest that correcting rotational ROM measurements by the amount of $HRV\Delta$ may provide a more accurate assessment of the soft tissue adaptations of the shoulder. Further, it appears that the magnitude of $HRV\Delta$ may influence the amount of true ($HRVcIR\Delta$ and $HRVcER\Delta$) soft tissue rotational gains or deficits, which we feel only strengthens the argument for incorporating HRV measures

into the ROM screening process. For example, let's take into consideration the following clinical scenarios:

Table 7.1. Clinical Scenarios

	IR Δ	ER Δ	TAM Δ	HRV Δ	HRVcIR	HRVcER
Clinical Scenario #1	-30°	0°	-30°	10°	-20°	-10°
Clinical Scenario #2	-10°	0°	-10°	15°	5°	-15°

In clinical scenario #1, the athlete presents with a large IR deficit. Utilizing the TAM concept alone, it would be assumed that the IR deficit was due only to restrictions of the soft tissues of the posterior shoulder.¹⁰⁴ It is likely that the clinician would create detrimental changes in the soft tissues of the posterior shoulder if he/she attempted to improve the IR deficit by 25° to achieve a TAM Δ within 5°. However, using the more recent guidelines by Manske et al¹⁰⁵ (without corrections for HRV Δ), the athlete presents with *p*-GIRD. Therefore, it would be indicated to improve IR in the dominant limb >10° to achieve an IR deficit of less than the recommended cut-off of 18°-20°. In addition, ER would need to be improved by at least 5° to eliminate the ER deficiency. However, utilizing these guidelines would leave a motion deficit of approximately 10° that would need to be improved in order to reach the recommended TAM difference to be within 5° of the nondominant side. The clinician would have to decide in which direction to continue mobilizing, which in this scenario it would not likely cause harm if the deficit was split between directions. However, if ROM interventions were prescribed based on HRV-corrected measures, the clinician would know precisely in which direction and magnitude to guide his/her efforts to improve motion. For this athlete, HRV-corrected measures indicate that IR needs to be improved by 20° and ER needs to be improved by 10° in the dominant limb.

In clinical scenario #2, the athlete presents with 10° IR and TAM deficits. If a clinician chose to utilize the TAM concept alone, IR would need to be increased by 5° on the dominant side.¹⁰⁴ Again, this would likely cause detrimental changes in the soft tissues of the posterior shoulder considering the athlete has a 5° IR gain according to when his/her motion is corrected for the HRVΔ. According to Manske et al¹⁰⁵ (without correcting for HRVΔ), the clinician would need to improve dominant-sided ER by 5° to eliminate the ER deficiency, and the remaining 5° deficit would be directed towards improving IR in the dominant limb. It appears that utilizing the guidelines by Manske et al¹⁰⁵ (without correcting for HRVΔ) in the second scenario would remove the risk of injury due to motion deficits. In this second scenario, utilizing HRV-corrected ROM measures would direct the clinician to improve dominant-sided ER by 10°. However, this would mean that the TAM would be greater in the dominant limb by 5° due to the dominant-sided IR gain of 5°. It is currently unclear whether the dominant side TAM should be increased beyond the TAM of the nondominant limb. In this situation, the clinician should use his/her best clinical judgement, and may consider not to improve the TAM of the dominant limb beyond the TAM of the nondominant side. Nonetheless, these clinical scenarios provide further evidence that HRV-corrected measures should be incorporated into shoulder screenings in order to more accurately differentiate the contributions of both bony and soft tissue adaptations to ROM asymmetries in overhead throwing/striking athletes.

Last, we were unable to identify any significant relationships between HRV and shoulder strength for the overall sample of tennis players. However, after reviewing the data carefully, we suspected that the use of ER:IR ratios may have obscured the ability to

detect relationships between HRV and gross strength. We secondarily analyzed our data and observed significant moderate to good relationships between dominant-sided HRV and mass-normalized rotational strength, and between HRV Δ and mass-normalized rotational strength. While we acknowledge these findings were beyond the scope of this dissertation, these findings suggest that other ways of examining strength data may be necessary to fully reveal any existing relationships between HRV adaptations and rotational strength of the shoulder. These findings layout groundwork for future studies.

7.1 Future Directions

This study included a sample of healthy junior and collegiate tennis players. While significant IR deficits were observed in two of the three age groups, none of the players met current clinical criteria for *p*-GIRD.¹⁰⁵ It would be helpful to compare a group of tennis players that meet the criteria for *p*-GIRD against a group without *p*-GIRD to determine differences attributed to HRV adaptations, particularly as it relates to injury. While a large-scale longitudinal study may have the greatest potential, the feasibility of such a study is not probable, particularly for a junior faculty member. As such, a more feasible large-scale cross-sectional study investigating the differences in HRV-corrected ROM measures between those with and without a history of injury would be of value to researchers and clinicians. Currently, *p*-GIRD criteria are based solely on clinically-based goniometric ROM measures. To our knowledge, no studies have classified pathological motion deficits based on HRV-corrected ROM measures in overhead throwing/striking athletes. Further, there may be variability and/or adaptations in the carrying angle at 90° that occur in overhead throwing/striking athletes, which may factor into ROM interpretations and injury risk. This study could also incorporate athletes from various

overhead throwing/striking sports to determine if there are any differences the type of overhead athlete.

Another follow-up study to this dissertation would be one that prospectively incorporates ROM interventions aimed at reducing motion deficits and/or maintaining ROM over a period of time (a playing season) and the effects these interventions have on injury rates. For this particular study, participants would be grouped into one of three groups: a control group, an intervention group that utilizes mobility exercises based on clinical criteria recommended by Manske et al,¹⁰⁵ and an intervention group that utilizes HRV-corrected ROM measurements to guide mobility exercises. This study may be more suited for the collegiate or professional level where there is better access to team staff, therefore better monitoring, that could assist with implementing appropriate interventions. Also, it would likely be more beneficial to implement this study during the off-season so that motion deficits could be corrected prior to the playing season. Then, mobility interventions could be directed at maintaining motion during the season while also monitoring for injury.

Future studies could also provide further insight into the association between HRV adaptations and strength performance of the shoulder rotators. While no significant relationships were observed for the overall sample of tennis players relative to rotational strength ratios, further analysis of our data revealed significant positive relationships between HRV and rotational strength of the shoulder in the collegiate tennis players. This finding suggests that there may be a positive benefit associated with HRV adaptations on the development of strength. However, the benefits may not become evident until the later years of adolescence or young adulthood due to the natural development of strength.

Therefore, it would be beneficial to evaluate this relationship in an appropriately-sized sample of collegiate overhead throwing/striking athletes to see if relationships between dominant-sided HRV or $HRV\Delta$ and mass-normalized rotational strength can be replicated. Additionally, it would be helpful to determine if this relationship is consistent across different populations of overhead throwing/striking athletes. While many consider an increase in strength a performance enhancement, there may be an unknown detrimental effect. It is suggested that increased HRV in the dominant limb allows for increased ER during the late cocking phase of the overhead throw.^{21,45,57,61,169,170} This increase in ER provides a greater arc of motion over which the thrower can generate force against the ball, thus increasing the ball's velocity. However, there is some evidence that links an excessive degree of HRV in the dominant limb with an increased risk of elbow injuries.^{56,61,63} As such, there could be a compounding effect between a larger arc of motion and the ability to generate greater amounts of rotational force due to greater adaptations in HRV, which could potentially be linked with an increased risk of injuries distal (i.e., the elbow) to the shoulder. Therefore, a beneficial component to add to this study would be to determine if there is an injury history that corresponds with the relationship between HRV and rotational shoulder strength.

References

1. Lintner D, Noonan TJ, Kibler WB. Injury patterns and biomechanics of the athlete's shoulder. *Clin Sports Med.* 2008;27(4):527-551.
2. Whiteley R. Baseball throwing mechanics as they relate to pathology and performance - a review. *J Sports Sci Med.* 2007;6(1):1-20.
3. Fleisig GS, Kingsley DS, Loftice JW, et al. Kinetic comparison among the fastball, curveball, change-up, and slider in collegiate baseball pitchers. *Am J Sports Med.* 2006;34(3):423-430.
4. Conte S, Camp CL, Dines JS. Injury trends in major league baseball over 18 seasons: 1998-2015. *Am J Orthop (Belle Mead NJ).* 2016;45(3):116-123.
5. Hill DE, Andrews JR. Stopping sports injuries in young athletes. *Clin Sports Med.* 2011;30(4):841-849.
6. Leonard J, Hutchinson MR. Shoulder injuries in skeletally immature throwers: review and current thoughts. *Br J Sports Med.* 2010;44(5):306-310.
7. Kibler WB, Safran MR. Musculoskeletal injuries in the young tennis player. *Clin Sports Med.* 2000;19(4):781-792.
8. Jayanthi NA, LaBella CR, Fischer D, Pasulka J, Dugas LR. Sports-specialized intensive training and the risk of injury in young athletes: a clinical case-control study. *Am J Sports Med.* 2015;43(4):794-801.
9. Pluim BM, Staal JB, Windler GE, Jayanthi N. Tennis injuries: occurrence, aetiology, and prevention. *Br J Sports Med.* 2006;40(5):415-423.
10. Jayanthi NA, O'Boyle J, Durazo-Arvizu RA. Risk factors for medical withdrawals in United States tennis association junior national tennis tournaments: a descriptive epidemiologic study. *Sports Health.* 2009;1(3):231-235.
11. Lyman S, Fleisig GS, Waterbor JW, et al. Longitudinal study of elbow and shoulder pain in youth baseball pitchers. *Med Sci Sports Exerc.* 2001;33(11):1803-1810.
12. DeFroda SF, Kriz PK, Hall AM, Zurakowski D, Fadale PD. Risk stratification for ulnar collateral ligament injury in major league baseball players: a retrospective study from 2007 to 2014. *Orthop J Sports Med.* 2016;4(2):2325967115627126.
13. Olsen SJ, 2nd, Fleisig GS, Dun S, Loftice J, Andrews JR. Risk factors for shoulder and elbow injuries in adolescent baseball pitchers. *Am J Sports Med.* 2006;34(6):905-912.
14. Tate A, Turner GN, Knab SE, Jorgensen C, Strittmatter A, Michener LA. Risk factors associated with shoulder pain and disability across the lifespan of competitive swimmers. *J Athl Train.* 2012;47(2):149-158.
15. Fabricant PD, Lakomkin N, Sugimoto D, Tepolt FA, Straccolini A, Kocher MS. Youth sports specialization and musculoskeletal injury: a systematic review of the literature. *Phys Sportsmed.* 2016;44(3):257-262.
16. Kibler WB, Safran M. Tennis injuries. *Med Sport Sci.* 2005;48:120-137.
17. Laudner K, Sipes R. The incidence of shoulder injury among collegiate overhead athletes. *J Intercollegiate Sport.* 2009;2(2):260-268.

18. Borsa PA, Laudner KG, Sauers EL. Mobility and stability adaptations in the shoulder of the overhead athlete: a theoretical and evidence-based perspective. *Sports Med.* 2008;38(1):17-36.
19. Cools AM, Johansson FR, Borms D, Maenhout A. Prevention of shoulder injuries in overhead athletes: a science-based approach. *Braz J Phys Ther.* 2015;19(5):331-339.
20. Sciascia A, Kibler WB. The pediatric overhead athlete: what is the real problem? *Clin J Sport Med.* 2006;16(6):471-477.
21. Kibler WB, Kuhn JE, Wilk K, et al. The disabled throwing shoulder: spectrum of pathology-10-year update. *Arthroscopy.* 2013;29(1):141-161 e126.
22. Kibler WB, Wilkes T, Sciascia A. Mechanics and pathomechanics in the overhead athlete. *Clin Sports Med.* 2013;32(4):637-651.
23. Tokish JM. Acquired and adaptive changes in the throwing athlete: implications on the disabled throwing shoulder. *Sports Med Arthrosc.* 2014;22(2):88-93.
24. Wilk KE, Obma P, Simpson CD, Cain EL, Dugas JR, Andrews JR. Shoulder injuries in the overhead athlete. *J Orthop Sports Phys Ther.* 2009;39(2):38-54.
25. Malina RM. Early sport specialization: roots, effectiveness, risks. *Curr Sports Med Rep.* 2010;9(6):364-371.
26. Jayanthi N, Pinkham C, Dugas L, Patrick B, Labella C. Sports specialization in young athletes: evidence-based recommendations. *Sports Health.* 2013;5(3):251-257.
27. Ericsson KA, Krampe RT, Tesch-Römer C. The role of deliberate practice in the acquisition of expert performance. *Psychol Rev.* 1993;100(3):363.
28. Gladwell M. *Outliers: The Story of Success.* London, NY: Allen Lane; 2008.
29. LaPrade RF, Agel J, Baker J, et al. AOSSM early sport specialization consensus statement. *Orthop J Sports Med.* 2016;4(4):2325967116644241.
30. Intensive training and sports specialization in young athletes. American Academy of Pediatrics. Committee on Sports Medicine and Fitness. *Pediatrics.* 2000;106(1 Pt 1):154-157.
31. Brenner JS, Council On Sports Medicine and Fitness. Sports specialization and intensive training in young athletes. *Pediatrics.* 2016;138(3).
32. Valovich McLeod TC, Decoster LC, Loud KJ, et al. National Athletic Trainers' Association position statement: prevention of pediatric overuse injuries. *J Athl Train.* 2011;46(2):206-220.
33. DiFiori JP, Benjamin HJ, Brenner J, et al. Overuse injuries and burnout in youth sports: a position statement from the American Medical Society for Sports Medicine. *Clin J Sport Med.* 2014;24(1):3-20.
34. Garber CE, Blissmer B, Deschenes MR, et al. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Med Sci Sports Exerc.* 2011;43(7):1334-1359.
35. Janssen I, Leblanc AG. Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. *Int J Behav Nutr Phys Act.* 2010;7:40.

36. Marks BL. Health benefits for veteran (senior) tennis players. *Br J Sports Med.* 2006;40(5):469-476.
37. Pluim BM, Staal JB, Marks BL, Miller S, Miley D. Health benefits of tennis. *Br J Sports Med.* 2007;41(11):760-768.
38. Elder SJ, Roberts SB. The effects of exercise on food intake and body fatness: a summary of published studies. *Nutr Rev.* 2007;65(1):1-19.
39. Goolsby MA, Boniquit N. Bone health in athletes. *Sports Health.* 2017;9(2):108-117.
40. Eijsvogels TM, George KP, Thompson PD. Cardiovascular benefits and risks across the physical activity continuum. *Curr Opin Cardiol.* 2016;31(5):566-571.
41. Chandler TJ, Ellenbecker TS, Roetert EP. Sport-specific muscle strength imbalances in tennis. *Strength & Conditioning.* 1998;20(2):7-10.
42. Cools AM, Palmans T, Johansson FR. Age-related, sport-specific adaptations of the shoulder girdle in elite adolescent tennis players. *J Athl Train.* 2014;49(5):647-653.
43. Ellenbecker T, Roetert EP. Age specific isokinetic glenohumeral internal and external rotation strength in elite junior tennis players. *J Sci Med Sport.* 2003;6(1):63-70.
44. Ellenbecker TS, Mattalino AJ. Concentric isokinetic shoulder internal and external rotation strength in professional baseball pitchers. *J Orthop Sports Phys Ther.* 1997;25(5):323-328.
45. Crockett HC, Gross LB, Wilk KE, et al. Osseous adaptation and range of motion at the glenohumeral joint in professional baseball pitchers. *Am J Sports Med.* 2002;30(1):20-26.
46. Reagan KM, Meister K, Horodyski MB, Werner DW, Carruthers C, Wilk K. Humeral retroversion and its relationship to glenohumeral rotation in the shoulder of college baseball players. *Am J Sports Med.* 2002;30(3):354-360.
47. Thomas SJ, Swanik KA, Swanik C, Huxel KC. Glenohumeral rotation and scapular position adaptations after a single high school female sports season. *J Athl Train.* 2009;44(3):230-237.
48. Wilk K, Arrigo C. An integrated approach to upper extremity exercises. *Orthop Phys Ther Clin North Am.* 1992;1(2):337-360.
49. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part I: pathoanatomy and biomechanics. *Arthroscopy.* 2003;19(4):404-420.
50. Kibler WB, Chandler TJ, Livingston BP, Roetert EP. Shoulder range of motion in elite tennis players. Effect of age and years of tournament play. *Am J Sports Med.* 1996;24(3):279-285.
51. Hibberd EE, Oyama S, Myers JB. Increase in humeral retroversion accounts for age-related increase in glenohumeral internal rotation deficit in youth and adolescent baseball players. *Am J Sports Med.* 2014;42(4):851-858.
52. Moore-Reed SD, Kibler WB, Myers NL, Smith BJ. Acute changes in passive glenohumeral rotation following tennis play exposure in elite female players. *Int J Sports Phys Ther.* 2016;11(2):230-236.

53. Oyama S, Hibberd EE, Myers JB. Changes in humeral torsion and shoulder rotation range of motion in high school baseball players over a 1-year period. *Clin Biomech (Bristol, Avon)*. 2013;28(3):268-272.
54. Mihata T, McGarry MH, Neo M, Ohue M, Lee TQ. Effect of anterior capsular laxity on horizontal abduction and forceful internal impingement in a cadaveric model of the throwing shoulder. *Am J Sports Med*. 2015;43(7):1758-1763.
55. Sabick MB, Kim YK, Torry MR, Keirns MA, Hawkins RJ. Biomechanics of the shoulder in youth baseball pitchers: implications for the development of proximal humeral epiphysiolysis and humeral retrotorsion. *Am J Sports Med*. 2005;33(11):1716-1722.
56. Myers JB, Oyama S, Rucinski TJ, Creighton RA. Humeral retrotorsion in collegiate baseball pitchers with throwing-related upper extremity injury history. *Sports Health*. 2011;3(4):383-389.
57. Whiteley RJ, Adams RD, Nicholson LL, Ginn KA. Reduced humeral torsion predicts throwing-related injury in adolescent baseballers. *J Sci Med Sport*. 2010;13(4):392-396.
58. Whiteley R, Ginn K, Nicholson L, Adams R. Indirect ultrasound measurement of humeral torsion in adolescent baseball players and non-athletic adults: reliability and significance. *J Sci Med Sport*. 2006;9(4):310-318.
59. Whiteley R, Ocegüera M. GIRD, TRROM, and humeral torsion-based classification of shoulder risk in throwing athletes are not in agreement and should not be used interchangeably. *J Sci Med Sport*. 2016;19(10):816-819.
60. Whiteley RJ, Ginn KA, Nicholson LL, Adams RD. Sports participation and humeral torsion. *J Orthop Sports Phys Ther*. 2009;39(4):256-263.
61. Polster JM, Bullen J, Obuchowski NA, Bryan JA, Soloff L, Schickendantz MS. Relationship between humeral torsion and injury in professional baseball pitchers. *Am J Sports Med*. 2013;41(9):2015-2021.
62. Noonan TJ, Shanley E, Bailey LB, et al. Professional pitchers with glenohumeral internal rotation deficit (GIRD) display greater humeral retrotorsion than pitchers without GIRD. *Am J Sports Med*. 2015;43(6):1448-1454.
63. Noonan TJ, Thigpen CA, Bailey LB, et al. Humeral torsion as a risk factor for shoulder and elbow injury in professional baseball pitchers. *Am J Sports Med*. 2016;44(9):2214-2219.
64. Taylor RE, Zheng C, Jackson RP, et al. The phenomenon of twisted growth: humeral torsion in dominant arms of high performance tennis players. *Comput Methods Biomech Biomed Engin*. 2009;12(1):83-93.
65. Struminger AH, Atanda AJ, Buckley TA, Richards JG, Swanik CB. Ultrasound comparisons of bilateral asymmetries among youth and collegiate overhead athletes of different sports. *J Athl Train*. 2018;53(6s):S-17.
66. Jones HH, Priest JD, Hayes WC, Tichenor CC, Nagel DA. Humeral hypertrophy in response to exercise. *J Bone Joint Surg Am*. 1977;59(2):204-208.
67. Haapasalo H, Kontulainen S, Sievanen H, Kannus P, Jarvinen M, Vuori I. Exercise-induced bone gain is due to enlargement in bone size without a change in volumetric bone density: a peripheral quantitative computed tomography study of the upper arms of male tennis players. *Bone*. 2000;27(3):351-357.

68. Ireland A, Degens H, Maffulli N, Rittweger J. Tennis service stroke benefits humerus bone: is torsion the cause? *Calcif Tissue Int.* 2015;97(2):193-198.
69. Chant CB, Litchfield R, Griffin S, Thain LM. Humeral head retroversion in competitive baseball players and its relationship to glenohumeral rotation range of motion. *J Orthop Sports Phys Ther.* 2007;37(9):514-520.
70. Greenberg EM, Fernandez-Fernandez A, Lawrence JT, McClure P. The development of humeral retrotorsion and its relationship to throwing sports. *Sports Health.* 2015;7(6):489-496.
71. Astolfi MM, Struminger AH, Royer TD, Kaminski TW, Swanik CB. Adaptations of the shoulder to overhead throwing in youth athletes. *J Athl Train.* 2015;50(7):726-732.
72. Bigliani LU, Codd TP, Connor PM, Levine WN, Littlefield MA, Hershon SJ. Shoulder motion and laxity in the professional baseball player. *Am J Sports Med.* 1997;25(5):609-613.
73. Downar JM, Sauers EL. Clinical measures of shoulder mobility in the professional baseball player. *J Athl Train.* 2005;40(1):23-29.
74. Ellenbecker TS, Roetert EP, Bailie DS, Davies GJ, Brown SW. Glenohumeral joint total rotation range of motion in elite tennis players and baseball pitchers. *Med Sci Sports Exerc.* 2002;34(12):2052-2056.
75. Greenberg EM, Lawrence JT, Fernandez-Fernandez A, McClure P. Humeral retrotorsion and glenohumeral motion in youth baseball players compared with age-matched nonthrowing athletes. *Am J Sports Med.* 2017;45(2):454-461.
76. Hibberd EE, Oyama S, Tatman J, Myers JB. Dominant-limb range-of-motion and humeral-retrotorsion adaptation in collegiate baseball and softball position players. *J Athl Train.* 2014;49(4):507-513.
77. Meister K, Day T, Horodyski M, Kaminski TW, Wasik MP, Tillman S. Rotational motion changes in the glenohumeral joint of the adolescent/Little League baseball player. *Am J Sports Med.* 2005;33(5):693-698.
78. Ruotolo C, Price E, Panchal A. Loss of total arc of motion in collegiate baseball players. *J Shoulder Elbow Surg.* 2006;15(1):67-71.
79. Wilk KE, Macrina LC, Fleisig GS, et al. Deficits in glenohumeral passive range of motion increase risk of shoulder injury in professional baseball pitchers: a prospective study. *Am J Sports Med.* 2015;43(10):2379-2385.
80. Wilk KE, Macrina LC, Fleisig GS, et al. Correlation of glenohumeral internal rotation deficit and total rotational motion to shoulder injuries in professional baseball pitchers. *Am J Sports Med.* 2011;39(2):329-335.
81. Almeida GP, Silveira PF, Rosseto NP, Barbosa G, Ejnisman B, Cohen M. Glenohumeral range of motion in handball players with and without throwing-related shoulder pain. *J Shoulder Elbow Surg.* 2013;22(5):602-607.
82. Clarsen B, Bahr R, Andersson SH, Munk R, Myklebust G. Reduced glenohumeral rotation, external rotation weakness and scapular dyskinesis are risk factors for shoulder injuries among elite male handball players: a prospective cohort study. *Br J Sports Med.* 2014;48(17):1327-1333.
83. Quadros GA, Dohnert MB. Humeral retroversion and shoulder rotational mobility in young handball practitioners. *Acta Ortop Bras.* 2015;23(6):299-302.

84. Shanley E, Rauh MJ, Michener LA, Ellenbecker TS, Garrison JC, Thigpen CA. Shoulder range of motion measures as risk factors for shoulder and elbow injuries in high school softball and baseball players. *Am J Sports Med.* 2011;39(9):1997-2006.
85. Chandler TJ, Kibler WB, Uhl TL, Wooten B, Kiser A, Stone E. Flexibility comparisons of junior elite tennis players to other athletes. *Am J Sports Med.* 1990;18(2):134-136.
86. Chang BF, Liu CC, Chang HY. Characteristic of shoulder and hip rotation range of motion in adolescent tennis players. *J Sports Med Phys Fitness.* 2018;58(4):450-456.
87. Chinn CJ, Priest JD, Kent BE. Upper extremity range of motion, grip strength, and girth in highly skilled tennis players. *Phys Ther.* 1974;54(5):474-483.
88. Ellenbecker TS. Shoulder internal and external rotation strength and range of motion of highly skilled junior tennis players. *Isokinet Exerc Sci.* 1992;2(2):65-72.
89. Ellenbecker TS, Roetert EP. Effects of a 4-month season on glenohumeral joint rotational strength and range of motion in female collegiate tennis players. *J Strength Cond Res.* 2002;16(1):92-96.
90. Ellenbecker TS, Roetert EP, Piorkowski PA, Schulz DA. Glenohumeral joint internal and external rotation range of motion in elite junior tennis players. *J Orthop Sports Phys Ther.* 1996;24(6):336-341.
91. Marcondes FB, de Jesus JF, Bryk FF, de Vasconcelos RA, Fukuda TY. Posterior shoulder tightness and rotator cuff strength assessments in painful shoulders of amateur tennis players. *Braz J Phys Ther.* 2013;17(2):185-194.
92. Moreno-Perez V, Moreside J, Barbado D, Vera-Garcia FJ. Comparison of shoulder rotation range of motion in professional tennis players with and without history of shoulder pain. *Man Ther.* 2015;20(2):313-318.
93. Schmidt-Wiethoff R, Rapp W, Mauch F, Schneider T, Appell HJ. Shoulder rotation characteristics in professional tennis players. *Int J Sports Med.* 2004;25(2):154-158.
94. Stanley A, McGann R, Hall J, McKenna L, Briffa NK. Shoulder strength and range of motion in female amateur-league tennis players. *J Orthop Sports Phys Ther.* 2004;34(7):402-409.
95. Torres RR, Gomes JL. Measurement of glenohumeral internal rotation in asymptomatic tennis players and swimmers. *Am J Sports Med.* 2009;37(5):1017-1023.
96. Vad VB, Gebeh A, Dines D, Altchek D, Norris B. Hip and shoulder internal rotation range of motion deficits in professional tennis players. *J Sci Med Sport.* 2003;6(1):71-75.
97. Reeser JC, Joy EA, Porucznik CA, Berg RL, Colliver EB, Willick SE. Risk factors for volleyball-related shoulder pain and dysfunction. *PM R.* 2010;2(1):27-36.
98. Saccol MF, Almeida GP, de Souza VL. Anatomical glenohumeral internal rotation deficit and symmetric rotational strength in male and female young beach volleyball players. *J Electromyogr Kinesiol.* 2015.

99. Schwab LM, Blanch P. Humeral torsion and passive shoulder range in elite volleyball players. *Phys Ther Sport*. 2009;10(2):51-56.
100. Wang HK, Cochrane T. Mobility impairment, muscle imbalance, muscle weakness, scapular asymmetry and shoulder injury in elite volleyball athletes. *J Sports Med Phys Fitness*. 2001;41(3):403-410.
101. Witwer A, Sauers E. Clinical measures of shoulder mobility in college water-polo players. *J Sport Rehabil*. 2006;15(1):45.
102. Holt K, Boettcher C, Halaki M, Ginn KA. Humeral torsion and shoulder rotation range of motion parameters in elite swimmers. *J Sci Med Sport*. 2017;20(5):469-474.
103. Brown LP, Niehues SL, Harrah A, Yavorsky P, Hirshman HP. Upper extremity range of motion and isokinetic strength of the internal and external shoulder rotators in major league baseball players. *Am J Sports Med*. 1988;16(6):577-585.
104. Wilk KE, Meister K, Andrews JR. Current concepts in the rehabilitation of the overhead throwing athlete. *Am J Sports Med*. 2002;30(1):136-151.
105. Manske R, Wilk KE, Davies G, Ellenbecker T, Reinold M. Glenohumeral motion deficits: friend or foe? *Int J Sports Phys Ther*. 2013;8(5):537-553.
106. Nutt C, Mirkovic M, Hill R, Ranson C, Cooper S-M. Reference values for glenohumeral joint rotational range of motion in elite tennis players. *Int J Sports Phys Ther*. 2018;13(3):501-510.
107. Chiang CC, Hsu CC, Chiang JY, Chang WC, Tsai JC. Flexibility of internal and external glenohumeral rotation of junior female tennis players and its correlation with performance ranking. *J Phys Ther Sci*. 2016;28(12):3296-3299.
108. Wilk KE, Macrina LC, Arrigo C. Passive range of motion characteristics in the overhead baseball pitcher and their implications for rehabilitation. *Clin Orthop Relat Res*. 2012;470(6):1586-1594.
109. Myers JB, Oyama S, Goerger BM, Rucinski TJ, Blackburn JT, Creighton RA. Influence of humeral torsion on interpretation of posterior shoulder tightness measures in overhead athletes. *Clin J Sport Med*. 2009;19(5):366-371.
110. Shanley E, Thigpen CA, Clark JC, et al. Changes in passive range of motion and development of glenohumeral internal rotation deficit (GIRD) in the professional pitching shoulder between spring training in two consecutive years. *J Shoulder Elbow Surg*. 2012;21(11):1605-1612.
111. Saccol MF, Gracitelli GC, da Silva RT, et al. Shoulder functional ratio in elite junior tennis players. *Phys Ther Sport*. 2010;11(1):8-11.
112. Cools AM, Vanderstukken F, Vereecken F, et al. Eccentric and isometric shoulder rotator cuff strength testing using a hand-held dynamometer: reference values for overhead athletes. *Knee Surg Sports Traumatol Arthrosc*. 2016;24(12):3838-3847.
113. Ellenbecker TS. A total arm strength isokinetic profile of highly skilled tennis players. *Isokinet Exerc Sci*. 1991;1(1):9-21.
114. Chandler TJ, Kibler WB, Stracener EC, Ziegler AK, Pace B. Shoulder strength, power, and endurance in college tennis players. *Am J Sports Med*. 1992;20(4):455-458.
115. Amin NH, Ryan J, Fening SD, Soloff L, Schickendantz MS, Jones M. The relationship between glenohumeral internal rotational deficits, total range of

- motion, and shoulder strength in professional baseball pitchers. *J Am Acad Orthop Surg.* 2015;23(12):789-796.
116. Guney H, Harput G, Colakoglu F, Baltaci G. The effect of glenohumeral internal-rotation deficit on functional rotator-strength ratio in adolescent overhead athletes. *J Sport Rehabil.* 2016;25(1):52-57.
 117. Rhi SY, So WY. Analysis of range of motion and isokinetic strength of internal and external rotation according to humeral retroversion of the dominant shoulder in youth baseball players: a pilot study. *Iran J Public Health.* 2014;43(2):178-184.
 118. Tennis Industry Association. 2016 state of the industry analyzes research on participation, equipment and more. <http://www.tennisindustry.org/cms/index.cfm/news/2016-state-of-the-industry-analyzes-research-on-participation-equipment-and-more/>. Published 2016. Accessed January 25, 2017.
 119. Gaw CE, Chounthirath T, Smith GA. Tennis-related injuries treated in United States emergency departments, 1990 to 2011. *Clin J Sport Med.* 2014;24(3):226-232.
 120. Kovacs MS. Applied physiology of tennis performance. *Br J Sports Med.* 2006;40(5):381-385; discussion 386.
 121. Maquirriain J, Baglione R. Epidemiology of tennis injuries: an eight-year review of Davis Cup retirements. *Eur J Sport Sci.* 2016;16(2):266-270.
 122. Winge S, Jorgensen U, Lassen Nielsen A. Epidemiology of injuries in Danish championship tennis. *Int J Sports Med.* 1989;10(5):368-371.
 123. Colberg RE, Aune KT, Choi AJ, Fleisig GS. Incidence and prevalence of musculoskeletal conditions in collegiate tennis athletes. *J Med Sci Tennis.* 2015;20(3):137-144.
 124. Hutchinson MR, Laprade RF, Burnett QM, 2nd, Moss R, Terpstra J. Injury surveillance at the USTA Boys' Tennis Championships: a 6-yr study. *Med Sci Sports Exerc.* 1995;27(6):826-830.
 125. Safran M. Tennis injuries and strategies for prevention. Gender differences in the American junior elite tennis player. *Med Sci Tennis.* 2000;5(11):41-45.
 126. Jayanthi N, Sallay P, Hunker P, Przybylski M. Skill-level related injuries in recreational competition tennis players. *Med Sci Tennis.* 2005;10(1):12-15.
 127. Abrams GD, Renstrom PA, Safran MR. Epidemiology of musculoskeletal injury in the tennis player. *Br J Sports Med.* 2012;46(7):492-498.
 128. Cassell E, McGrath AC. *Lobbing injury out of tennis: a review of the literature.* Clayton, Australia: Monash University Accident Research Centre; 1999.
 129. Kibler WB, McQueen C, Uhl T. Fitness evaluations and fitness findings in competitive junior tennis players. *Clin Sports Med.* 1988;7(2):403-416.
 130. Neumann DA. *Kinesiology of the musculoskeletal system: foundations for rehabilitation.* 2nd ed. St. Louis, MO: Mosby/Elsevier; 2010.
 131. Renfree KJ, Wright TW. Anatomy and biomechanics of the acromioclavicular and sternoclavicular joints. *Clin Sports Med.* 2003;22(2):219-237.
 132. Terry GC, Chopp TM. Functional anatomy of the shoulder. *J Athl Train.* 2000;35(3):248-255.
 133. Martetschlager F, Warth RJ, Millett PJ. Instability and degenerative arthritis of the sternoclavicular joint: a current concepts review. *Am J Sports Med.* 2014;42(4):999-1007.

134. Ludewig PM, Behrens SA, Meyer SM, Spoden SM, Wilson LA. Three-dimensional clavicular motion during arm elevation: reliability and descriptive data. *J Orthop Sports Phys Ther.* 2004;34(3):140-149.
135. McClure PW, Michener LA, Sennett BJ, Karduna AR. Direct 3-dimensional measurement of scapular kinematics during dynamic movements in vivo. *J Shoulder Elbow Surg.* 2001;10(3):269-277.
136. Saccomanno MF, C DEI, Milano G. Acromioclavicular joint instability: anatomy, biomechanics and evaluation. *Joints.* 2014;2(2):87-92.
137. Klimkiewicz JJ, Williams GR, Sher JS, Karduna A, Des Jardins JD, Iannotti JP. The acromioclavicular capsule as a restraint to posterior translation of the clavicle: A biomechanical analysis. *J Shoulder Elbow Surg.* 1999;8(2):119-124.
138. Williams GR, Jr., Shakil M, Klimkiewicz J, Iannotti JP. Anatomy of the scapulothoracic articulation. *Clin Orthop Relat Res.* 1999(359):237-246.
139. Howell SM, Galinat BJ. The glenoid-labral socket. A constrained articular surface. *Clin Orthop Relat Res.* 1989;243(243):122-125.
140. Soslowsky LJ, Flatow EL, Bigliani LU, Mow VC. Articular geometry of the glenohumeral joint. *Clin Orthop Relat Res.* 1992(285):181-190.
141. Iannotti JP, Gabriel JP, Schneck SL, Evans BG, Misra S. The normal glenohumeral relationships. An anatomical study of one hundred and forty shoulders. *J Bone Joint Surg Am.* 1992;74(4):491-500.
142. Warner JJ, Bowen MK, Deng XH, Hannafin JA, Arnoczky SP, Warren RF. Articular contact patterns of the normal glenohumeral joint. *J Shoulder Elbow Surg.* 1998;7(4):381-388.
143. Soslowsky LJ, Flatow EL, Bigliani LU, Pawluk RJ, Ateshian GA, Mow VC. Quantitation of in situ contact areas at the glenohumeral joint: a biomechanical study. *J Orthop Res.* 1992;10(4):524-534.
144. Moor BK, Kuster R, Osterhoff G, et al. Inclination-dependent changes of the critical shoulder angle significantly influence superior glenohumeral joint stability. *Clin Biomech (Bristol, Avon).* 2016;32:268-273.
145. Gerber C, Snedeker JG, Baumgartner D, Viehofer AF. Supraspinatus tendon load during abduction is dependent on the size of the critical shoulder angle: a biomechanical analysis. *J Orthop Res.* 2014;32(7):952-957.
146. Viehofer AF, Gerber C, Favre P, Bachmann E, Snedeker JG. A larger critical shoulder angle requires more rotator cuff activity to preserve joint stability. *J Orthop Res.* 2016;34(6):961-968.
147. Viehofer AF, Snedeker JG, Baumgartner D, Gerber C. Glenohumeral joint reaction forces increase with critical shoulder angles representative of osteoarthritis: a biomechanical analysis. *J Orthop Res.* 2016;34(6):1047-1052.
148. Gottschalk MB, Ghasem A, Todd D, Daruwalla J, Xerogeanes J, Karas S. Posterior shoulder instability: does glenoid retroversion predict recurrence and contralateral instability? *Arthroscopy.* 2015;31(3):488-493.
149. Hohmann E, Tetsworth K. Glenoid version and inclination are risk factors for anterior shoulder dislocation. *J Shoulder Elbow Surg.* 2015;24(8):1268-1273.
150. Hughes RE, Bryant CR, Hall JM, et al. Glenoid inclination is associated with full-thickness rotator cuff tears. *Clin Orthop Relat Res.* 2003;407:86-91.

151. Sweitzer BA, Thigpen CA, Shanley E, et al. A comparison of glenoid morphology and glenohumeral range of motion between professional baseball pitchers with and without a history of SLAP repair. *Arthroscopy*. 2012;28(9):1206-1213.
152. Moor BK, Bouaicha S, Rothenfluh DA, Sukthankar A, Gerber C. Is there an association between the individual anatomy of the scapula and the development of rotator cuff tears or osteoarthritis of the glenohumeral joint? A radiological study of the critical shoulder angle. *Bone Joint J*. 2013;95-b(7):935-941.
153. Moor BK, Rothlisberger M, Muller DA, et al. Age, trauma and the critical shoulder angle accurately predict supraspinatus tendon tears. *Orthop Traumatol Surg Res*. 2014;100(5):489-494.
154. Moor BK, Wieser K, Slankamenac K, Gerber C, Bouaicha S. Relationship of individual scapular anatomy and degenerative rotator cuff tears. *J Shoulder Elbow Surg*. 2014;23(4):536-541.
155. Symeonides PP, Hatzokos I, Christoforides J, Pournaras J. Humeral head torsion in recurrent anterior dislocation of the shoulder. *J Bone Joint Surg Br*. 1995;77(5):687-690.
156. Tetreault P, Krueger A, Zurakowski D, Gerber C. Glenoid version and rotator cuff tears. *J Orthop Res*. 2004;22(1):202-207.
157. Hill JA, Tkach L, Hendrix RW. A study of glenohumeral orientation in patients with anterior recurrent shoulder dislocations using computerized axial tomography. *Orthop Rev*. 1989;18(1):84-91.
158. Berton A, Gulotta LV, Petrillo S, et al. The effect of humeral version on teres minor muscle moment arm, length, and impingement in reverse shoulder arthroplasty during activities of daily living. *J Shoulder Elbow Surg*. 2015;24(4):578-586.
159. Kominiak P, Gusta A, Ferenc M, Pawlowski Z. [Reconstruction of humeral head retroversion in the treatment of anterior shoulder instability]. *Chir Narzadow Ruchu Ortop Pol*. 2004;69(5):301-303.
160. Gregory TM, Sankey A, Augereau B, et al. Accuracy of glenoid component placement in total shoulder arthroplasty and its effect on clinical and radiological outcome in a retrospective, longitudinal, monocentric open study. *PLoS One*. 2013;8(10):e75791.
161. Kronberg M, Brostrom LA. Rotation osteotomy of the proximal humerus to stabilise the shoulder. Five years' experience. *J Bone Joint Surg Br*. 1995;77(6):924-927.
162. Krahl VE, Evans FG. Humeral torsion in man. *Am J Phys Anthropol*. 1945;3(3):229-253.
163. Larson SG. Humeral torsion and throwing proficiency in early human evolution. *J Hum Evol*. 2015;85:198-205.
164. Churchill SE. Particulate versus integrated evolution of the upper body in late pleistocene humans: a test of two models. *Am J Phys Anthropol*. 1996;100(4):559-583.
165. Edelson G. The development of humeral head retroversion. *J Shoulder Elbow Surg*. 2000;9(4):316-318.
166. Krahl VE. The torsion of the humerus; its localization, cause and duration in man. *Am J Anat*. 1947;80(3):275-319.

167. Evans FG, Krahl VE. The torsion of the humerus: A phylogenetic survey from fish to man. *Am J Anat.* 1945;76(3):303-337.
168. Myers JB, Oyama S, Clarke JP. Ultrasonographic assessment of humeral retroversion in baseball players: a validation study. *Am J Sports Med.* 2012;40(5):1155-1160.
169. Osbahr DC, Cannon DL, Speer KP. Retroversion of the humerus in the throwing shoulder of college baseball pitchers. *Am J Sports Med.* 2002;30(3):347-353.
170. Pieper HG. Humeral torsion in the throwing arm of handball players. *Am J Sports Med.* 1998;26(2):247-253.
171. Whiteley R, Adams R, Ginn K, Nicholson L. Playing level achieved, throwing history, and humeral torsion in Masters baseball players. *J Sports Sci.* 2010;28(11):1223-1232.
172. Yamamoto N, Itoi E, Minagawa H, et al. Why is the humeral retroversion of throwing athletes greater in dominant shoulders than in nondominant shoulders? *J Shoulder Elbow Surg.* 2006;15(5):571-575.
173. Edelson G. Variations in the retroversion of the humeral head. *J Shoulder Elbow Surg.* 1999;8(2):142-145.
174. Kronberg M, Brostrom LA, Soderlund V. Retroversion of the humeral head in the normal shoulder and its relationship to the normal range of motion. *Clin Orthop Relat Res.* 1990;253:113-117.
175. Matsumura N, Ogawa K, Kobayashi S, et al. Morphologic features of humeral head and glenoid version in the normal glenohumeral joint. *J Shoulder Elbow Surg.* 2014;23(11):1724-1730.
176. Perry J. Anatomy and biomechanics of the shoulder in throwing, swimming, gymnastics, and tennis. *Clin Sports Med.* 1983;2(2):247-270.
177. Kibler WB. The role of the scapula in athletic shoulder function. *Am J Sports Med.* 1998;26(2):325-337.
178. Paine R, Voight ML. The role of the scapula. *Int J Sports Phys Ther.* 2013;8(5):617-629.
179. Provencher CM, Makani A, McNeil JW, Pomerantz ML, Golijjanin P, Gross D. The role of the scapula in throwing disorders. *Sports Med Arthrosc.* 2014;22(2):80-87.
180. Voight ML, Thomson BC. The role of the scapula in the rehabilitation of shoulder injuries. *J Athl Train.* 2000;35(3):364-372.
181. Cools AM, Struyf F, De Mey K, Maenhout A, Castelein B, Cagnie B. Rehabilitation of scapular dyskinesia: from the office worker to the elite overhead athlete. *Br J Sports Med.* 2014;48(8):692-697.
182. Kronberg M, Brostrom LA. Humeral head retroversion in patients with unstable humeroscapular joints. *Clin Orthop Relat Res.* 1990(260):207-211.
183. Churchill RS, Brems JJ, Kotschi H. Glenoid size, inclination, and version: an anatomic study. *J Shoulder Elbow Surg.* 2001;10(4):327-332.
184. Kandemir U, Allaire RB, Jolly JT, Debski RE, McMahon PJ. The relationship between the orientation of the glenoid and tears of the rotator cuff. *J Bone Joint Surg Br.* 2006;88(8):1105-1109.

185. Friedman RJ, Hawthorne KB, Genez BM. The use of computerized tomography in the measurement of glenoid version. *J Bone Joint Surg Am.* 1992;74(7):1032-1037.
186. Nyffeler RW, Werner CM, Sukthankar A, Schmid MR, Gerber C. Association of a large lateral extension of the acromion with rotator cuff tears. *J Bone Joint Surg Am.* 2006;88(4):800-805.
187. Bouaicha S, Ehrmann C, Slankamenac K, Regan WD, Moor BK. Comparison of the critical shoulder angle in radiographs and computed tomography. *Skeletal Radiol.* 2014;43(8):1053-1056.
188. Mintzer CM, Waters PM, Brown DJ. Glenoid version in children. *J Pediatr Orthop.* 1996;16(5):563-566.
189. Fealy S, Rodeo SA, Dicarlo EF, O'Brien SJ. The developmental anatomy of the neonatal glenohumeral joint. *J Shoulder Elbow Surg.* 2000;9(3):217-222.
190. Wyland DJ, Pill SG, Shanley E, et al. Bony adaptation of the proximal humerus and glenoid correlate within the throwing shoulder of professional baseball pitchers. *Am J Sports Med.* 2012;40(8):1858-1862.
191. Drakos MC, Barker JU, Osbahr DC, et al. Effective glenoid version in professional baseball players. *Am J Orthop (Belle Mead NJ).* 2010;39(7):340-344.
192. Bishop JL, Kline SK, Aalderink KJ, Zuel R, Bey MJ. Glenoid inclination: in vivo measures in rotator cuff tear patients and associations with superior glenohumeral joint translation. *J Shoulder Elbow Surg.* 2009;18(2):231-236.
193. Blonna D, Giani A, Bellato E, et al. Predominance of the critical shoulder angle in the pathogenesis of degenerative diseases of the shoulder. *J Shoulder Elbow Surg.* 2016;25(8):1328-1336.
194. Konrad GG, Markmiller M, Jolly JT, et al. Decreasing glenoid inclination improves function in shoulders with simulated massive rotator cuff tears. *Clin Biomech (Bristol, Avon).* 2006;21(9):942-949.
195. Peltz CD, Divine G, Drake A, et al. Associations between in-vivo glenohumeral joint motion and morphology. *J Biomech.* 2015;48(12):3252-3257.
196. Spiegl UJ, Horan MP, Smith SW, Ho CP, Millett PJ. The critical shoulder angle is associated with rotator cuff tears and shoulder osteoarthritis and is better assessed with radiographs over MRI. *Knee Surg Sports Traumatol Arthrosc.* 2016;24(7):2244-2251.
197. Tokgoz N, Kanatli U, Voyvoda NK, Gultekin S, Bolukbasi S, Tali ET. The relationship of glenoid and humeral version with supraspinatus tendon tears. *Skeletal Radiol.* 2007;36(6):509-514.
198. Wong AS, Gallo L, Kuhn JE, Carpenter JE, Hughes RE. The effect of glenoid inclination on superior humeral head migration. *J Shoulder Elbow Surg.* 2003;12(4):360-364.
199. Privitera DM, Siegel EJ, Miller LR, Sinz NJ, Higgins LD. Glenoid version and its relationship to glenohumeral instability and labral tears. *J Shoulder Elbow Surg.* 2016;25(7):1056-1063.
200. Cooper DE, Arnoczky SP, O'Brien SJ, Warren RF, DiCarlo E, Allen AA. Anatomy, histology, and vascularity of the glenoid labrum. an anatomical study. *J Bone Joint Surg Am.* 1992;74(1):46-52.

201. Lazarus MD, Sidles JA, Harryman DT, 2nd, Matsen FA, 3rd. Effect of a chondral-labral defect on glenoid concavity and glenohumeral stability. A cadaveric model. *J Bone Joint Surg Am.* 1996;78(1):94-102.
202. Matsen FA, 3rd, Harryman DT, 2nd, Sidles JA. Mechanics of glenohumeral instability. *Clin Sports Med.* 1991;10(4):783-788.
203. Lugo R, Kung P, Ma CB. Shoulder biomechanics. *Eur J Radiol.* 2008;68(1):16-24.
204. Clark J, Sidles JA, Matsen FA. The relationship of the glenohumeral joint capsule to the rotator cuff. *Clin Orthop Relat Res.* 1990(254):29-34.
205. Burkart AC, Debski RE. Anatomy and function of the glenohumeral ligaments in anterior shoulder instability. *Clin Orthop Relat Res.* 2002(400):32-39.
206. O'Brien SJ, Neves MC, Arnoczky SP, et al. The anatomy and histology of the inferior glenohumeral ligament complex of the shoulder. *Am J Sports Med.* 1990;18(5):449-456.
207. Wilk KE, Arrigo CA, Andrews JR. Current concepts: the stabilizing structures of the glenohumeral joint. *J Orthop Sports Phys Ther.* 1997;25(6):364-379.
208. Habermeyer P, Schuller U, Wiedemann E. The intra-articular pressure of the shoulder: an experimental study on the role of the glenoid labrum in stabilizing the joint. *Arthroscopy.* 1992;8(2):166-172.
209. Inokuchi W, Sanderhoff Olsen B, Sojbjerg JO, Sneppen O. The relation between the position of the glenohumeral joint and the intraarticular pressure: an experimental study. *J Shoulder Elbow Surg.* 1997;6(2):144-149.
210. Kumar VP, Balasubramaniam P. The role of atmospheric pressure in stabilising the shoulder. an experimental study. *J Bone Joint Surg Br.* 1985;67(5):719-721.
211. Alexander S, Southgate DF, Bull AM, Wallace AL. The role of negative intraarticular pressure and the long head of biceps tendon on passive stability of the glenohumeral joint. *J Shoulder Elbow Surg.* 2013;22(1):94-101.
212. Hurschler C, Wulker N, Mendila M. The effect of negative intraarticular pressure and rotator cuff force on glenohumeral translation during simulated active elevation. *Clin Biomech (Bristol, Avon).* 2000;15(5):306-314.
213. Murray IR, Goudie EB, Petrigliano FA, Robinson CM. Functional anatomy and biomechanics of shoulder stability in the athlete. *Clin Sports Med.* 2013;32(4):607-624.
214. Huegel J, Williams AA, Soslowsky LJ. Rotator cuff biology and biomechanics: a review of normal and pathological conditions. *Curr Rheumatol Rep.* 2015;17(1):476.
215. Kronberg M, Nemeth G, Brostrom LA. Muscle activity and coordination in the normal shoulder. An electromyographic study. *Clin Orthop Relat Res.* 1990(257):76-85.
216. Hawkes DH, Alizadehkhayat O, Fisher AC, Kemp GJ, Roebuck MM, Frostick SP. Normal shoulder muscular activation and co-ordination during a shoulder elevation task based on activities of daily living: an electromyographic study. *J Orthop Res.* 2012;30(1):53-60.
217. Inman VT, Saunders JB, Abbott LC. Observations on the function of the shoulder joint. *J Bone Joint Surg Am.* 1944;26(1):1-30.

218. Halder AM, Halder CG, Zhao KD, O'Driscoll SW, Morrey BF, An KN. Dynamic inferior stabilizers of the shoulder joint. *Clin Biomech (Bristol, Avon)*. 2001;16(2):138-143.
219. Halder AM, Zhao KD, Odriscoll SW, Morrey BF, An KN. Dynamic contributions to superior shoulder stability. *J Orthop Res*. 2001;19(2):206-212.
220. Su WR, Budoff JE, Luo ZP. Posterosuperior displacement due to rotator cuff tears. *Arthroscopy*. 2011;27(11):1472-1477.
221. Su WR, Budoff JE, Luo ZP. The effect of posterosuperior rotator cuff tears and biceps loading on glenohumeral translation. *Arthroscopy*. 2010;26(5):578-586.
222. Su WR, Budoff JE, Luo ZP. The effect of anterosuperior rotator cuff tears on glenohumeral translation. *Arthroscopy*. 2009;25(3):282-289.
223. Thompson WO, Debski RE, Boardman ND, 3rd, et al. A biomechanical analysis of rotator cuff deficiency in a cadaveric model. *Am J Sports Med*. 1996;24(3):286-292.
224. Parsons IM, Apreleva M, Fu FH, Woo SL. The effect of rotator cuff tears on reaction forces at the glenohumeral joint. *J Orthop Res*. 2002;20(3):439-446.
225. Apreleva M, Parsons IMt, Warner JJ, Fu FH, Woo SL. Experimental investigation of reaction forces at the glenohumeral joint during active abduction. *J Shoulder Elbow Surg*. 2000;9(5):409-417.
226. Liu J, Hughes RE, Smutz WP, Niebur G, Nan-An K. Roles of deltoid and rotator cuff muscles in shoulder elevation. *Clin Biomech (Bristol, Avon)*. 1997;12(1):32-38.
227. Howell SM, Imobersteg AM, Seger DH, Marone PJ. Clarification of the role of the supraspinatus muscle in shoulder function. *J Bone Joint Surg Am*. 1986;68(3):398-404.
228. Ackland DC, Pandy MG. Moment arms of the shoulder muscles during axial rotation. *J Orthop Res*. 2011;29(5):658-667.
229. de Castro MP, Ribeiro DC, de Camargo Forte F, de Toledo JM, Costa Krug R, Fagundes Loss J. Estimated force and moment of shoulder external rotation muscles: differences between transverse and sagittal planes. *J Appl Biomech*. 2012;28(6):701-707.
230. Langenderfer JE, Carpenter JE, Johnson ME, An KN, Hughes RE. A probabilistic model of glenohumeral external rotation strength for healthy normals and rotator cuff tear cases. *Ann Biomed Eng*. 2006;34(3):465-476.
231. Langenderfer JE, Patthanacharonphon C, Carpenter JE, Hughes RE. Variability in isometric force and moment generating capacity of glenohumeral external rotator muscles. *Clin Biomech (Bristol, Avon)*. 2006;21(7):701-709.
232. Hallström E, Kärholm J. Shoulder rhythm in patients with impingement and in controls: Dynamic RSA during active and passive abduction. *Acta Orthop*. 2009;80(4):456-464.
233. Poppen NK, Walker PS. Normal and abnormal motion of the shoulder. *J Bone Joint Surg Am*. 1976;58(2):195-201.
234. Scibek JS, Carcia CR. Assessment of scapulohumeral rhythm for scapular plane shoulder elevation using a modified digital inclinometer. *World J Orthop*. 2012;3(6):87-94.

235. Konda S, Yanai T, Sakurai S. Configuration of the shoulder complex during the arm-cocking phase in baseball pitching. *Am J Sports Med.* 2015;43(10):2445-2451.
236. Crosbie J, Kilbreath SL, Hollmann L, York S. Scapulohumeral rhythm and associated spinal motion. *Clin Biomech (Bristol, Avon).* 2008;23(2):184-192.
237. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part III: The SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. *Arthroscopy.* 2003;19(6):641-661.
238. Kibler WB, Ludewig PM, McClure PW, Michener LA, Bak K, Sciascia AD. Clinical implications of scapular dyskinesis in shoulder injury: the 2013 consensus statement from the 'Scapular Summit'. *Br J Sports Med.* 2013;47(14):877-885.
239. Pink MM, Perry J. Biomechanics. In: Jobe FW, ed. *Operative Techniques in Upper Extremity Sports Injuries.* St. Louis: Mosby; 1996:109-123.
240. Kibler WB, Sciascia A. Current concepts: scapular dyskinesis. *Br J Sports Med.* 2010;44(5):300-305.
241. Braman JP, Engel SC, Laprade RF, Ludewig PM. In vivo assessment of scapulohumeral rhythm during unconstrained overhead reaching in asymptomatic subjects. *J Shoulder Elbow Surg.* 2009;18(6):960-967.
242. Kibler WB, Uhl TL, Maddux JW, Brooks PV, Zeller B, McMullen J. Qualitative clinical evaluation of scapular dysfunction: a reliability study. *J Shoulder Elbow Surg.* 2002;11(6):550-556.
243. McClure P, Tate AR, Kareha S, Irwin D, Zlupko E. A clinical method for identifying scapular dyskinesis, part 1: reliability. *J Athl Train.* 2009;44(2):160-164.
244. Tate AR, McClure P, Kareha S, Irwin D, Barbe MF. A clinical method for identifying scapular dyskinesis, part 2: validity. *J Athl Train.* 2009;44(2):165-173.
245. Uhl TL, Kibler WB, Gecewich B, Tripp BL. Evaluation of clinical assessment methods for scapular dyskinesis. *Arthroscopy.* 2009;25(11):1240-1248.
246. Ratcliffe E, Pickering S, McLean S, Lewis J. Is there a relationship between subacromial impingement syndrome and scapular orientation? a systematic review. *Br J Sports Med.* 2014;48(16):1251-1256.
247. Thomas SJ, Swanik CB, Kaminski TW, Higginson JS, Swanik KA, Nazarian LN. Assessment of subacromial space and its relationship with scapular upward rotation in college baseball players. *J Sport Rehabil.* 2013;22(3):216-223.
248. Myers JB, Oyama S, Hibberd EE. Scapular dysfunction in high school baseball players sustaining throwing-related upper extremity injury: a prospective study. *J Shoulder Elbow Surg.* 2013;22(9):1154-1159.
249. Seitz AL, McClelland RI, Jones WJ, Jean RA, Kardouni JR. A comparison of change in 3D scapular kinematics with maximal contractions and force production with scapular muscle tests between asymptomatic overhead athletes with and without scapular dyskinesis. *Int J Sports Phys Ther.* 2015;10(3):309-318.
250. Struyf F, Nijs J, Meeus M, et al. Does scapular positioning predict shoulder pain in recreational overhead athletes? *Int J Sports Med.* 2014;35(1):75-82.
251. Hannah DC, Scibek JS, Carcia CR. Strength profiles in healthy individuals with and without scapular dyskinesis. *Int J Sports Phys Ther.* 2017;12(3):305-313.

252. McQuade KJ, Borstad J, de Oliveira AS. Critical and theoretical perspective on scapular stabilization: what does it really mean, and are we on the right track? *Phys Ther.* 2016;96(8):1162-1169.
253. Elliott B. Biomechanics and tennis. *Br J Sports Med.* 2006;40(5):392-396.
254. Fleisig GS, Barrentine SW, Escamilla RF, Andrews JR. Biomechanics of overhand throwing with implications for injuries. *Sports Med.* 1996;21(6):421-437.
255. Martin C, Bideau B, Ropars M, Delamarche P, Kulpa R. Upper limb joint kinetic analysis during tennis serve: assessment of competitive level on efficiency and injury risks. *Scand J Med Sci Sports.* 2014;24(4):700-707.
256. Pearson OM, Lieberman DE. The aging of Wolff's "law": ontogeny and responses to mechanical loading in cortical bone. *Am J Phys Anthropol.* 2004;Suppl 39:63-99.
257. Teichtahl AJ, Wluka AE, Wijethilake P, Wang Y, Ghasem-Zadeh A, Cicuttini FM. Wolff's law in action: a mechanism for early knee osteoarthritis. *Arthritis Res Ther.* 2015;17:207.
258. Duncan RL, Turner CH. Mechanotransduction and the functional response of bone to mechanical strain. *Calcif Tissue Int.* 1995;57(5):344-358.
259. Chen JH, Liu C, You L, Simmons CA. Boning up on Wolff's Law: mechanical regulation of the cells that make and maintain bone. *J Biomech.* 2010;43(1):108-118.
260. Robling AG, Castillo AB, Turner CH. Biomechanical and molecular regulation of bone remodeling. *Annu Rev Biomed Eng.* 2006;8:455-498.
261. Bass SL, Saxon L, Daly RM, et al. The effect of mechanical loading on the size and shape of bone in pre-, peri-, and postpubertal girls: a study in tennis players. *J Bone Miner Res.* 2002;17(12):2274-2280.
262. Bogenschutz ED, Smith HD, Warden SJ. Midhumerus adaptation in fast-pitch softballers and the effect of throwing mechanics. *Med Sci Sports Exerc.* 2011;43(9):1698-1706.
263. Calbet JA, Moysi JS, Dorado C, Rodriguez LP. Bone mineral content and density in professional tennis players. *Calcif Tissue Int.* 1998;62(6):491-496.
264. Dalen N, Laftman P, Ohlson H, Stromberg L. The effect of athletic activity on the bone mass in human diaphyseal bone. *Orthopedics.* 1985;8(9):1139-1141.
265. Daly RM, Saxon L, Turner CH, Robling AG, Bass SL. The relationship between muscle size and bone geometry during growth and in response to exercise. *Bone.* 2004;34(2):281-287.
266. Ducher G, Bass SL, Saxon L, Daly RM. Effects of repetitive loading on the growth-induced changes in bone mass and cortical bone geometry: a 12-month study in pre/peri- and postmenarcheal tennis players. *J Bone Miner Res.* 2011;26(6):1321-1329.
267. Ducher G, Courteix D, Meme S, Magni C, Viala JF, Benhamou CL. Bone geometry in response to long-term tennis playing and its relationship with muscle volume: a quantitative magnetic resonance imaging study in tennis players. *Bone.* 2005;37(4):457-466.

268. Ducher G, Daly RM, Bass SL. Effects of repetitive loading on bone mass and geometry in young male tennis players: a quantitative study using MRI. *J Bone Miner Res.* 2009;24(10):1686-1692.
269. Ducher G, Jaffre C, Arlettaz A, Benhamou CL, Courteix D. Effects of long-term tennis playing on the muscle-bone relationship in the dominant and nondominant forearms. *Can J Appl Physiol.* 2005;30(1):3-17.
270. Ducher G, Prouteau S, Courteix D, Benhamou CL. Cortical and trabecular bone at the forearm show different adaptation patterns in response to tennis playing. *J Clin Densitom.* 2004;7(4):399-405.
271. Haapasalo H, Kannus P, Sievanen H, et al. Effect of long-term unilateral activity on bone mineral density of female junior tennis players. *J Bone Miner Res.* 1998;13(2):310-319.
272. Haapasalo H, Sievanen H, Kannus P, Heinonen A, Oja P, Vuori I. Dimensions and estimated mechanical characteristics of the humerus after long-term tennis loading. *J Bone Miner Res.* 1996;11(6):864-872.
273. Huddleston AL, Rockwell D, Kulund DN, Harrison RB. Bone mass in lifetime tennis athletes. *JAMA.* 1980;244(10):1107-1109.
274. Ireland A, Maden-Wilkinson T, Ganse B, Degens H, Rittweger J. Effects of age and starting age upon side asymmetry in the arms of veteran tennis players: a cross-sectional study. *Osteoporos Int.* 2014;25(4):1389-1400.
275. Ireland A, Maden-Wilkinson T, McPhee J, et al. Upper limb muscle-bone asymmetries and bone adaptation in elite youth tennis players. *Med Sci Sports Exerc.* 2013;45(9):1749-1758.
276. Kannus P, Haapasalo H, Sankelo M, et al. Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. *Ann Intern Med.* 1995;123(1):27-31.
277. Kannus P, Haapasalo H, Sievanen H, Oja P, Vuori I. The site-specific effects of long-term unilateral activity on bone mineral density and content. *Bone.* 1994;15(3):279-284.
278. Kontulainen S, Sievanen H, Kannus P, Pasanen M, Vuori I. Effect of long-term impact-loading on mass, size, and estimated strength of humerus and radius of female racquet-sports players: a peripheral quantitative computed tomography study between young and old starters and controls. *J Bone Miner Res.* 2003;18(2):352-359.
279. Krahl H, Michaelis U, Pieper HG, Quack G, Montag M. Stimulation of bone growth through sports. A radiologic investigation of the upper extremities in professional tennis players. *Am J Sports Med.* 1994;22(6):751-757.
280. Sanchis-Moysi J, Dorado C, Olmedillas H, Serrano-Sanchez JA, Calbet JA. Bone and lean mass inter-arm asymmetries in young male tennis players depend on training frequency. *Eur J Appl Physiol.* 2010;110(1):83-90.
281. Sanchis-Moysi J, Dorado C, Olmedillas H, Serrano-Sanchez JA, Calbet JA. Bone mass in prepubertal tennis players. *Int J Sports Med.* 2010;31(6):416-420.
282. Sanchis-Moysi J, Dorado C, Vicente-Rodriguez G, Milutinovic L, Garces GL, Calbet JA. Inter-arm asymmetry in bone mineral content and bone area in postmenopausal recreational tennis players. *Maturitas.* 2004;48(3):289-298.

283. Warden SJ, Bogenschutz ED, Smith HD, Gutierrez AR. Throwing induces substantial torsional adaptation within the midshaft humerus of male baseball players. *Bone*. 2009;45(5):931-941.
284. Schiessl H, Frost HM, Jee WS. Estrogen and bone-muscle strength and mass relationships. *Bone*. 1998;22(1):1-6.
285. Rauch F, Bailey DA, Baxter-Jones A, Mirwald R, Faulkner R. The 'muscle-bone unit' during the pubertal growth spurt. *Bone*. 2004;34(5):771-775.
286. Schoenau E, Neu CM, Beck B, Manz F, Rauch F. Bone mineral content per muscle cross-sectional area as an index of the functional muscle-bone unit. *J Bone Miner Res*. 2002;17(6):1095-1101.
287. Werner SL, Guido JA, McNeice RP, Richardson JL, Delude NA, Stewart GW. Biomechanics of youth windmill softball pitching. *Am J Sports Med*. 2005;33(4):552-560.
288. Chen FS, Diaz VA, Loebenberg M, Rosen JE. Shoulder and elbow injuries in the skeletally immature athlete. *J Am Acad Orthop Surg*. 2005;13(3):172-185.
289. Itami Y, Mihata T, Shibano K, Sugamoto K, Neo M. Site and severity of the increased humeral retroversion in Symptomatic baseball players: a 3-dimensional computed tomographic analysis. *Am J Sports Med*. 2016;44(7):1825-1831.
290. Kurokawa D, Yamamoto N, Ishikawa H, et al. Differences in humeral retroversion in dominant and nondominant sides of young baseball players. *J Shoulder Elbow Surg*. 2017;26(6):1083-1087.
291. Mihata T, Takeda A, Kawakami T, et al. Isolated glenohumeral range of motion, excluding side-to-side difference in humeral retroversion, in asymptomatic high-school baseball players. *Knee Surg Sports Traumatol Arthrosc*. 2016;24(6):1911-1917.
292. Nakase C, Mihata T, Itami Y, Takeda A, Neo M. Relationship between humeral retroversion and length of baseball career before the age of 16 years. *Am J Sports Med*. 2016;44(9):2220-2224.
293. Pritchett JW. Growth plate activity in the upper extremity. *Clin Orthop Relat Res*. 1991(268):235-242.
294. Tokish JM, Curtin MS, Young-Kyu K, Hawkins RJ, Torry MR. Glenohumeral internal rotation deficit in the asymptomatic professional pitcher and its relationship to humeral retroversion. *J Sports Sci Med*. 2008;7(1):78-83.
295. Thomas SJ, Swanik CB, Kaminski TW, et al. Humeral retroversion and its association with posterior capsule thickness in collegiate baseball players. *J Shoulder Elbow Surg*. 2012;21(7):910-916.
296. Yamamoto A, Kobayashi T, Tajika T, et al. Relationship between humeral torsion angle and shoulder range of motion among juvenile baseball players. *Sports Orthop Traumatol*. 2015;31(3):182-187.
297. Meister K. Injuries to the shoulder in the throwing athlete. Part one: biomechanics/pathophysiology/classification of injury. *Am J Sports Med*. 2000;28(2):265-275.
298. Bailey LB, Shanley E, Hawkins R, et al. Mechanisms of shoulder range of motion deficits in asymptomatic baseball players. *Am J Sports Med*. 2015;43(11):2783-2793.

299. Burkhart SS, Morgan CD. The peel-back mechanism: its role in producing and extending posterior type II SLAP lesions and its effect on SLAP repair rehabilitation. *Arthroscopy*. 1998;14(6):637-640.
300. Fleisig GS, Andrews JR, Dillman CJ, Escamilla RF. Kinetics of baseball pitching with implications about injury mechanisms. *Am J Sports Med*. 1995;23(2):233-239.
301. Chu SK, Jayabalan P, Kibler WB, Press J. The kinetic chain revisited: new concepts on throwing mechanics and injury. *PM R*. 2016;8(3 Suppl):S69-77.
302. Kibler WB, Sciascia A. Kinetic chain contributions to elbow function and dysfunction in sports. *Clin Sports Med*. 2004;23(4):545-552, viii.
303. Dines JS, Frank JB, Akerman M, Yocum LA. Glenohumeral internal rotation deficits in baseball players with ulnar collateral ligament insufficiency. *Am J Sports Med*. 2009;37(3):566-570.
304. Sabick MB, Torry MR, Lawton RL, Hawkins RJ. Valgus torque in youth baseball pitchers: a biomechanical study. *J Shoulder Elbow Surg*. 2004;13(3):349-355.
305. De Wilde LF, Berghs BM, VandeVyver F, Schepens A, Verdonk RC. Glenohumeral relationship in the transverse plane of the body. *J Shoulder Elbow Surg*. 2003;12(3):260-267.
306. Werner SL, Fleisig GS, Dillman CJ, Andrews JR. Biomechanics of the elbow during baseball pitching. *J Orthop Sports Phys Ther*. 1993;17(6):274-278.
307. Fleisig G, Nicholls R, Elliott B, Escamilla R. Kinematics used by world class tennis players to produce high-velocity serves. *Sports Biomech*. 2003;2(1):51-64.
308. Konda S, Yanai T, Sakurai S. Scapular rotation to attain the peak shoulder external rotation in tennis serve. *Med Sci Sports Exerc*. 2010;42(9):1745-1753.
309. Wilk KE, Arrigo C. Current concepts in the rehabilitation of the athletic shoulder. *J Orthop Sports Phys Ther*. 1993;18(1):365-378.
310. Jobe FW, Kvitne RS, Giangarra CE. Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. *Orthop Rev*. 1989;18(9):963-975.
311. Jobe FW, Giangarra CE, Kvitne RS, Glousman RE. Anterior capsulolabral reconstruction of the shoulder in athletes in overhand sports. *Am J Sports Med*. 1991;19(5):428-434.
312. Kvitne RS, Jobe FW. The diagnosis and treatment of anterior instability in the throwing athlete. *Clin Orthop Relat Res*. 1993(291):107-123.
313. Rubenstein DL, Jobe FW, Glousman RE, Kvitne RS, Pink M, Giangarra CE. Anterior capsulolabral reconstruction of the shoulder in athletes. *J Shoulder Elbow Surg*. 1992;1(5):229-237.
314. Mazoue CG, Andrews JR. Injuries to the shoulder in athletes. *South Med J*. 2004;97(8):748-755.
315. Jobe CM. Posterior superior glenoid impingement: expanded spectrum. *Arthroscopy*. 1995;11(5):530-536.
316. Walch G, Boileau P, Noel E, Donell ST. Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: an arthroscopic study. *J Shoulder Elbow Surg*. 1992;1(5):238-245.

317. Ellenbecker TS, Mattalino AJ, Elam E, Caplinger R. Quantification of anterior translation of the humeral head in the throwing shoulder. Manual assessment versus stress radiography. *Am J Sports Med.* 2000;28(2):161-167.
318. Borsa PA, Scibek JS, Jacobson JA, Meister K. Sonographic stress measurement of glenohumeral joint laxity in collegiate swimmers and age-matched controls. *Am J Sports Med.* 2005;33(7):1077-1084.
319. Borsa PA, Wilk KE, Jacobson JA, et al. Correlation of range of motion and glenohumeral translation in professional baseball pitchers. *Am J Sports Med.* 2005;33(9):1392-1399.
320. Crawford SD, Sauers EL. Glenohumeral joint laxity and stiffness in the functional throwing position of high school baseball pitchers. *J Athl Train.* 2006;41(1):52-59.
321. Borsa PA, Dover GC, Wilk KE, Reinold MM. Glenohumeral range of motion and stiffness in professional baseball pitchers. *Med Sci Sports Exerc.* 2006;38(1):21-26.
322. Lintner SA, Levy A, Kenter K, Speer KP. Glenohumeral translation in the asymptomatic athlete's shoulder and its relationship to other clinically measurable anthropometric variables. *Am J Sports Med.* 1996;24(6):716-720.
323. Levy AS, Lintner S, Kenter K, Speer KP. Intra- and interobserver reproducibility of the shoulder laxity examination. *Am J Sports Med.* 1999;27(4):460-463.
324. Sethi PM, Tibone JE, Lee TQ. Quantitative assessment of glenohumeral translation in baseball players: a comparison of pitchers versus nonpitching athletes. *Am J Sports Med.* 2004;32(7):1711-1715.
325. Sonnery-Cottet B, Edwards TB, Noel E, Walch G. Results of arthroscopic treatment of posterosuperior glenoid impingement in tennis players. *Am J Sports Med.* 2002;30(2):227-232.
326. Halbrecht JL, Tirman P, Atkin D. Internal impingement of the shoulder: comparison of findings between the throwing and nonthrowing shoulders of college baseball players. *Arthroscopy.* 1999;15(3):253-258.
327. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part II: evaluation and treatment of SLAP lesions in throwers. *Arthroscopy.* 2003;19(5):531-539.
328. Warner JJ, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Patterns of flexibility, laxity, and strength in normal shoulders and shoulders with instability and impingement. *Am J Sports Med.* 1990;18(4):366-375.
329. Mihata T, Lee Y, McGarry MH, Abe M, Lee TQ. Excessive humeral external rotation results in increased shoulder laxity. *Am J Sports Med.* 2004;32(5):1278-1285.
330. Chambers L, Altchek DW. Microinstability and internal impingement in overhead athletes. *Clin Sports Med.* 2013;32(4):697-707.
331. Cools AM, Declercq G, Cagnie B, Cambier D, Witvrouw E. Internal impingement in the tennis player: rehabilitation guidelines. *Br J Sports Med.* 2008;42(3):165-171.
332. Reinold MM, Curtis AS. Microinstability of the shoulder in the overhead athlete. *Int J Sports Phys Ther.* 2013;8(5):601-616.

333. Merolla G, Bhat MG, Porcellini G. Atraumatic shoulder instability: pathophysiology clinical assessment and practical management. *Musculoskelet Regen.* 2015;1.
334. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Glenohumeral range of motion deficits and posterior shoulder tightness in throwers with pathologic internal impingement. *Am J Sports Med.* 2006;34(3):385-391.
335. Wilk KE, Macrina LC, Fleisig GS, et al. Deficits in glenohumeral passive range of motion increase risk of elbow injury in professional baseball pitchers: a prospective study. *Am J Sports Med.* 2014;42(9):2075-2081.
336. Kibler WB, Sciascia A, Thomas SJ. Glenohumeral internal rotation deficit: pathogenesis and response to acute throwing. *Sports Med Arthrosc.* 2012;20(1):34-38.
337. Garrison JC, Cole MA, Conway JE, Macko MJ, Thigpen C, Shanley E. Shoulder range of motion deficits in baseball players with an ulnar collateral ligament tear. *Am J Sports Med.* 2012;40(11):2597-2603.
338. Tyler TF, Nicholas SJ, Roy T, Gleim GW. Quantification of posterior capsule tightness and motion loss in patients with shoulder impingement. *Am J Sports Med.* 2000;28(5):668-673.
339. Thomas SJ, Swanik CB, Higginson JS, et al. A bilateral comparison of posterior capsule thickness and its correlation with glenohumeral range of motion and scapular upward rotation in collegiate baseball players. *J Shoulder Elbow Surg.* 2011;20(5):708-716.
340. Hung CJ, Hsieh CL, Yang PL, Lin JJ. Relationships between posterior shoulder muscle stiffness and rotation in patients with stiff shoulder. *J Rehabil Med.* 2010;42(3):216-220.
341. Reinold MM, Wilk KE, Macrina LC, et al. Changes in shoulder and elbow passive range of motion after pitching in professional baseball players. *Am J Sports Med.* 2008;36(3):523-527.
342. Kibler WB, Sciascia A, Moore S. An acute throwing episode decreases shoulder internal rotation. *Clin Orthop Relat Res.* 2012;470(6):1545-1551.
343. Martin C, Kulpa R, Ezanno F, Delamarche P, Bideau B. Influence of playing a prolonged tennis satch on shoulder internal range of motion. *Am J Sports Med.* 2016;44(8):2147-2151.
344. Takenaga T, Sugimoto K, Goto H, et al. Posterior shoulder capsules are thicker and stiffer in the throwing shoulders of healthy college baseball players: a quantitative assessment using shear-wave ultrasound elastography. *Am J Sports Med.* 2015;43(12):2935-2942.
345. Ishigaki T, Ishida T, Samukawa M, et al. Does restriction of glenohumeral horizontal adduction reflect posterior capsule thickening of the throwing shoulder? *J Phys Ther Sci.* 2015;27(5):1299-1302.
346. Harryman DT, 2nd, Sidles JA, Clark JM, McQuade KJ, Gibb TD, Matsen FA, 3rd. Translation of the humeral head on the glenoid with passive glenohumeral motion. *J Bone Joint Surg Am.* 1990;72(9):1334-1343.
347. Grossman MG, Tibone JE, McGarry MH, Schneider DJ, Veneziani S, Lee TQ. A cadaveric model of the throwing shoulder: a possible etiology of superior labrum anterior-to-posterior lesions. *J Bone Joint Surg Am.* 2005;87(4):824-831.

348. Huffman GR, Tibone JE, McGarry MH, Phipps BM, Lee YS, Lee TQ. Path of glenohumeral articulation throughout the rotational range of motion in a thrower's shoulder model. *Am J Sports Med.* 2006;34(10):1662-1669.
349. Mihata T, Gates J, McGarry MH, Neo M, Lee TQ. Effect of posterior shoulder tightness on internal impingement in a cadaveric model of throwing. *Knee Surg Sports Traumatol Arthrosc.* 2015;23(2):548-554.
350. DeAngelis JP, Hertz B, Wexler MT, et al. Posterior capsular plication constrains the glenohumeral joint by drawing the humeral head closer to the glenoid and resisting abduction. *Orthop J Sports Med.* 2015;3(8):2325967115599347.
351. Gates JJ, Gupta A, McGarry MH, Tibone JE, Lee TQ. The effect of glenohumeral internal rotation deficit due to posterior capsular contracture on passive glenohumeral joint motion. *Am J Sports Med.* 2012;40(12):2794-2800.
352. Clabbers KM, Kelly JD, Bader D, et al. Effect of posterior capsule tightness on glenohumeral translation in the late-cocking phase of pitching. *J Sport Rehabil.* 2007;16(1):41-49.
353. Proske U, Morgan DL. Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *J Physiol.* 2001;537(Pt 2):333-345.
354. Whitehead NP, Weerakkody NS, Gregory JE, Morgan DL, Proske U. Changes in passive tension of muscle in humans and animals after eccentric exercise. *J Physiol.* 2001;533(Pt 2):593-604.
355. Proske U, Morgan DL. Do cross-bridges contribute to the tension during stretch of passive muscle? *J Muscle Res Cell Motil.* 1999;20(5-6):433-442.
356. Hjelm N, Werner S, Renstrom P. Injury risk factors in junior tennis players: a prospective 2-year study. *Scand J Med Sci Sports.* 2012;22(1):40-48.
357. Roetert EP, Ellenbecker TS, Brown SW. Shoulder internal and external rotation range of motion in nationally ranked junior tennis players: a longitudinal analysis. *J Strength Cond Res.* 2000;14(2):140-143.
358. Gillet B, Begon M, Sevrez V, Berger-Vachon C, Rogowski I. Adaptive alterations in shoulder range of motion and strength in young tennis players. *J Athl Train.* 2017;52(2):137-144.
359. Laudner KG, Moline M, Meister K. Lack of a relationship between glenohumeral external-rotation strength and posterior shoulder tightness in baseball players. *J Sport Rehabil.* 2012;21(1):12-17.
360. Richards RR, An KN, Bigliani LU, et al. A standardized method for the assessment of shoulder function. *J Shoulder Elbow Surg.* 1994;3(6):347-352.
361. Cools AM, De Wilde L, Van Tongel A, Ceysens C, Ryckewaert R, Cambier DC. Measuring shoulder external and internal rotation strength and range of motion: comprehensive intra-rater and inter-rater reliability study of several testing protocols. *J Shoulder Elbow Surg.* 2014;23(10):1454-1461.
362. Kolber MJ, Hanney WJ. The reliability and concurrent validity of shoulder mobility measurements using a digital inclinometer and goniometer: a technical report. *Int J Sports Phys Ther.* 2012;7(3):306-313.
363. Stark T, Walker B, Phillips JK, Fejer R, Beck R. Hand-held dynamometry correlation with the gold standard isokinetic dynamometry: a systematic review. *PM R.* 2011;3(5):472-479.

364. Roy JS, MacDermid JC, Orton B, et al. The concurrent validity of a hand-held versus a stationary dynamometer in testing isometric shoulder strength. *J Hand Ther.* 2009;22(4):320-326.
365. Sullivan SJ, Chesley A, Hebert G, McFaul S, Scullion D. The validity and reliability of hand-held dynamometry in assessing isometric external rotator performance. *J Orthop Sports Phys Ther.* 1988;10(6):213-217.
366. Hebert LJ, Maltais DB, Lepage C, Saulnier J, Crete M, Perron M. Isometric muscle strength in youth assessed by hand-held dynamometry: a feasibility, reliability, and validity study. *Pediatr Phys Ther.* 2011;23(3):289-299.
367. Holt KL, Raper DP, Boettcher CE, Waddington GS, Drew MK. Hand-held dynamometry strength measures for internal and external rotation demonstrate superior reliability, lower minimal detectable change and higher correlation to isokinetic dynamometry than externally-fixed dynamometry of the shoulder. *Phys Ther Sport.* 2016;21:75-81.
368. Hayes K, Walton JR, Szomor ZL, Murrell GA. Reliability of 3 methods for assessing shoulder strength. *J Shoulder Elbow Surg.* 2002;11(1):33-39.
369. Kolber MJ, Beekhuizen K, Cheng MS, Fiebert IM. The reliability of hand-held dynamometry in measuring isometric strength of the shoulder internal and external rotator musculature using a stabilization device. *Physiother Theory Pract.* 2007;23(2):119-124.
370. Riemann BL, Davies GJ, Ludwig L, Gardenhour H. Hand-held dynamometer testing of the internal and external rotator musculature based on selected positions to establish normative data and unilateral ratios. *J Shoulder Elbow Surg.* 2010;19(8):1175-1183.
371. Donatelli R, Ellenbecker TS, Ekedahl SR, Wilkes JS, Kocher K, Adam J. Assessment of shoulder strength in professional baseball pitchers. *J Orthop Sports Phys Ther.* 2000;30(9):544-551.
372. Bohannon RW. Make tests and break tests of elbow flexor muscle strength. *Phys Ther.* 1988;68(2):193-194.
373. Feuerherd R, Sutherlin MA, Hart JM, Saliba SA. Reliability of and the relationship between ultrasound measurement and three clinical assessments of humeral torsion. *Int J Sports Phys Ther.* 2014;9(7):938-947.
374. Hannah DC, Scibek JS, Carcia CR, Phelps AL. Reliability and validity of a 1-person technique to measure humeral torsion using ultrasound. *J Athl Train.* 2018;53(6):590-596.
375. Reuther KE, Sheridan S, Thomas SJ. Differentiation of bony and soft-tissue adaptations of the shoulder in professional baseball pitchers. *J Shoulder Elbow Surg.* 2018;27(8):1491-1496.
376. Hernigou P, Duparc F, Hernigou A. Determining humeral retroversion with computed tomography. *J Bone Joint Surg Am.* 2002;84-a(10):1753-1762.
377. Dashottar A, Borstad JD. Validity of measuring humeral torsion using palpation of bicipital tuberosities. *Physiother Theory Pract.* 2013;29(1):67-74.
378. Moreno-Perez V, Elvira J, Fernandez-Fernandez J, Vera-Garcia FJ. A comparative study of passive shoulder rotation range of motion, isometric rotation strength and serve speed between elite tennis players with and without history of shoulder pain. *Int J Sports Phys Ther.* 2018;13(1):39-49.

379. Gillet B, Begon M, Diger M, Berger-Vachon C, Rogowski I. Shoulder range of motion and strength in young competitive tennis players with and without history of shoulder problems. *Phys Ther Sport*. 2018;31:22-28.

Appendix 1

Data Collection Form

TESTER: _____ SUBJECT #: _____ ORDER: _____

BIRTHDATE: _____ AGE: _____ GRADE: _____ SEX: _____

HEIGHT: _____ WEIGHT: _____ HAND: _____

SERVE: _____ GROUND: _____ BACKHAND: _____

INCLUSION/EXCLUSION

- Are you a current member of an area high school team or tennis club/association, and tennis must be your primary sport, **OR** intercollegiate-level tennis players will be current members of an area college or university sponsored tennis team competing in the National Collegiate Athletic Association or National Association of Intercollegiate Athletics?
Y N
- Have you had an injury to the shoulder within the past 6 weeks?
Y N
- Have you had any recent shoulder or elbow surgery within the past 6 months?
Y N
- Do you currently have any elbow or shoulder pain that limits your ability to play?
Y N
- Have you been diagnosed with any neurological diseases that could affect muscle strength or motion of your arms
Y N

Notes: _____

PLAYING HISTORY

At what age did you begin playing tennis? _____

How many months out of the year do you play tennis? _____

Have you ever played any other overhead sports (ex., baseball or softball) as part of an organized team/association? If so, how many years did you play and provide an age range? _____

In the past two years, have you played any other overhead sports as part of an organized team/association?

If so, how many months out of the year did you play? _____

Does your shoulder feel unstable (as if it is going to dislocate)?	Yes	No
How unstable is your shoulder? (mark line)		
<p style="text-align: center;"> 0 _____ 10 Very stable Very <u>un</u>stable </p>		

Circle the number in the box that indicates your ability to do the following activities:		
0 = unable to do; 1 = very difficult to do; 2 = somewhat difficult; 3 = not difficult		
Activity	Right Arm	Left Arm
1. Put on a coat	0 1 2 3	0 1 2 3
2. Sleep on your painful or affected side	0 1 2 3	0 1 2 3
3. Wash back/do up bra in back	0 1 2 3	0 1 2 3
4. Manage toileting	0 1 2 3	0 1 2 3
5. Comb hair	0 1 2 3	0 1 2 3
6. Reach a high shelf	0 1 2 3	0 1 2 3
7. Lift 10 lbs. above shoulder	0 1 2 3	0 1 2 3
8. Throw a ball overhead	0 1 2 3	0 1 2 3
9. Do usual work – List:	0 1 2 3	0 1 2 3
10. Do usual sport – List:	0 1 2 3	0 1 2 3

Richards RR, An K-N, Bigliani LU, et al. A standardized method for the assessment of shoulder function. *J Shoulder Elbow Surg.* 1994;3:347–352.

RANGE OF MOTION				
Total shoulder motion goniometer preferred				
	<i>Right</i>		<i>Left</i>	
	Active	Passive	Active	Passive
Forward elevation (maximum arm-trunk angle)				
External rotation (arm comfortably at side)				
External rotation (arm at 90 degree abduction)				
Internal rotation (arm at 90 degree abduction)				
Cross-body adduction (antecubital fossa to opposite acromion)				
Abduction				

SIGNS									
0 = none; 1 = mild; 2 = moderate; 3 = severe									
		Right			Left				
Supraspinatus/greater tuberosity tenderness		0	1	2	3	0	1	2	3
AC joint tenderness		0	1	2	3	0	1	2	3
Biceps tendon tenderness (or rupture)		0	1	2	3	0	1	2	3
Other tenderness – list:		0	1	2	3	0	1	2	3
Impingement I (passive forward elevation in slight internal rotation)		Y	N			Y	N		
Impingement II (passive internal rotation with 90 degree flexion)		Y	N			Y	N		
Impingement III (90 degree active abduction – classic painful arc)		Y	N			Y	N		
Subacromial crepitus		Y	N			Y	N		
Scars – location:		Y	N			Y	N		
Atrophy – location:		Y	N			Y	N		
Deformity – describe:		Y	N			Y	N		

Richards RR, An K-N, Bigliani LU, et al. A standardized method for the assessment of shoulder function. *J Shoulder Elbow Surg.* 1994;3:347–352.

STRENGTH (record MRC grade)				
0 = no contraction; 1 = flicker; 2 = movement with gravity eliminated 3 = movement against gravity; 4 = movement against some resistance; 5 = normal power				
	Right		Left	
Testing affected by pain?	Y	N	Y	N
Forward elevation				
Abduction				
External rotation (arm comfortably at side)				
Internal rotation (arm comfortably at side)				
Shoulder elevation (shoulder shrug)				
Scapular retraction				
Scapular protraction				
Scaption (prone shoulder flexion in scapular plane)				

INSTABILITY								
0 = none; 1 = mild (0 – 1 cm translation) 2 = moderate (1 – 2 cm translation or translates to glenoid rim) 3 = severe (>2 cm translation or over rim of glenoid)								
	Right				Left			
Anterior translation	0	1	2	3	0	1	2	3
Posterior translation	0	1	2	3	0	1	2	3
Inferior translation (sulcus sign)	0	1	2	3	0	1	2	3
Anterior apprehension test positive?	Y	N			Y	N		
Reproduces symptoms?	Y	N			Y	N		
Voluntary instability?	Y	N			Y	N		
Relocation test positive?	Y	N			Y	N		
Generalized ligamentous laxity?	Y				N			
Other physical findings:								
Examiner: _____ Date: ___/___/___								

Richards RR, An K-N, Bigliani LU, et al. A standardized method for the assessment of shoulder function. *J Shoulder Elbow Surg.* 1994;3:347–352.

RANGE OF MOTION

	Right Shoulder			Left Shoulder		
	Trial 1	Trial 2	Trial 3	Trial 1	Trial 2	Trial 3
Internal Rotation						
External Rotation						

Notes: _____

STRENGTH

	30-30-30			90-0		
	Trial 1	Trial 2	Trial 3	Trial 1	Trial 2	Trial 3
Internal Rotation						
External Rotation						

Notes: _____

HUMERAL RETROVERSION

	Right Shoulder			Left Shoulder		
	Trial 1	Trial 2	Trial 3	Trial 1	Trial 2	Trial 3

Notes: _____

Appendix 2

Consent Forms



DUQUESNE UNIVERSITY

600 FORBES AVENUE ♦ PITTSBURGH, PA 15282

CONSENT TO PARTICIPATE IN A RESEARCH STUDY

TITLE:

Humeral Retroversion, Range of Motion, and Strength Adaptations in Tennis Players

INVESTIGATOR:

Daniel Hannah, MA, LAT, ATC
Rehabilitation Science Program
Duquesne University, 600 Forbes Ave.
232 Health Sciences Building
Pittsburgh, PA 15243
hannahd@duq.edu
412.396.4738 (lab)
[REDACTED] (mobile)

ADVISOR:

Jason Scibek, PhD, LAT, ATC
Department of Athletic Training
Duquesne University, 600 Forbes Ave.
119 Health Sciences Building
Pittsburgh, PA 15243
scibekj@duq.edu
412.396.5960 (office)

SOURCE OF SUPPORT:

This study is being performed as partial fulfillment of the requirements for the Doctor of Philosophy degree in Rehabilitation Science in the John G. Rangos, Sr. School of Health Sciences at Duquesne University.

PURPOSE:

We are asking you to take part in a research study because we are trying to learn more about how the shoulder adapts to playing tennis. The adaptations we are evaluating are those related to the amount of twist in the bone of your upper arm, the range of motion in your shoulder, and shoulder strength.

In order to qualify for participation, you must:

- be between 14 and 25 years of age
- be a current member of an area high school team or tennis club/association, and tennis must be your primary sport, **OR** be an intercollegiate-level tennis players/current members of a college or university sponsored tennis team

competing in the National Collegiate Athletic Association or National Association of Intercollegiate Athletics

- have no known shoulder injury in the prior 6 weeks prior to this testing
- have no elbow or shoulder surgery in the previous 6 months
- have no current elbow or shoulder pain that limits your ability to play tennis
- have no known nerve conditions that would affect muscle strength or motion of your arms.

PARTICIPANT PROCEDURES:

The things you will be asked to do in this study are:

1. We will start the testing by asking questions about how long you have played tennis. We will measure your height and weight. Throughout the testing you will be allowed to wear garments that will preserve modesty (i.e. tank tops, sports bras, halter tops)
2. You will be asked to fill out a brief questionnaire with your parent/guardian to ensure eligibility for this study. In order to confirm eligibility, a certified athletic trainer will examine your shoulder using clinical orthopedic tests that will be used to assess the ability of your shoulder to move, the strength of the shoulder muscles, the ability of the muscles and ligaments of the shoulder to stabilize the shoulder, and the location of any shoulder pain. In the event that any clinical signs of impairment are identified your participation in the study will cease and you will be directed to follow up with health services or a primary care physician for further assessment.
3. We will measure how much motion you have in both of your shoulders. You will lie on your back on a table when we measure your motion. We will raise up your arm by your side and rotate your arm forwards and backwards. This motion is similar to when you serve a tennis ball or an overhead slam/smash.
4. We will measure the strength of your dominant shoulder (the shoulder you use to serve or overhead slam/smash). We want to see how strong you are. We will measure strength in two positions. This first position you will be in a sitting position. The second position you will be lying on your back in the same position used to measure your shoulder motion. You will be asked to push against a small scale that will be held by the researcher. The researcher will hold the scale strongly to prevent you from moving. In both positions, you will be asked to push as hard as you can; a 100% effort. We will give you a few practice attempts for each test position. In total, you will give 12 maximum efforts that last only 5 seconds each.
5. We will measure the amount of twist in both your right and left upper arm bones. You will lie on your back in the same position used to measure shoulder motion and strength. We will ask you to lie still. We will hold your arm in a specific position. We will use a machine called ultrasound to help measure the amount of twist. This will allow us to see your upper arm bone in detail. We will align the ultrasound with your bone and measure the tilt of the device with a digital level. This machine will not cause any harm or pain.

These are the only requests that will be made of you.

RISKS AND BENEFITS:

There are no risks greater than those encountered in everyday life with participation in this study. You may experience minor muscle soreness or fatigue during the strength testing due to the physical nature of the strength tests. The level of discomfort should be no more than you would encounter during a routine examination of your shoulder. In the event that you feel any discomfort notify the investigator in order to allow for additional rest periods during the testing.

Diagnostic ultrasound is safe and has no known risks associated with its use.

During this study you may learn about differences between your dominant and nondominant sides in shoulder motion, strength, and/or the amount of twist in the upper bone of your arm. There are current suggestions for allowable differences for shoulder motion and strength differences in overhead athletes. We will provide you with information about your measures as compared to normal measures. Bone twist measures of the upper arm have not been previously measured in tennis players. Currently, the evidence is not conclusive as to whether this asymmetry is benign, a performance enhancing adaptation in overhead athletes, or poses as an increase in the risk of future injury. We do not know if you will be helped by being in this study. We may learn something that will help others who develop adaptations from playing tennis that may help reduce the risk of injury in the future.

COMPENSATION:

There will be no money given to you for participating in this study, but your participation will also not cost you anything.

CONFIDENTIALITY:

Your participation in this study and any personal information that you provide will be kept confidential at all times and to every extent possible.

Your name will never appear on any survey or research instruments. All written and electronic forms and study materials will be kept secure. Your response(s) will only appear in statistical data summaries. Any study materials with personal identifying information will be maintained for five years after the completion of the research and then destroyed.

RIGHT TO WITHDRAW:

You are under no obligation to participate in this study. You are free to withdraw your consent to participate at any time by calling (██████████) or emailing (hannahd@duq.edu) the Principal Investigator, Daniel Hannah. The contact information is also listed on the first page of this document. In addition, you may feel free to withdraw from the study during the data collection process. Simply let the researchers know and we will comply with your request. You can tell us if we can use any information we already collected from you, or you can have us delete/destroy the information.

SUMMARY OF RESULTS:

A summary of the results of this research will be supplied to you, at no cost, upon request.

VOLUNTARY CONSENT:

I have read the above statements and understand what is being requested of me. I also understand that my participation is voluntary and that I am free to withdraw my consent at any time, for any reason. On these terms, I certify that I am willing to participate in this research project.

I understand that should I have any further questions about my participation in this study, I may call Daniel Hannah at [REDACTED] or Dr. Jason Scibek at 412.396.5960. Should I have any questions regarding protection of human subject issues, I may contact Dr. David Delmonico, Chair of the Duquesne University Institutional Review Board, at 412.396.1886.

Participant's Signature

Date

Researcher's Signature

Date



DUQUESNE UNIVERSITY

600 FORBES AVENUE ♦ PITTSBURGH, PA 15282

CHILD'S AGREEMENT TO PARTICIPATE IN A RESEARCH STUDY

TITLE:

Humeral Retroversion, Range of Motion, and Strength Adaptations in Tennis Players

WHO IS DOING THE RESEARCH?

Daniel Hannah, MA, LAT, ATC
Rehabilitation Science Program
Duquesne University, 600 Forbes Ave.
232 Health Sciences Building
Pittsburgh, PA 15243
hannahd@duq.edu
412.396.4738 (lab)
[REDACTED] (mobile)

Jason Scibek, PhD, LAT, ATC
Department of Athletic Training
Duquesne University, 600 Forbes Ave.
119 Health Sciences Building
Pittsburgh, PA 15243
scibekj@duq.edu
412.396.5960 (office)

WHY ARE THE RESEARCHERS DOING THIS STUDY?

We are asking you to take part in a research study because we are trying to learn more about how the shoulder adapts to playing tennis. The adaptations we are evaluating are those related to the amount of twist in the bone of your upper arm, the range of motion in your shoulder, and shoulder strength.

In order to participate, you must:

- be 14 years old or older
- be a current member of an area high school team or tennis club/association, and tennis must be your primary sport
- have no known shoulder injury in the prior 6 weeks prior to this testing
- have no elbow or shoulder surgery in the previous 6 months
- have no current elbow or shoulder pain that limits your ability to play tennis
- have no known nerve conditions that would affect muscle strength or motion of your arms.

WHAT DO YOU HAVE TO DO?

The things you will be asked to do in this study are:

1. We will start the testing by asking questions about how long you have played tennis. We will measure your height and weight. Throughout the testing you will be allowed to wear garments that will preserve modesty (i.e. tank tops, sports bras, halter tops)
2. You will be asked to fill out a brief questionnaire with your parent/guardian to ensure eligibility for this study. In order to confirm eligibility, a certified athletic trainer will examine your shoulder using clinical orthopedic tests that will be used will assess the ability of your shoulder to move, the strength of the shoulder muscles, the ability of the muscles and ligaments of the shoulder to stabilize the shoulder, and the location of any shoulder pain. In the event that any clinical signs of impairment are identified your participation in the study will cease and you will be directed to follow up with health services or a primary care physician for further assessment.
3. We will measure how much motion you have in both of your shoulders. You will lie on your back on a table when we measure your motion. We will raise up your arm by your side and rotate your arm forwards and backwards. This motion is similar to when you serve a tennis ball or an overhead slam/smash.
4. We will measure the strength of your dominant shoulder (the shoulder you use to serve or overhead slam/smash). We want to see how strong you are. We will measure strength in two positions. This first position you will be in a sitting position. The second position you will be lying on your back in the same position used to measure your shoulder motion. You will be asked to push against a small scale that will be held by the researcher. The researcher will hold the scale strongly to prevent you from moving. In both positions, you will be asked to push as hard as you can; a 100% effort. We will give you a few practice attempts for each test position. In total, you will give 12 maximum efforts that last only 5 seconds each.
5. We will measure the amount of twist in both your right and left upper arm bones. You will lie on your back in the same position used to measure shoulder motion and strength. We will ask you to lie still. We will hold your arm in a specific position. We will use a machine called ultrasound to help measure the amount of twist. This will allow us to see your upper arm bone in detail. We will align the ultrasound with your bone and measure the tilt of the device with a digital level. This machine will not cause any harm or pain.

HOW LONG WILL YOU BE IN THE RESEARCH STUDY?

The study will take place during a single testing session lasting 45 minutes.

IS THIS STUDY HARMFUL? HOW IS IT HELPFUL?

Your involvement in this study is not any more harmful than other things you do in your life. You may experience minor muscle soreness or fatigue during the strength testing due to the physical nature of the strength tests. The level of discomfort should be no more than you would encounter during a routine examination of your shoulder by a healthcare provider. If you feel any discomfort notify the researcher to allow for additional rest periods during the testing. If there are any questions or steps that you do not feel comfortable answering or performing, you do not have to do so.

Diagnostic ultrasound is safe and has no known risks associated with its use.

During this study you may learn about differences between your right and left sides in shoulder motion, strength, and/or the amount of twist in the upper bone of your arm. There are current suggestions for allowable differences for shoulder motion and strength differences in overhead athletes. We will provide you with information about your measures as compared to normal measures. Bone twist measures of the upper arm have not been previously measured in tennis players. Current information is not conclusive as to whether this asymmetry is harmless, improves the ability to perform overhead activities, or poses as an increase in the risk of future injury. We do not know if you will be helped by being in this study. We may learn something that will help others who develop adaptations from playing tennis that may help reduce the risk of injury in the future.

Again, if anything hurts or you are uncomfortable with some of the questions, please let us know and we will stop or do whatever we can to make you feel better.

WILL YOU GET PAID TO DO THIS STUDY?

There will be no money given to you for participating in this study, but your participation will also not cost you anything.

ARE OTHER PEOPLE GOING TO KNOW WHAT YOU DID OR SAID?

We will keep the things you say and do confidential.

If we find useful information in our research we will want to share it with others, either by writing a paper about it, or talking about it with other professionals. If we do this, we will never give out your name or talk about you in a way that someone could figure out who you are or what you said in the research. If there are other things during the research that have your name on them, we will keep them locked in a password protected file or a locked filing cabinet for five years, then we will shred them or delete them from our computer.

CAN YOU QUIT IF YOU WANT?

Yes. You don't even have to start if you don't want. If you do start, and decide you don't want to do it anymore, just tell one of the researchers, or tell one of your caregivers/parents so they can tell us. Don't worry; no one will be mad at you if you decide to stop. If you decide to stop, you can tell us if we can use any information we already got from you, or you can have us delete it all. It's up to you.

CAN YOU HEAR ABOUT WHAT HAPPENED?

After the study is completely over, the researchers have to get all of the information together and look at it. Once we do, we will type up a paper about it, and you can have a copy of our paper if you want. Just let us know that you would like to have a copy of it and we will provide it to you for free.

OK...WOULD YOU LIKE TO DO IT?

If you agree to participate, please sign on the line below that says “Participant’s Signature.” This means you are ready to participate. If you still have questions, you can ask them by calling Daniel Hannah at [REDACTED] or Dr. Jason Scibek at 412.396.5960. If you have questions regarding how you are protected in the study, then the best person to contact would be Dr. David Delmonico, Chair of the Duquesne University Institutional Review Board, at 412.396.1886.

Participant’s Signature

Date

Parent/Legal Guardian’s Signature

Date

Researcher's Signature

Date



DUQUESNE UNIVERSITY

600 FORBES AVENUE ♦ PITTSBURGH, PA 15282

PARENTAL PERMISSION FORM

TITLE:

Humeral Retroversion, Range of Motion, and Strength Adaptations in Tennis Players

WHO IS DOING THE RESEARCH?

Daniel Hannah, MA, LAT, ATC
Rehabilitation Science Program
Duquesne University, 600 Forbes Ave.
232 Health Sciences Building
Pittsburgh, PA 15243
hannahd@duq.edu
412.396.4738 (lab)
[REDACTED] (mobile)

ADVISOR:

Jason Scibek, PhD, LAT, ATC
Department of Athletic Training
Duquesne University, 600 Forbes Ave.
119 Health Sciences Building
Pittsburgh, PA 15243
scibekj@duq.edu
412.396.5960 (office)

SOURCE OF SUPPORT:

This study is being performed as partial fulfillment of the requirements for the Doctor of Philosophy degree in Rehabilitation Science in the John G. Rangos, Sr. School of Health Sciences at Duquesne University.

WHY IS THIS RESEARCH STUDY BEING DONE?

We are asking your child to take part in a research study because we are trying to learn more about how the shoulder adapts to playing tennis. The adaptations we are evaluating are those related to the amount of twist in the bone of your child's upper arm (humeral retroversion), the range of motion in your child's shoulder, and your child's shoulder strength.

In order to qualify for participation, your child must:

- be 14 years of age or older
- be a current member of an area high school team or tennis club/association, and tennis must be his/her primary sport, **OR** be an intercollegiate-level tennis players/current members of a college or university sponsored tennis team

competing in the National Collegiate Athletic Association or National Association of Intercollegiate Athletics

- have no known shoulder injury in the prior 6 weeks prior to this testing
- have no elbow or shoulder surgery in the previous 6 months
- have no current elbow or shoulder pain that limits their ability to play tennis
- have no known nerve conditions that would affect muscle strength or motion of their arms.

WHAT WILL MY CHILD BE ASKED TO DO?

The things your child will be asked to do in this study include:

We will start the testing by asking questions about how long your child has played tennis. We will measure their height and weight. Throughout the testing they will be allowed to wear garments that will preserve modesty (i.e. tank tops, sports bras, halter tops)

Your child will be asked to fill out a brief questionnaire to ensure eligibility for this study. In order to confirm eligibility, a certified athletic trainer will examine your child's shoulder using clinical orthopedic tests that will be used to assess the shoulder's ability to move, the strength of the shoulder muscles, the ability of the muscles and ligaments of the shoulder to stabilize the shoulder, and the location of any shoulder pain. In the event that any clinical signs of impairment are identified your child's participation in the study will cease and you will be directed to follow up with health services or a primary care physician for further assessment of your child's condition.

We will measure the amount of rotational motion available in both shoulders of your child. These simple measures are routinely performed during an orthopedic evaluation.

Next, the investigator will measure your child's strength of the muscles surrounding the shoulder. He/She will be asked to place their arm or shoulder in a specific position. They will then be asked to push against a handheld strength gauge so that shoulder strength can be measured. During this part of the testing they will either be seated or lying on a table. Again, they will be given verbal instructions and opportunities to practice the activity.

To measure the amount of twist (humeral retroversion) of your child's upper arm bones, the investigator will use a diagnostic ultrasound machine to view and align bony landmarks of the upper arm bone. Diagnostic ultrasound will allow the investigator to view the bony anatomy in real-time. Once the landmarks are aligned with the ultrasound, the investigator will measure the position of their forearm with a digital inclinometer. A digital inclinometer is a handheld device used to measure joint motion similar to a carpenter's level. Separate measurements will be taken from both arms while they are positioned on their back on a treatment table.

Your child's participation in this study will involve a single testing session. The testing session will last 45 minutes.

These are the only requests that will be made of your child.

WHAT ARE THE RISKS AND BENEFITS OF THIS STUDY?

There are no risks greater than those encountered in everyday life with participation in this study. Your child may experience minor muscle soreness or fatigue during the strength testing due to the physical nature of the strength tests. The level of discomfort should be no more than what they would encounter during a routine examination of their shoulder. In the event that your child feels any discomfort please let them know that they should notify the investigator in order to allow for additional rest periods during the testing. You may also feel free to intervene.

Diagnostic ultrasound is safe and has no known risks associated with its use.

During this study, you may learn about asymmetries between your child's dominant and nondominant sides in shoulder motion, strength, and/or humeral retroversion. There are current suggestions for allowable differences for shoulder motion and strength differences in overhead athletes. We will provide you with information about your child's measures as compared to normal measures. Bone twist measures of the upper arm have not been previously measured in tennis players. Currently, the evidence is not conclusive as to whether this asymmetry is benign, a performance enhancing adaptation in overhead athletes, or poses as an increase in the risk of future injury. We do not know if your child will be helped by being in this study. We may learn something that will help others who develop adaptations from playing tennis that may help reduce the risk of injury in the future.

WILL MY CHILD BE PAID FOR TAKING PART IN THIS RESEARCH STUDY?

There will be no money given to your child for participating in this study, but your child's participation will also not cost you anything.

CONFIDENTIALITY:

Your child's participation in this study and any personal information that you or your child provides will be kept confidential at all times and to every extent possible.

Your child's name will never appear on any survey or research instruments. All written and electronic forms and study materials will be kept secure. No identity will be made in data analysis. Any study materials with personal identifying information will be maintained for five years after the completion of the research and then destroyed.

RIGHT TO WITHDRAW:

You are under no obligation to give your permission for your child to participate in this study, and you may withdraw your permission at any time by notifying a member of the research team. You may also choose your child's data to be completely withdrawn from the study or allow any data collected to be used in the final statistical analysis.

SUMMARY OF RESULTS:

A summary of the results of this research will be supplied to you, at no cost, upon request.

VOLUNTARY CONSENT:

I have read the above statements and understand what is being requested of me and my child. I also understand that my child’s participation is voluntary and that I am free to withdraw my permission for my child at any time, for any reason.

On these terms, I agree that I am willing to allow my child to participate in this research project.

I understand that should I have any further questions about my child’s participation in this study, I may contact Daniel Hannah at [REDACTED] or Dr. Jason Scibek at 412.396.5960. Should I have questions regarding protection of human subject issues, I may contact Dr. David Delmonico, Chair of the Duquesne University Institutional Review Board, at 412.396.1886.

Please select **ONE** of the following options:

- I require that I be present with my child during the orthopedic screening and data collection procedures.
- I allow my child to participate in this study without my presence.

Parent/Legal Guardian’s Signature

Date

Researcher's Signature

Date