EFFECTS OF THE PRONE-PASSIVE STRETCHING TECHNIQUE
ON GLENOHUMERAL INTERNAL ROTATION

by

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ABSTRACT

Effects of the Prone Passive Stretching Technique on Glenohumeral Internal Rotation

Introduction: Posterior shoulder tightness is widely considered a causative factor for multiple disorders of the glenohumeral (GH) joint. Specifically, Glenohumeral Internal Rotation Deficit (GIRD) creates the potential for a cascading effect in overhead athletes leading to shoulder dysfunction. Both conservative and surgical interventions have been discussed in the literature to reduce the symptoms of GIRD and posterior GH tightness.

Purpose: The purpose of this study was to evaluate the effectiveness of a novel prone-passive internal rotation (IR) stretching technique compared to the cross-body stretching technique at improving glenohumeral IR. The prone technique has been described once in the literature, but never studied, while other techniques have been investigated through empirical research. Hypothesis: The novel prone-passive stretching technique is more effective at improving IR ROM, IR deficit, and total motion, when compared to the previously studied cross-body stretching technique. Methods: Following a pilot study, 34 healthy and non-injured athletic participants who demonstrated a deficit between non-dominant and dominant shoulders of $\geq 10^\circ$ were recruited. Participants were randomly assigned to a study group (passive-prone) and control group (cross-body). Pre-test digital inclinometer measurements revealed pre-test GH IR and external (ER) range of motion; a minimum of 12 treatments were applied in both groups and post-treatment measurements
were taken using the same instrumentation. All measurements were taken by the same research assistant. **Results:** Improvements in group mean gain scores IR ROM, IR deficit, and total motion, in the experimental compared to the control group, were not statistically significant: IR ROM (13.23° ± 7.78°, 8.47° ± 8.71° p = 0.104), IR deficit (-12.64° ± 11.49°, -9.13 ± 8.33° p = 0.441), and total motion (14.81° ± 11.27°, 9.97° ± 11.99° p = 0.232). **Conclusion:** The prone-passive stretching technique did not demonstrate significant improvement in IR ROM, IR deficit, and total motion in the glenohumeral joint in participants with unilateral IR deficit more than the cross-body stretching technique. However, due to an observed trend, the results of this study may motivate clinicians to utilize the prone-passive stretching technique for the treatment of unilaterally restricted IR ROM in overhead athletes.
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CHAPTER I: INTRODUCTION/LITERATURE REVIEW

Internal rotation of the glenohumeral joint becomes limited in the dominant shoulder of many overhead athletes. This condition, many times asymptomatic in terms of pathology, could become troublesome for the athlete as the condition progresses. Clinicians often prescribe a stretching treatment to counter restrictions in the dominant shoulder, including those that affect the glenohumeral joint, such as glenohumeral internal rotation deficit (GIRD) or posterior shoulder tightness (PST).

Various techniques are employed by clinicians to treat such deficits between shoulders with a goal of restoring normal motion. The purpose of this study is to compare the effect a novel passive stretching technique has on glenohumeral (GH) internal rotation compared to a self-stretching technique in individuals with PST and or GIRD. This stretch has not been investigated through prior study.

This section intends to validate the purpose of this study, but also demonstrate potential voids in previous research regarding the study question, “Is the prone internal rotation (IR) stretching technique effective at improving glenohumeral joint IR?” The literature review is organized into three critical components of interest to the study: 1) an explanation of GIRD or internal rotation limitations, 2) conflicting agreements surrounding the etiology of conditions such as GIRD and or PST, and 3) intervention treatments to counter the effects of unilaterally restricted IR ROM in overhead athletes.
GIRD

The human glenohumeral (GH) joint allows the most uninhibited joint motion in the human body. Restriction of normal ROM in the GH joint complex is a common musculoskeletal disorder that creates functional problems especially for the overhead-throwing athlete. Posterior shoulder tightness (PST), and/or glenohumeral internal rotation deficit (GIRD), is of specific interest to this study since limitation, or reduction of IR, is the primary factor in the evaluation and subsequent diagnosis of such pathologic conditions (Burkhart, Morgan, & Kibler, 2003a; Meister, 2000). Previously, Burkhart et al. (2003a), who have studied baseball players specifically, coined the most often referenced definition of GIRD as being, “loss in degrees of GH IR of the throwing shoulder compared with the non-throwing shoulder.” Further, Burkhart et al. has more specifically described GIRD as a limitation in IR of greater than or equal to 20°, or greater than 10 percent when compared to IR ROM in the non-dominant extremity. It is common for overhead athletes to exhibit a shift towards GH external rotation (ER) motion in the dominant shoulder and reduced IR compared to the non-dominant extremity. This phenomenon of the overhead athlete shoulder is explained through the total-motion concept. Total motion, as described by Crockett and associates (2002) in a study that investigated increases in ER in patients with IR deficit, refers to the combined IR and ER ROM in the GH joint. In this study, the authors found subjects with limited IR ROM, however the increase noted in ER ROM created similar total motion measurements in both the involved and non-involved GH joint. While the total-motion concept may support theories regarding improvements in pitching performance, most
notably through increased velocity, it does not attempt to address the potential for a baseball pitcher to exhibit limitation in IR ROM.

This phenomenon of the GH joint is not unique to athletes involved in baseball, but has been a common finding in most other overhead dominant sports as well (Beach, Whitney, & Dickoffhoffman, 1992; Crockett et al., 2002; Ellenbecker, Roetert, Bailie, Davies, & Brown, 2002; Ellenbecker, Roetert, Piorkowski, & Schulz, 1996; Kibler & Chandler, 2003).

Cascading Effect of GIRD / PST

Many authors have investigated the deleterious effects of PST and/or GIRD, which include a common injury to the shoulder, the superior labrum anterior posterior (SLAP) lesion (Burkhart & Morgan, 1998; Kibler, 1998), and variations of impingement syndrome, sub-acromial, or extrinsic, and internal GH impingement (Myers, Laudner, Pasquale, Bradley, & Lephart, 2006; Paleo, Jobe, Pink, Kvitne, & ElAttrache, 2000; Tyler, Nicholas, Lee, Mullaney, & McHugh, 2010; Warner, Micheli, Arslanian, Kennedy, & Kennedy, 1990). In this regard, PST and GIRD are not necessarily viewed as injuries, but more likely as a causative factor that leads to a cascade of pathologic issues in the shoulder complex. Ultimately, the overhand athlete’s reduction in IR may lead to a host of musculoskeletal disorders of the GH joint complex. Many studies have investigated baseball players, specifically pitchers, due to the stressful nature of the overhand throw and the propensity of these athletes to develop significant shoulder injuries (Ellenbecker et al., 2002; Tyler et al., 2010).

Most studies have explained GIRD / PST as being a significant contributor toward the development of other shoulder injuries. Pappas, Zawacki, and McCarthy (1985) may
have initiated the study on the effects of limited shoulder motion in overhead athletes. In this seminal work, which explained techniques for evaluating and rehabilitating the shoulder, Pappas et al. discussed how pathology in the shoulder gradually develops as a result of a lack of flexibility in soft tissues, specifically capsular structures.

In a recent study to determine if professional baseball players with and without a history of shoulder injury exhibit differences in GH ROM, Scher et al. (2010) produced data consistent with those previously mentioned and similarly support GIRD as a precursor to shoulder pathology. Interestingly, Scher et al. also suggests that some cases of GIRD may go unrecognized because his data revealed evidence of GIRD in subjects with less than 20° difference in IR ROM, the benchmark for identifying the disparity in throwing and non-throwing ROM as previously defined by Burkhart et al. (2003a). Similarly, Wilk et al. (2011) found professional baseball pitchers who exhibit GIRD and/or total rotational motion deficit were nearly twice as likely as those without the condition to sustain a dominant shoulder or elbow injury. The correlation between GIRD and throwing extremity injuries has been demonstrated in high school aged athletes as well. In another recent study, Shanley et al. (2011) reported baseball and softball high school athletes with significant levels of GIRD to be four times as likely to sustain a shoulder or elbow injury during the same season when compared to those with a 10°-20° deficit (1.5 – 2 times more likely than no deficit).

Most literature reviewed for this study involves participants in baseball, swimming, and tennis, and the participants were of adult age. Pappas et al. (1985) first mentioned the likelihood of an increased potential for limitations in flexibility in more experienced baseball players. Similarly, Jobe, Giangarra, Kvitne, & Glousman (1991)
proposed that repetitive throwing, over time, stretches the anterior capsular ligaments, leading to a potential for rotator cuff injury. Kibler, Chandler, Livingtson, and Roetart (1996) also demonstrated, in elite tennis players, a negative correlation between years of play and increasing limitations in IR ROM. In a unique study, which compared elite tennis players (mean age = 16.4) and professional baseball pitchers (mean age = 26.6), Ellenbecker et al. (2002) demonstrated that both study groups met criteria to be categorized as GIRD. However, both groups were experienced athletes who undoubtedly placed repetitive stress on the GH complex.

Very little research has investigated the prevalence of GIRD and/or PST in youth baseball or other overhand youth athletes. However, the studies have all demonstrated evidence that signs of PST and or GIRD may be prevalent in young baseball players. Nakamizo, Nakamura, Nobuhara, and Yamamoto (2008) identified GIRD in 10 out of 25 male little league Japanese pitchers (mean age = 11.4). While not investigating GIRD or PST, a study by Sabick and colleagues revealed signs that levels of shear stress in the late cocking phase of overhand pitching in youth baseball players was large enough to result in deformation of epiphyseal cartilage (Sabick, Kim, Torry, Keirns, & Hawkins, 2005). Perhaps most revealing to the concept of an early onset of the cascading effects of GIRD and/or PST was a study conducted by Meister and colleagues (2005) who evaluated 294 baseball players aged 8-16 years old. This seminal study found that total ROM decreased as age increased. In particular, IR ROM averaged 39° ± 6.5° in 8 year olds and decreased to 21° ± 8° in 16 year olds. Notably, the most significant decrease in total ROM occurred between 13 and 14 years old. With such a large number of subjects in this study, the
authors demonstrated with statistical power and clinical relevance that limitation in GH ROM quite possibly begins early in life, at least in overhand throwers.

**Etiology of GIRD and/or PST**

Unlike most musculoskeletal conditions, those of interest to this study, GIRD and/or PST, are disorders that arise from an insidious cause. Previous research has provided data to shed light on the issue of limited IR ROM and increased ER ROM in the overhand athlete, but differing views exist as to the cause of GIRD and/or PST. The intent of this study is to investigate an intervention to reverse or counter the effects of GIRD and/or PST.

From a historical perspective, King, Brelsford, and Tullos (1969), in an analysis of elbow injuries and patho-mechanics, were the first to mention an appreciable difference between dominant throwing arm and non-throwing arm decreases in IR and increases in ER ROM in the professional baseball pitcher population. This observation was made based on goniometric measurements of GH motions. The purpose of the King et al. study was not to investigate limitations in GH ROM, but to investigate pathologic conditions.

Prior to the study by King et al. (1969), Bennett postulated a cause of debilitating shoulder pain in the professional baseball pitcher brought on by repetitive pull on the posterior capsule by the triceps tendon in the area of the posterior and inferior glenoid capsule, leading to the development of a characteristic bone spur or osteophyte in this location. This spur is known as the Bennett’s lesion in clinical diagnoses (Bennett, 1941; Bennett, 1959). The Bennett’s lesion, although a finding of importance to clinicians interested in shoulder pathology, does not itself explain the effects of GIRD and/or PST.
The investigation by Pappas et al. (1985) concurred with prior opinion regarding the changes in GH motion of the throwing shoulder. Also, Pappas et al. may have been the first to report on both passive and active stretching techniques used to evaluate IR ROM by using horizontal flexion (adduction) to assess the structures of the posterior shoulder. The recommendations of that research have had far reaching implications in future studies. Pappas, by virtue of his experience with throwing athletes and by evaluating GH ROM through active and passive movement, determined the restricted motion in IR was caused by both tight anterior and posterior structures, either capsular or muscular (Pappas et al., 1985). As a result of this study, Pappas and colleagues strongly emphasized a supervised stretching routine to counter the effects of restricted GH ROM.

Soft Tissue Tightness and Contracture

Similar to studies on musculoskeletal pathology, research surrounding the etiology of GIRD and PST has focused on two different pathologic anatomic structures. Early researchers produced data analysis regarding the throwing shoulder that led to discussion about muscular and capsular tightness, or contracture, as a cause of PST (Bennett, 1959; King et al., 1969; Pappas et al., 1985).

In a study investigating GH ROM and laxity in 148 professional baseball players, researchers identified significant differences of IR ROM in dominant versus non-dominant shoulders and attempted to correlate this with observed GH laxity between pitchers and non-pitchers. Findings were significant for limitations in IR ROM differences between groups, however there was no notable correlation between ROM and laxity. In this study, Bigliani et al. (1997) postulate the reduction and changes in total motion are attributable to repetitive micro-tearing of soft-tissues during overhead
throwing, which leads to tightening of the posterior GH capsule. Later, Sauers and colleagues demonstrated similar findings between laxity and GH ROM in 51 recreational athletes (Sauers, Borsa, Herling, & Stanley, 2001).

Posterior capsule and/or posterior musculature tightness, as a reliable cause of limitation in IR ROM, tends to be the most widely accepted philosophy of GIRD and/or PST etiology. The posterior GH capsule was presented as a cause of restricted IR ROM by Lombardo, Jobe, Kerlan, Carter, and Shields, Jr. (1977). The authors of this study presented four case studies, all involving professional baseball players, who underwent excision of a Bennett’s lesion, and upon visualization of the posterior capsule the surgeon noted a posterior capsular thickening. In another surgical treatment observation, Ticker, Beim, and Warner (2000) reported on nine patients who underwent an arthroscopic release of the posterior GH capsule; in all patients, posterior capsule thickening was observed. Thickening of the posterior inferior capsular ligament of the GH joint has been reported in other studies, but more recently an imaging study using magnetic resonance imaging to diagnose GIRD in a sample of six professional baseball players revealed the same findings, along with other disorders, in all participants (Tehranzadeh, Fronek, & Resnick, 2007).

Tyler, Nicholas, Roy, and Gleim (2000) claim to be the first to clinically link posterior capsule tightness to the loss of internal rotation range of motion, excluding posterior shoulder muscular tightness as a cause. The authors also propose their belief that the posterior GH capsule leads to losses in IR ROM by recommending that a clinician can expect that for every four degrees of internal range of motion lost there will be one centimeter of posterior capsule tightness. The procedure used to assess posterior
capsule tightness had been validated through a prior research study (Tyler, Roy, Nicholas, & Gleim, 1999). This procedure was used to assess PST in impingement patients in another study by Tyler and associates where they found symptoms associated with the pathology improved along with reduction in PST and GIRD, however the investigators elude to GIRD arising from humeral head translation, not posterior GH capsule tightness (Tyler et al., 2010).

In a widely cited series entitled The Disabled Throwing Shoulder, Burkhart et al. (2003a) again proposed loss of internal rotation as a result of postero-inferior capsule contracture. However, these authors do not dismiss other potential causative factors of GIRD and/or PST, such as posterior shoulder musculature tightness.

In a review, Blanch (2004) proposed that the posterior shoulder musculature is an important causative factor of restricted GH joint ROM. While referring to the intimate blending of the GH capsule and shoulder musculature, Blanch recommended that it would be more appropriate to refer to tightness of the posterior shoulder rather than specifically implicating the capsule. This study was conducted in an attempt to create a specialized shoulder rehabilitation program aimed at swimmers.

In a recent cadaveric study using 8 fresh specimens instrumented with strain gauges in the capsule, Borstad and Dashottar (2011) examined strain effects of various stretching positions on the posterior gleno-humeral capsule. While the research demonstrates significantly higher levels of strain in the middle section of the capsule with the humerus flexed and internally rotated, the researchers could not completely identify the capsule tissue as the only causative factor of posterior shoulder tightness. Strain was
also increased in the teres minor and the infraspinatus muscles of the posterior shoulder girdle.

Far fewer studies on PST and/or GIRD have explained the causative factor as an issue of the posterior shoulder musculature, including the rotator cuff. Recently, Oyama, Myers, Blackburn, and Coleman (2011) observed a significant increase in the area size of the infraspinatus muscle, along with decreased IR and horizontal adduction, in a sample of 20 healthy subjects who underwent an eccentric external rotation (ER) workout up to 24 hours after a baseline ultrasound measurement, p. < .001.

In a case report of a non-athlete with PST symptoms associated with sub-acromial impingement, Poser and Casonato (2008) applied a treatment only to posterior musculature, focusing on the infraspinatus and teres minor, and did not attempt to treat the posterior capsule. This soft tissue treatment resulted in significant improvements in restoring IR ROM, however this case study investigated only one patient. In a study by Hung, Hsieh, Yang, and Lin (2010) investigating twenty non-athlete patients with stiff shoulder syndrome, researchers hypothesized a correlation between muscle stiffness and GIRD by using a Myotonometer (Neurogenic Technologies, Inc., Montana, USA) an instrument that measures muscle fiber tension. The authors concluded that posterior deltoid muscle tightness attributed to fifty-one percent (51%) of variance in IR ROM in the subjects, a higher correlation than either the infraspinatus or teres minor (r= 0.65-0.72 versus r= 0.57-0.61). An obvious limitation in this study was the use of the Myotonometer, an instrument which assesses relaxed muscle tone over the superficial layer of skin and the authors do mention this in their report.
Humeral Translation and Physiologic Adaptation

Harryman and colleagues (1990) were likely the first to explain GH joint translation in a cadaveric arthrokinematic study. These authors coined the term *obligate translation*, which is a shifting of the humeral head in the opposite direction of tight capsular structure. This seminal study set the stage for future study regarding translation in the GH joint and the emphasis on capsular etiology of PST and or GIRD. More recent cadaveric studies corroborate the Harryman et al. findings but mention the likelihood that posterior capsule tightness, through overhead motion, likely plays a significant factor in not only humeral translation, but other shoulder pathology such as labrum tears and impingement (Grossman et al., 2005; Werner, Nyffeler, Jacob, & Gerber, 2004). Arthrokinematic studies have demonstrated compressive forces of approximately 400 N and inferior shear of approximately 200 N during the follow through phase of the overhead baseball pitch (Dillman, Fleisig, & Andrews, 1993; Fleisig, Andrews, Dillman, & Escamilla, 1995).

Humeral translation refers to movement of the humeral head on the glenoid, creating a potential for increased stress on soft tissue and/or bony structures within the GH joint when translation becomes excessive. The translation then is thought to cause physiologic adaptations in the GH joint: soft tissue and osseous structures. For instance, clinical studies investigating anterior shoulder instability propose altered GH humeral translation as a cause of laxity in the anterior shoulder (Jobe et al., 1991; Paley et al., 2000).

Grossman et al. (2005) determined posterior capsule tightness, created through capsule plication in cadaveric shoulders, creates an abnormal translation posterior-
superiorly on the glenoid instead of the normal posterior-inferior direction during the cocking phase of overhead throwing. The authors described this altered movement to be a causative factor in pathology of the labrum, such as the SLAP tear. This posterior superior movement may be a mechanism of interest for posterior or internal GH impingement as well, whereby the greater tuberosity of the humerus impacts soft tissue structures in the posterior aspect of the shoulder (Bach, 2006; Cools, Declercq, Cagnie, Cambier, & Witvrouw, 2008; Halbrecht, Tirman, & Atkin, 1999; Jobe, 1995; Myers et al., 2006).

Only one prior cadaveric study reviewed for this research on the effectiveness of a passive IR stretching technique for the shoulder found no correlation between posterior capsule tightening and altered humeral translation (Poitras et al., 2010). Unlike the studies mentioned previously, Poitras et al. did not measure translation during the kinematic throwing movements like the others did, but instead evaluated an elevated abduction movement.

However, in an in-vivo study on forty-three asymptomatic throwing shoulders, researchers found no correlation between humeral translation and altered range of motion, such as found in GIRD (Borsa et al., 2005). Physiologic adaption is likely to occur whenever the humeral head translates on the glenoid during overhead motion, which may contribute to contracture or tightness of the posterior shoulder soft tissues.

**Osseous Adaptation**

There is another philosophy on adaptations within the GH joint that may lead to shoulder pathology; adaptations in osseous structure have been presented as a causative factor as well as soft tissue. Humeral retroversion, or retrotorsion, has been described as
the humeral head that is posteriorly situated perpendicular to the glenoid fossa compared to the alignment in a normal shoulder. Prior studies have demonstrated increased retroversion in the dominant shoulder of throwing athletes compared to the non-dominant arm (Crockett et al., 2002; Osbahr, Cannon, & Speer, 2002). The Osbahr et al. study investigated nineteen college baseball pitchers, while the Crockett et al. study evaluated twenty-five professional baseball pitchers compared to twenty-five non-throwing subjects. Both studies produced similar findings in terms of humeral retroversion being correlated to increased external rotation in the throwing shoulders. Both Crockett et al. and Osbahr et al. describe the cause of retroversion from a skeletal development perspective and not an occurrence from an acute event. The previously mentioned work by Sabick and colleagues (2005), which found stresses on the throwing arm and shoulder to be significant enough to cause osseous adaptation, supports this finding. Osbahr and colleagues (2002) utilized a questionnaire to assess throwing frequency between the ages of eight to sixteen years old; however, there was no correlation between duration and amount of throwing and increased humeral retroversion and these ages.

Osbahr et al. (2002) and Sabick et al. (2005) also observed a correlation between humeral retroversion and increased external rotation in the throwing shoulders. Additionally, both studies have discussed this increase in external rotation, a common finding in GIRD, to be a potential benefit to the baseball pitcher in two ways: A pitcher who can reach greater ranges of external rotation would likely generate increased velocity, and, a more posteriorly situated humeral head may actually provide increased stability to the joint by way of bony restriction. Both studies found evidence to support osseous adaptation in the shoulder joint as a cause of symptoms associated with PST,
while Crockett and associates (2002) claim humeral retroversion as the primary contributor to posterior capsular tightness. On the other hand, Osbahr and colleagues (2002) determined the cause of PST to be a combination of both bony and capsular adaptation.

While many causes of PST and/or GIRD have been described in the literature, researchers continue to dispute the philosophies described here. Other potential causes have been sparsely described, for instance, Ludewig and Borstad (2003) describe abnormal spine posture as a potential cause of altered shoulder kinematics. However, this finding has yet to be suggested in an overhead athletic population. A majority of researchers explain humeral translation as a cause of posterior capsular tightness versus tight posterior musculature as a cause of PST, but at least one study described prior work in this area as misleading since isolating the posterior capsule from the posterior shoulder musculature is difficult (Michener, McClure, & Karduna, 2003). In a recent study investigating a link between humeral torsion and elbow/shoulder injuries in collegiate baseball players, the authors suggested osseous adaptation was more likely a factor in PST rather than changes in soft tissues, yet while correlating torsion produced to elbow injuries a similar link was not found in shoulder pathology (Myers et al., 2009).

It is well accepted that repetitive stress on the throwing shoulder, and subsequent micro tearing, does indeed lead to adaptive changes in tissue creating altered range of motion. Although causative theories tend to be conflicting regarding PST, treatment interventions do not. Thus, we turn to the focus of this dissertation and explain prior study investigating the effectiveness of previously investigated interventions to reverse the effects of PST and / or GIRD, including the prone-passive IR stretching technique.
**Intervention Techniques to Counter Reduced Internal Rotation**

This section reviews significant research surrounding various intervention strategies and are categorized as either surgical, or non-surgical. The latter of the two is the category where the prone stretch is situated, so this category details the findings of prior study which have investigated other internal rotation stretching techniques. The current study is a seminal investigation to determine the effectiveness of the prone-passive internal rotation technique on gleno-humeral internal rotation.

**Conservative Intervention**

In 1995, a descriptive article was produced by Johansen, Callis, Potts, and Shall that explained the modified internal rotation stretching technique for the shoulder, however, no study has investigated this stretch until this study. The prone stretching technique utilized in the current study is similar to the technique explained by Johansen and colleagues. In their descriptive essay, the authors thoroughly explain the exercise as a prone stretch that involves passive assistance from the clinician. The scapula is easily stabilized as it protrudes posteriorly while the humerus rotates internally. While the author’s did not evaluate this procedure using an experimental design they presented a modified technique that should improve the manner in which clinicians treat compromised internal ROM in the shoulder. Johansen et al. (1995), explained the need for future research to evaluate this novel technique, however, to date there has been no study to investigate its effectiveness, therefore the framework of this section explains research that has investigated other intervention techniques that have been empirically tested.
The cross-body shoulder stretch is a self-stretching technique whereby the subject flexes the shoulder to approximately ninety degrees, then pulls the involved extremity across the body in a horizontal adduction movement, stretching the posterior shoulder musculature (Prentice, 1990). This self-stretch, which serves as the control treatment for the current study, has been explained thoroughly in the literature; however, a more recent study compared this stretch to another internal rotation self-stretching technique, the sleeper stretch (McClure et al., 2007). This study investigated fifty-four participants and found that the cross-body stretch participants experienced a clinically significant improvement in internal rotation; the group mean and standard deviation for cross-body stretch (20.0° ± 12.9°) compared to the sleeper stretch group (12.4° ± 10.4°), and the no stretching control group (5.9° ± 9.4°). In both of the stretching groups, all participants were screened to meet an equal or greater than 10° between shoulders difference in internal rotation, and were asked to perform the technique by holding the stretch 30 seconds 5 times daily.

The cross-body stretch is the recommended technique in many rehabilitation programs for improving or restoring internal rotation (Meister, 2000; Wilk, Meister, & Andrews, 2002). Arguments against this technique have focused on the inability of a patient to control for scapular mobility, which is why some practitioners choose other self-stretch methods (Laudner, Sipes, & Wilson, 2008). Also, in a cadaveric study of nine maintained shoulders, researchers found that the cross-body stretch may actually stretch the posterior deltoid more than the posterior capsule of the gleno-humeral joint (Muraki, Aoki, Uchiyama, Murakami, & Miyamoto, 2006).
The sleeper stretch, which is another common self-stretching technique to improve internal rotation has been purported as an appropriate exercise to improve range of motion deficits as well (Burkhart et al., 2003b; Laudner et al., 2008). Laudner and fellow researchers (2008) determined that the sleeper stretch improved internal rotation to a statistically significant level (p= 0.003) with a mean improvement of 3.1° compared to no change in a non-stretched control group (p= 0.62). Pre- and post-test measurements in the study group were taken before and after participants performed 3 sets of a 30 second stretch, which Laudner and colleagues (2008) noted may have elucidated an acute stretch reflex and not a permanent elongation of tissue.

Manual therapy is commonly used by a clinician to restore or improve ROM limitations, or contractures in joints and soft tissue. In addition to passive stretching, common examples of manual therapy include joint mobilization, therapeutic massage, and myofascial release. Although there is far less literature to support the use of these other manual techniques compared to stretching, it is important to review these options for the potential effect they have on improving internal rotation specific to the shoulder.

Manual therapy techniques such as therapeutic massage and trigger point therapy, as well as myofascial release, are typically reserved for patients with posterior shoulder tightness associated with pain (Poser & Casonato, 2008). It appears that prior studies to evaluate techniques such as those mentioned above demonstrate outcomes related to symptomatic subjects only. For example, Poser and Casanato (2008) investigated one patient with shoulder pain who was treated with 3 treatment sessions lasting 10 minutes each, focusing solely on the posterior rotator cuff musculature. Pre- and post-test internal rotation measurements, 68° and 88° respectively, resulted in a 20° improvement. There
was no report of a timeline for this treatment or measurement timing. In a statement paper to explain management of shoulder pain in swimmers, Blanch (2004) discussed the potential effectiveness of using trigger point therapies and massage of the infraspinatus muscle to improve internal rotation in the short term; however, no data are available to support this finding (Blanch, 2004). In another case study, one symptomatic patient with posterior shoulder myofascial pain was treated with the spray and stretch technique, whereby a topical anesthetic is administered to painful areas and then subsequently stretched, and after two treatments the patient reported decreased pain and markedly improved range of motion (Nielsen, 1981).

A similarity that exists between these reports is that all of the authors are of the opinion that posterior musculature tightness is the causative factor for reduced internal rotation and shoulder pain. In addition, all three of the previously mentioned articles refer to stretching as a supplementary intervention to aid in treatment. At least two of these studies discuss that this type of manual therapy is useful while treating both pain and range of motion deficits in the shoulder (Blanch, 2004; Poser & Casonato, 2008).

Joint mobilization of the glenohumeral joint has enjoyed more support from the literature as a technique employed in manual therapy intervention for unilateral internal rotation deficit. The literature has produced a theoretical and empirical basis for the effect joint mobilization has on the physiologic and mechanical soft tissue responses upon which to build an experimental study (Cyriax, 1975; Threlkeld, 1992). Goldman and Sauers (2004) compared posterior shoulder mobilization treatment to proprioceptive neuromuscular stretching (PNF) interventions in 31 asymptomatic professional baseball players. Each group underwent three sets of either PNF or grade 3 and 4 joint
mobilizations and internal rotation measurements were taken immediately before and after the intervention. The acute improvements observed for internal rotation were found to be equally effective (p= 0.133) at increasing internal rotation range of motion and posterior shoulder tightness.

In a similar but more recent study that evaluated more than the acute effects of a manual therapy treatment, Manske and colleagues separated 39 asymptomatic college-age participants into two groups: cross-body stretching only, and cross-body stretching plus posterior joint mobilizations (Manske, Meschke, Porter, Smith, & Reiman, 2010). Participants were asked to perform the cross body stretch at least 15 times over a 4 week period, while participants in the clinician-assisted group received mobilization treatments at least 2 times per week over the same period. Manske et al. reported significant changes in IR ROM within both groups from pre- to post-measurement at 4 weeks. The mean improvement for the stretching only group was 15.5° ± 11.7°, and for the stretching with mobilization group, 19° ± 12.7°. Although these findings represent a substantial increase in internal ROM for both groups, there was no statistically significant difference between groups. According to this study, both manual therapy techniques, used together or in conjunction, appear to increase internal rotation in ROM deficient participants.

From these reports, it appears that manual therapy is an appropriate clinical choice for improving both asymptomatic and symptomatic internal rotation restrictions in athletic and general populations. Through this review, it is also clear that unilateral internal ROM restrictions are commonly found in overhead athletes, although many of the references cited here have investigated general population participants as well and have found similar restrictions to ROM.
Surgical Intervention

In cases where conservative treatment of symptomatic internal rotation deficit does not respond favorably to conservative treatment, surgical treatment may become an option for the patient. Upon arthroscopic evaluation of the pathologic shoulder experiencing internal rotation deficit, typically associated with impingement syndrome, most surgeons report thickening of the posterior shoulder capsule, which creates a posterior capsule contracture (Bach, 2006; Ticker et al., 2000; Yoneda et al., 2006).

Bach (2006), Ticker et al. (2000), and Yoneda et al. (2006) have all reported the use of an arthroscopic capsular release, or capsulotomy in patients that do not respond to conservative treatment. Yoneda et al. reported on 16 overhead athletes who underwent this procedure with a minimum of a 2 year follow up. Yoneda et al. reported a group average of 21° reduction in the ROM restriction from pre-surgical measurement to the two-year follow up and 87% of athletes returned to pre-intervention levels of activity. This research also explained that all subjects participated in a supervised post-surgical rehabilitation program which included passive stretching techniques. Specific ROM data was not reported in this study.

In a similar study, Ticker and associates (2000) found thickening of the posterior capsule in 9 (n = 9) general population patients who underwent posterior capsular release for shoulder pathology, including internal rotation deficit. All patients had failed to improve with conservative treatment alone, but after arthroscopic release and a subsequent supervised rehabilitation program, which included passive stretching, the authors reported a statistically significant (p ≤ .01) group internal rotation improvement from 10° before surgery to 48° (37° improvement) post-surgery and rehabilitation at an
average of 18 months follow up for all patients. Statistical interpretations of SD and effect size were not reported in this study.

Although speculation still exists as to the exact cause of the restrictions in internal rotation ROM in dominant versus non-dominant shoulders, many of the research studies cited earlier in this section have concluded conditions like GIRD or PST will likely cascade into symptomatic pathology when the deficit is not corrected. Likewise, a majority of the research in this area has concluded that a stretching program is advisable to help restore or maintain asymptomatic rotational deficiencies, particularly in overhead athletes (Beach et al., 1992; Borsa et al., 2005; Burkhart and Morgan, 1998; Burkhart, Morgan, and Kibler, 2003b; Kibler and Chandler, 2003; Meister, 2000; Pappas et al., 1985; Ticker et al., 2000; Tyler et al., 2010; Warner et al., 1990; Wilk et al., 2002).

With a large body of research to support the conservative treatment of internal rotation restrictions in dominant shoulders, it is clear that the proposed study to evaluate the effects of a novel prone stretching technique to improve or restore range of motion is both timely and suitable for building on the work of others in this area.

Our research question was, “Is the prone-passive internal rotation stretching technique effective at increasing range of motion (ROM) in the GH joint compared to a self-stretching technique?” We hypothesized that differences exist between a novel prone stretching technique and the cross-body, self-stretching technique to increase internal rotation in the GH joint.

This study evaluated the effectiveness of a modified prone-passive IR stretching technique, similarly described by Johansen and colleagues (1995), compared to the cross-body self-stretching technique, which is previously described as an appropriate method of
improving IR in participants exhibiting signs of GIRD and or PST (McClure et al., 2007). The passive stretching technique has been described once in the literature but never tested.
CHAPTER II: METHODS

The paradigm (Figure 2.1) employed for this study is based on a prospective, randomized experimental design to measure the effectiveness of the proposed stretching techniques in individuals demonstrating unilateral differences of greater than or equal to 10 degrees in internal rotation ROM of the GH joint based on pre-test measures. Participants were recruited for evaluation of unilateral differences with convenience as a selection tool using athletes that participate in overhead dominant sports. Participants that met the threshold for determining a unilateral internal ROM difference were randomly assigned to control and experimental groups, into one of two interventions: self-stretching (control group) or prone-passive stretching (experimental group).

**Figure 2.1 Experimental Design**
Participants

This study recruited competitive athletes participating in intercollegiate or recreational level upper extremity dominant sports. Participants were recruited from student populations of both public and private colleges in Southwest Idaho based on the presence of asymptomatic unilateral internal rotation limitation of at least 10°; therefore, participants were recruited via a convenience sample from those that display the condition described as PST or GIRD. Prior investigation into GIRD and PST has demonstrated its prevalence in a variety of overhead sports, but specifically baseball (Borsa et al., 2005; Burkhart et al., 2003a; Ellenbecker et al., 2002).

Volunteer participants were excluded from this study if they had a recent history of any of the following: shoulder surgery, shoulder injury requiring treatment in the past year, or shoulder pain at the time of study. It was assumed that participants may be actively participating in a supervised strength and conditioning program that does not include an existing internal rotation stretching program, such as the techniques investigated in this study.

Participation in this study was voluntary and those recruited to participate were informed of the purpose of the study, potential benefits of participation in either group, as well as potential side effects of internal rotation stretching of the GH joint. All prospective subjects read and signed an informed consent (Appendix D). Thirty-five subjects completed a shoulder medical history and demographic information form prior to participation (Appendix E). One subject did not complete the study; therefore, the total sample size was 34 participants. Participants were randomly assigned to one of two groups: Control n= 17 (9 male, 8 female), Experimental n= 17 (10 male, 7 female). The
age range for all participants was 18-29 years old with a mean age of 20.3 years old ± 2.54. The Institutional Review Board (IRB) at Boise State University approved this study.

**Measurement / Procedure**

This study was conducted under the auspices of the Athletic Training/Motor Control Research Laboratory at Boise State University. For this study, passive internal and external ROM was measured. Measurement was performed using a digital inclinometer (GX products digital inclinometer: Hong Kong, China), which has been found useful in prior studies that evaluated ROM (Moore, Laudner, McLoda, & Shaffer, 2011; Awan, Smith, & Boone, 2002; de Winter, Heemskerk, Terwee, et al., 2004). For this study, we attached the magnetic bottom of the inclinometer to a 0.125” x 1.5” x 6.5” industrial grade steel plate, which was affixed to the outer surface of a flat, medium-sized, soccer shin guard (Vizari: Paramount, CA, USA), figure 2.2.

![Measurement Instrument](image)

**Figure 2.2 Measurement Instrument**

Baseline and follow up measurements of GH ROM using this device were performed by the same trained research assistant to allow for blinding of the tester and
principal investigator to grouping and measurements, respectively. Baseline and follow-up measurements were done with the participant in the supine position, on the same treatment table, with the GH joint abducted to 90° and the elbow flexed to 90°. This position has previously been demonstrated as an appropriate measure of ROM with the scapulo-thoracic articulation immobilized (Kibler, 1998; Ellenbecker et al., 1996). The modified inclinometer was fastened securely to the forearm, using the ulnar styloid and olecranon process as bony landmarks to centrally position the apparatus on the extremity. Similar measurement techniques using these parameters while using an inclinometer have been reported by Manske et al. (2010). Prior to measurements, the tester passively rotated the extremity through the total arc to help the participant relax and become accustomed to the testing motion and endpoints of motion.

A 0.5" section of medium density memory foam (Econoline Industries: LaPlume, CA, USA) was placed between the humerus and treatment table. This was done for two reasons: 1. the foam elevates the humeral head to perpendicular alignment on the glenoid fossa, and 2. the memory foam allows the upper arm to rotate freely while keeping the desired alignment of the elbow and shoulder at 90°. The forearm was secured in a vertical position of GH neutral, then passively moved in external and internal motion with one hand on the participant’s wrist and another stabilizing the elbow. The end range of GH motion was identified when a firm endpoint is noted, and/or when compensatory movement caused by the scapula flexing forward on the thorax is observed or palpated in the shoulder girdle by the tester (Figure 2.3). The tester recorded the measurement once the end range of motion was established and held for approximately three seconds (Appendix E). The benefit of using this passive technique was that it helped to remove
the possibility of muscle insufficiency as a cause of motion difference or compensatory joint movement in the scapulo-thoracic region.

**Figure 2.3  Internal (left) and External (right) ROM Measurement Technique**

Baseline measurements were taken no sooner than 24 hours prior to the initial stretching intervention and follow-up measurements taken no later than 48 hours after the final intervention session was completed. Measurements were taken three times for both internal and external ROM for pre- and post-testing (Appendix C).

**Intra- and Inter-Tester Reliability**

Prior to the study, intra- and inter-tester reliability analysis was performed for IR and ER ROM on 22 dominant and non-dominant shoulders. The tester was blinded to the measurement readout while a research assistant recorded each score. A Cochran’s alpha was employed to determine the reliability of these measurements; analysis revealed strong reliability in the four different measures (Table 2.1).
## Table 2.1  Intra- and Inter-Tester Reliability of Measurements

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Intra Tester 1</th>
<th>Intra Tester 2</th>
<th>Inter-Tester</th>
<th>SEM*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dominant IR</td>
<td>0.96</td>
<td>0.97</td>
<td>0.90</td>
<td>2.23</td>
</tr>
<tr>
<td>Non-Dom IR</td>
<td>0.98</td>
<td>0.98</td>
<td>0.94</td>
<td>2.22</td>
</tr>
<tr>
<td>Dominant ER</td>
<td>0.97</td>
<td>0.96</td>
<td>0.95</td>
<td>2.22</td>
</tr>
<tr>
<td>Non-Dom ER</td>
<td>0.97</td>
<td>0.97</td>
<td>0.88</td>
<td>1.57</td>
</tr>
</tbody>
</table>

*Note: Cochran’s alpha Intra- and Inter-Tester co-efficients.*

* Standard Error of Mean calculated for Inter-Tester reliability.

## Intervention

The control group performed the cross-body stretch as previously described by McClure et al. (2007) (Figure 2.4). Control group participants were given instructions with picture demonstrations and the principal investigator also explained and demonstrated this technique to each participant (Appendix A). For this stretch, the participant statically holds the position for thirty seconds for a total of five repetitions three times per week. All participants were advised to stretch to the point of normal stretch sensation, which has been described as mild discomfort (McClure et al., 2007). Mild discomfort is explained as moderate tension felt in the posterior shoulder without associated pain.
Figure 2.4  Experimental (left), Control (right) Stretching Techniques

The experimental group underwent the prone-passive stretching technique previously described in the literature; however, modifications to this technique have been made to accommodate participants exhibiting restricted ROM (Johansen et al., 1995). The modified technique requires the research assistant to hold the stretch compared to the use of a bolster as described in the Johansen et al. (1995) report (Figure 2.4). This intervention was conducted by a research assistant or the principal investigator. Assistants were formally trained in administering the stretch. An instructional form with pictures of the technique, detailed instructions, and an instructional video were given to each assistant performing this stretch (Appendix B). To assure the proper technique was performed, the principal investigator taught the technique directly to research assistants. The stretch was repeated a total of five times during each session and passively held for thirty seconds each time. Participants were given up to a minute break between each stretch. Specific attention to GH endpoints, shoulder and elbow alignment, discomfort, and any compensatory movements of the shoulder girdle were monitored during the administration of the stretch. To standardize treatments between groups, study participants were asked to perform the passive stretch three times per week over a 28-day
period. Therefore, all participants were asked to perform 12 separate stretching sessions. Treatment sessions were recorded by self-stretch participants and the research assistant working with the experimental group participants to emphasize adherence to the requirements for this study (Appendices G and H).
CHAPTER III: RESULTS

Analysis

A 2x2 repeated measure design was employed to analyze all mean pre-test and post-test total measurements while an independent t-test was used to analyze all mean gain scores as previously discussed by Campbell and Stanley (1963). For this study, gain scores were calculated by subtracting pre-test from post-test total gain mean. Correlation analysis was performed using the Pearson product moment statistic. Inter- and intra-tester reliability was demonstrated by Cochran’s Alpha. Statistical significance was defined a priori \( \leq 0.05 \). Analysis of effect size was performed with an estimated partial eta squared for all primary measures. Data was analyzed using SPSS 19.0 (SPSS Inc., a division of IBM: Armonk, NY).

Participant Descriptives

A Levene’s test for homogeneity of variance demonstrated no statistical differences between the two groups in regard to mean age, gender, years of overhead sports participation, and adherence to the treatment methodology (see Tables 3.1-3.4). As well, all measurement variables were homogenous during pre-test measures. On hundred-thirteen volunteers were screened for a \( \geq 10^\circ \) IR deficit between dominant and non-dominant shoulders of which 35 met the inclusion criteria. All volunteers reported current participation in either a sponsored college or university intercollegiate athletic or recreational sport program, which included baseball (n = 15), softball (n = 9), recreational
athletes (n = 8), and swimming (n = 2). All participants reported being healthy athletes with no history of shoulder injury or surgery in the past year. None of the participants reported having current shoulder pain on the medical history. One participant in the experimental group did not complete the study for unknown reasons. There were equal subject numbers between the groups (control n= 17 [9 male, 8 female], experimental n= 17 [10 male, 7 female]). Descriptive information for gender, age, and dominant shoulder is categorized by group in Table 3.1. Much like the general population, the study population included far fewer left-handed athletes.

<table>
<thead>
<tr>
<th>Table 3.1</th>
<th>Participant Descriptives</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group</strong></td>
<td><strong>Male</strong></td>
</tr>
<tr>
<td>Experimental</td>
<td>9</td>
</tr>
<tr>
<td>Control</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
</tr>
</tbody>
</table>

*Note: Age reported as mean (standard deviation)*

Table 3.2 further categorizes participation by sport and total years of experience in the respective activity.

<table>
<thead>
<tr>
<th>Table 3.2</th>
<th>Participant by Sport</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group</strong></td>
<td><strong>Baseball</strong></td>
</tr>
<tr>
<td>Experimental</td>
<td>6</td>
</tr>
<tr>
<td>Control</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
</tr>
</tbody>
</table>

*Note: Years of Experience reported as mean (standard deviation)*
Table 3.3 reflects adherence to the treatment for each group. Time in days participants took, on average, to have a post-measurement taken once enrolled in the study as well as the mean number of stretching sessions were reported.

Table 3.3  Participant Time from Pre to Post Measurement and Adherence to Stretching Schedule

<table>
<thead>
<tr>
<th>Group</th>
<th>Time To Post Measure (days)</th>
<th># of Sessions Reported</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experimental</td>
<td>29.18 (2.31)</td>
<td>12.00 (0.00)</td>
</tr>
<tr>
<td>Control</td>
<td>29.27 (2.44)</td>
<td>11.47 (1.09)</td>
</tr>
<tr>
<td>Total</td>
<td>29.35 (2.34)</td>
<td>11.70 (0.08)</td>
</tr>
</tbody>
</table>

*Note: Time and Sessions reported as mean (standard deviation)*

Table 3.4 summarizes sport participation and corresponding prevalence rates of IR deficit for all volunteers that were screened.

Table 3.4  ≥ 10° IR Deficit by Number of Sport Participants Screened

<table>
<thead>
<tr>
<th>Sport</th>
<th># Screened</th>
<th>Enrolled</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseball</td>
<td>33</td>
<td>16 *</td>
<td>48.0 %</td>
</tr>
<tr>
<td>Softball</td>
<td>25</td>
<td>9</td>
<td>36.0 %</td>
</tr>
<tr>
<td>Swimming</td>
<td>17</td>
<td>2</td>
<td>11.7 %</td>
</tr>
<tr>
<td>Recreational</td>
<td>38</td>
<td>8</td>
<td>21.0 %</td>
</tr>
<tr>
<td>Total</td>
<td>113</td>
<td>35</td>
<td>31.0 %</td>
</tr>
</tbody>
</table>

*One participant in this sport did not complete the study

Dominant Shoulder Internal Rotation Range of Motion (Dom IR ROM)

Analysis of dominant shoulder internal rotation revealed no significant effect on gleno-humeral IR ROM gain (13.24° ± 7.78°) compared to the control group (8.47° ± 8.78°, p = 0.104). See Figure 3.1 below.
Figure 3.1  Dominant IR ROM Mean Gain

Dominant IR ROM pre-test measurements for the experimental group (60.36° ± 13.58°) and control (58.42° ± 7.23°) are represented in Figure 3.2 below.

Figure 3.2  Dominant IR ROM Mean Pre-Test Values
Post-test experimental group IR ROM ($73.60^\circ \pm 12.79^\circ$) and control measures ($66.89^\circ \pm 10.62^\circ$) are illustrated in Figure 3.3 below.

**Figure 3.3  Dominant IR ROM Mean Post-Test Values**

**Internal Rotation Deficit (IR Deficit)**

Analysis of IR deficit between dominant and non-dominant shoulders was calculated by finding the difference between pre- and post-test dominant shoulder IR ROM and pre- and post-test, non-dominant shoulder IR ROM. The observed experimental group gain scores ($-12.64^\circ \pm 11.49^\circ$) as compared to control ($-9.13^\circ \pm 8.33^\circ$, $p = 0.441$) are illustrated in Figure 3.4.
The pre-test mean IR deficit between groups were similar. Experimental group deficit was $(17.22^\circ \pm 6.76^\circ)$ compared to control group $(17.02^\circ \pm 3.63^\circ)$ (represented in Figure 3.5).

**Figure 3.4** IR Deficit Mean Gain Values

The pre-test mean IR deficit between groups were similar. Experimental group deficit was $(17.22^\circ \pm 6.76^\circ)$ compared to control group $(17.02^\circ \pm 3.63^\circ)$ (represented in Figure 3.5).
Figure 3.5. IR Deficit Pre-Test Mean Values

Observed post-test measurements in the experimental group were \((4.58^\circ \pm 8.70^\circ)\), as compared to the control group \((7.89^\circ \pm 8.33^\circ)\) (Figure 3.6).

Figure 3.6. IR Deficit Post-Test Mean Values
Total Motion of the Gleno-Humeral Joint

Similar analysis was done to compare differences for total motion between groups. This calculation is the sum between pre- and post-test dominant shoulder IR ROM and ER ROM measurements. Observed mean gain scores for the experimental group (14.84° ± 11.27) were compared to the control group (9.97° ± 11.99°, p = 0.232); Figure 3.7. Subjects in the experimental group (170.22° ± 14.11°) exhibited slightly more total motion than the control group (165.60° ± 12.73°) in pre-test measurements (Figure 3.8). Observed post-
Figure 3.8  Total Motion Pre-Test Values

test total motion for the experimental group (185.05° ± 14.79°) as compared to the control group (175.57° ± 18.48°); Figure 3.9.

Figure 3.9  Total Motion Post-Test Values
Measurements and gain scores for all measurement pre- and post-tests, are categorized below in Tables 3.5 and 3.6, respectively. The level of significance for each measurement comparison is referenced in Table 3.6.

**Table 3.5 Pre- and Post- Test measurements.**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Test</th>
<th>Experimental</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dom IR ROM</td>
<td>Pre</td>
<td>60.36 (13.58)</td>
<td>58.42 (7.23)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>73.60 (12.79)</td>
<td>66.89 (10.62)</td>
</tr>
<tr>
<td>Non Dom IR ROM</td>
<td>Pre</td>
<td>77.58 (11.76)</td>
<td>75.44 (7.40)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>78.18 (15.03)</td>
<td>74.78 (9.29)</td>
</tr>
<tr>
<td>Dom ER ROM</td>
<td>Pre</td>
<td>109.86 (8.98)</td>
<td>107.18 (12.25)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>111.45 (7.48)</td>
<td>108.67 (9.92)</td>
</tr>
<tr>
<td>Non Dom ER ROM</td>
<td>Pre</td>
<td>99.45 (9.10)</td>
<td>100.07 (6.92)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>100.10 (7.42)</td>
<td>100.34 (5.99)</td>
</tr>
<tr>
<td>IR Deficit ROM</td>
<td>Pre</td>
<td>17.22 (6.76)</td>
<td>17.02 (3.63)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>4.58 (8.70)</td>
<td>7.89 (8.33)</td>
</tr>
<tr>
<td>Dominant Total Motion</td>
<td>Pre</td>
<td>170.22 (14.11)</td>
<td>165.60 (12.73)</td>
</tr>
<tr>
<td></td>
<td>Post</td>
<td>185.05 (14.79)</td>
<td>175.57 (18.48)</td>
</tr>
</tbody>
</table>

*Note: Means (Standard Deviation) in degrees*

Dom = Dominant Shoulder
Non Dom = Non-Dominant Shoulder

To illustrate the group mean change in each group for all measurements, a gains score was calculated by subtracting total post-test gain from total pre-test gain (Table 3.6). Effect size was calculated for each measurement to demonstrate the strength of relationships between the groups in each measure. For reference, the level of relationship for partial eta squared is 0.01 = small, 0.06 = medium, and 0.14 = large.
Table 3.6  Gain Scores for All Measurements (Post-Test and Pre-Test).

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Experimental Gain</th>
<th>Control Gain</th>
<th>Sig. level p.</th>
<th>Effect Size *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dominant IR</td>
<td>13.24 (7.78)</td>
<td>8.47 (8.78)</td>
<td>0.104</td>
<td>0.081</td>
</tr>
<tr>
<td>Non Dom. IR</td>
<td>0.60 (8.32)</td>
<td>-0.65 (6.96)</td>
<td>0.636</td>
<td>0.007</td>
</tr>
<tr>
<td>Dominant ER</td>
<td>1.60 (7.45)</td>
<td>1.50 (7.52)</td>
<td>0.971</td>
<td>0.000</td>
</tr>
<tr>
<td>Non Dom. ER</td>
<td>0.65 (4.17)</td>
<td>0.27 (5.23)</td>
<td>0.815</td>
<td>0.002</td>
</tr>
<tr>
<td>IR Deficit</td>
<td>-12.64 (11.49)</td>
<td>-9.13 (8.33)</td>
<td>0.441</td>
<td>0.032</td>
</tr>
<tr>
<td>Total Motion</td>
<td>14.84 (11.27)</td>
<td>9.97 (11.99)</td>
<td>0.232</td>
<td>0.044</td>
</tr>
</tbody>
</table>

Note: Mean gain (Standard Deviation) in degrees  
* Effect Size is estimated partial eta squared

Within groups analysis demonstrated the following results: 100% (n = 17) of the prone-passive participants increased IR ROM compared to 70% (n= 12) of the cross-body subjects; 100% (n = 34) of the participants in both groups decreased the amount of IR deficit; and, 95% (n = 16) of the prone-passive participants improved total degrees of motion compared to 82% (n = 14) in the cross-body treatment group.

**IR ROM, IR Deficit, and Total Motion in Males and Females**

Gain score analysis between genders did not reveal any statistically significant differences between genders. Tables 3.7 and 3.8 illustrate the mean gain scores observed in response to treatment between males and females in both groups.
Table 3.7  Gain Score for Dominant IR ROM, IR Deficit, and Total Motion, Males.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Experimental Gain</th>
<th>Control Gain</th>
<th>Sig. level p.</th>
</tr>
</thead>
<tbody>
<tr>
<td>IR ROM</td>
<td>14.39 (10.37)</td>
<td>6.71 (8.01)</td>
<td>0.087</td>
</tr>
<tr>
<td>IR Deficit</td>
<td>-17.75 (11.58)</td>
<td>-9.25 (7.16)</td>
<td>0.174</td>
</tr>
<tr>
<td>Total Motion</td>
<td>16.12 (12.63)</td>
<td>6.13 (11.34)</td>
<td>0.087</td>
</tr>
</tbody>
</table>

*Note: Mean gain (Standard Deviation) in degrees*

Table 3.8 references the mean gain score measurements for female participants in each group.

Table 3.8  Gain Score for Dominant IR ROM, IR Deficit, and Total Motion, Females.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Experimental Gain</th>
<th>Control Gain</th>
<th>Sig. level p.</th>
</tr>
</thead>
<tbody>
<tr>
<td>IR ROM</td>
<td>11.95 (3.47)</td>
<td>10.99 (9.84)</td>
<td>0.813</td>
</tr>
<tr>
<td>IR Deficit</td>
<td>-6.89 (8.77)</td>
<td>-8.95 (10.39)</td>
<td>0.852</td>
</tr>
<tr>
<td>Total Motion</td>
<td>13.39 (10.16)</td>
<td>15.46 (11.42)</td>
<td>0.719</td>
</tr>
</tbody>
</table>

*Note: Mean gain (Standard Deviation) in degrees*

**IR and ER Correlation**

A Pearson statistical analysis did not demonstrate a significant correlation between all dominant IR and ER gain scores (r = 0.082, p =.645). A scatterplot with a linear regression line (Figure 3.10) demonstrates a lack of correlation between the two variables in regard to change in range of motion.
Figure 3.10  Scatterplot with Linear Regression Line Demonstrating a lack of Correlation Between Dominant IR and ER Change
CHAPTER IV: DISCUSSION

The findings of this study have demonstrated the effects of the novel prone-passive stretching technique, and the cross-body technique on unilaterally restricted shoulder internal rotation in an overhead athlete population. We have introduced a previously untested treatment technique to the body of work in the area of conservative treatment for internal rotation deficit and/or posterior shoulder tightness.

Response to Intervention

At least two previous studies have demonstrated statistical significance for specific variables during the investigation of the cross-body technique (Manske et al., 2010; McClure et al., 2007). While our study did not achieve statistical significance between the prone-passive and cross-body stretching techniques, a determination of clinical significance could be inferred through the observed increase in IR ROM in the mean gain comparisons for the prone-passive subjects. Both the Manske et al. and McClure et al. studies refer to clinical significance and the application of the self-stretching technique as an appropriate method for improving IR restriction. While both of these studies demonstrated significance of an experimental group compared to a true control (no treatment applied), the purpose of this study was to determine if a novel stretching technique was more effective than a previously described clinical technique.
Gender Comparisons

Males responded more favorably than females to prone-passive treatment (Tables 3.7 and 3.8). Between groups, females did not respond differently depending on treatment group, whereas males increased IR and decreased IR deficit more in the prone-passive group than the cross body. The comparisons observed between genders is not likely directly related to type of stretching technique, but more so a reflection of the prevalence of IR deficit in baseball (Table 3.4). In addition to the increased prevalence of IR deficit in males screened for inclusion in our study, males also demonstrated increased degrees of deficit compared to female participants. Thus, males had more room for improvement than females.

What we observed in regard to gender differences is heavily substantiated through previous research and explained earlier in this paper. The reasons male overhead athletes are more prone to GIRD is not completely understood. However, prior research that has studied the tensile forces acting on the shoulder in the overhand baseball throwing motion, specifically in pitchers, compared to those observed in the sport of softball are not as substantial because overhead repetition is not comparable to that of the baseball pitcher. Prior research suggests that this more forceful, ballistic motion and the associated deceleration, is a likely contributor to the prevalence of GIRD in male overhead athletes compared to their female counterparts. It is plausible to suggest that both the static (capsular) and dynamic (muscular) deceleration forces needed to overcome the inertia of the accelerated extremity are greater in males due to the increased mass of the average male arm compared to that of the average female. Consequently, these forces
produce significant adaptive posterior shoulder muscle and capsule-ligamentous contracture, which likely leads to the observed higher prevalence of GIRD in males.

**Differing Views on Total Motion in an IR Deficit Sample**

Many have described a concomitant increase in external rotation (ER) when limitations to IR exist, with total motion remaining normal compared to the non-dominant shoulder. Others refer to this relationship as a shift toward ER (Borsa et al., 2005; Wilk et al., 2011). The assumption then may be that total motion remains the same even with a decrease in IR. Our findings do not concur with or support this assumption; rather, in our study, total motion was improved in the dominant shoulder after treatment for unilateral IR deficit. We observed increases to IR in the prone-passive group compared to the cross-body participants, while ER remained unchanged regardless of treatment. The total motion concept states that an equal amount of IR and ER exists in the dominant and non-dominant shoulders, even in cases of GIRD (Wilk et al., 2002). Our results suggest there are differences in the degrees of total motion when IR ROM has been improved, producing an increase in total motion in an individual with IR deficit. We speculate that dominant shoulder total motion may be increased in healthy, non-GIRD overhead athletes compared to the non-dominant shoulder. These findings lead us to believe that soft tissue contracture, primarily in the posterior shoulder musculature, leads to GIRD and/or posterior shoulder tightness (PST).

**Capsule or Muscle?**

When a clinician establishes a treatment protocol to deal with a condition such as GIRD or PST, it is assumed that an exact cause is already known. We still do not know
the exact reason why overhead throwers tend to gravitate toward unilateral internal rotation deficit, and probably from early on in their sport careers, but both prior and recent research is focusing on two soft tissue structures as the causative factor - the posterior gleno-humeral capsule and posterior shoulder musculature (Borstad & Dashotter, 2011). This recent cadaveric study has re-visited the notion of the capsule as the primary cause of GIRD, but certainly has left the door open to suggest muscular tightness as another contributor.

Collagenous tissue will elongate if tension is applied long enough as discussed in a seminal reference to the creep theory (Rigby, Harai, Spikes, & Eyring, 1959); however knowledge of the creep theory does not suggest a causative factor of GIRD. To help the clinician determine which structure of the posterior shoulder should be the focus of treatment, one should determine the level of stability both capsule and muscle provide to the glenohumeral joint. The capsule surrounding the shoulder joint is known as a static stabilizer while muscles, specifically the infraspinatus and teres minor of the posterior rotator cuff, are referred to as dynamic stabilizers of the joint. Each type of tissue provides stability to the shoulder joint and this is widely variable depending on the individual. One may think of capsular tissue as a primary stabilizer to the gleno-humeral joint, while the posterior rotator cuff may be thought of as a secondary stabilizing force to anterior translation, or internal rotation movements of the joint. Though the capsule has the ability to stretch, as seen in cases of traumatic injury such as dislocation, it does not necessarily have the ability to stretch like a musculotendinous unit does and also retain the stabilizing quality when contracted. Capsular tissue tends to be viscous but does not possess the same visco-elastic properties of skeletal muscle. Another way of thinking
about the differences between the two tissues is in the contractility properties of each. A capsule, to our knowledge, cannot be strengthened through therapeutic exercise like a muscle. Therefore, the musculotendinous units providing dynamic stabilization of the posterior shoulder may provide more stability to the gleno-humeral joint, especially in overhead dominant sports. This implies that a musculotendinous unit undergoes more tensile forces that lead to contracture than the capsule. Manske and colleagues (2010) demonstrated IR restrictions started to return as soon as four weeks after IR stretching and/or joint mobilization treatment stopped. This observation suggests that the viscoelastic property of muscle and the functional shortening thereof is the primary contributor of GIRD and/or PST in an otherwise healthy overhead athlete. Therefore, both soft tissues in the posterior shoulder girdle should be the focus of a conservative treatment protocol.

While we did not perform a follow-up measurement in this study, we did emphasize to participants the importance of continuing a maintenance internal rotation stretching program. Though IR deficits were decreased in this study, it would be illogical to think that ROM restrictions would not return in the absence of a maintenance stretching program for overhead athletes.

**Parameters of the Stretching Technique**

Our treatment parameters were based on prior research that investigated the effects of shoulder manual therapy. Each participant was asked to perform 12 stretching sessions over a 28-day period. Reported adherence was very good in this study (Table 3.3). Self-reporting in the cross-body stretching group could have potentially lead to less improvement in IR ROM compared to the prone-passive group in this study.
Creep theory of collagenous tissue emphasizes the importance of repeated stretching or, more simply, movement within soft tissue in order to maintain elasticity (Rigby et al., 1959). This theory can be thought of as a change in length proportional to the amount of strain applied over time. An interesting observation was noted by the clinicians providing the prone-passive stretching technique in this study. Un-measured improvements to IR ROM were noticed after only 4-6 treatment sessions in the experimental group. An obvious explanation to this occurrence would be the acute stretch response as demonstrated in many other studies investigating IR ROM restriction response to a single stretching treatment (Laudner et al., 2008; Moore et al., 2011). However, participants in the experimental group were not being treated on a daily basis; rather, treatments were administered three to four times per week. Even with multiple days in between sessions, we noticed a reduction to the previously encountered soft tissue limitations to IR ROM within the first 2 weeks of the program. While this observation was not measured or substantiated, this response to treatment may imply a shorter creep response, leading us to suggest a reduced number of treatments are needed to elicit improvements in IR ROM at the shoulder joint. This same phenomenon would also suggest a rather rapid reversal in tissue elongation in the absence of maintenance stretching.

**Tensile Forces of the Stretching Techniques**

The action of the prone-passive stretching technique creates a different type of reaction on the posterior gleno-humeral capsule as well as posterior shoulder musculature than does the self-stretch. The prone-passive stretch involves rotating of the shoulder compared to adduction of the joint during the cross-body technique. Rotational stress, or
torsion, is imparted on both capsular and musculotendinous tissues during the prone-passive technique. Torsional stress acts on the majority of the capsule surface area, which may allow for further stretching of the musculature when compared to the localized tensile forces in the posterior shoulder during the adduction movement of the cross-body stretch. The torsional stress may enhance elongation on multiple components of these tissues, facilitating a greater increase in ROM. A capsular twisting technique has previously been described as an effective treatment for adhesive capsulitis, a multidirectional restriction to shoulder motion (Henry, 1995).

The positioning of the patient in the prone position of this novel stretch allows other advantages compared to the self-stretch techniques proposed in this study. Instead of flexing the shoulder as in the sleeper stretch (whereby the humerus is forward flexed to 90° and somewhat horizontally adducted due to the weight of the upper body in a side lying position), the abducted position in the prone-passive stretch allows for greater capsular twist because the motions occurring in the shoulder girdle are occurring in both the sagittal and frontal planes (Hertling & Kessler, 2006). Flexing the arm, as in the sleeper stretch, may result in less capsular twisting, and therefore less tissue elongation when compared to the prone-passive technique.

With the patient lying prone, the clinician is able to produce significant forces upward against the restriction to IR in GIRD patients. In our study, this position was beneficial when stretching the participants that had significant limitations to IR. These participants, in particular, were more apt to mention mild stretching discomfort to the clinician applying the stretch. It was not uncommon for the participants with higher degrees of GIRD to mention some mild soreness in the posterior shoulder up to 24 hours
later. We would suggest this soreness may be a result of the breakdown of adhesions and/or contracture in significantly compromised posterior shoulder tissues since the soreness tended to dissipate rather quickly and did not compromise the treatment schedule.

**Importance of Maintaining Normal Glenohumeral Internal Rotation in Overhead Athletes**

It is well accepted that the prevalence of IR deficit in overhead athletes increases the risk of shoulder and elbow injuries to those individuals. A notable study has demonstrated this correlation, particularly in baseball athletes (Wilk et al., 2011). This type of study influences the preventative nature of strength and conditioning programs as well as athletic therapy protocols.

Common injuries observed in patients with GIRD tend to reflect the repetitive stresses the shoulder undergoes in overhead sports. For instance, rotator cuff tendonitis, sub-acromial impingement, and labrum injury are all reflective of the pathologic mechanics that are caused by restrictions to IR ROM in activities such as the overhand-throwing motion.

An in-depth knowledge of the throwing motion, such as the act of baseball pitching, alerts the clinician to the importance of the deceleration, or follow through phase of the motion. It is plausible to suggest that when experiencing restrictions to IR ROM, such as in GIRD, the humeral head is not able to decelerate within the tolerable mechanical restraints of the posterior rotator cuff, thereby causing injury due to a pathologic throwing motion. By maintaining or restoring IR, and thus normal
arthrokinematics in the throwing shoulder, one may be reducing the risk of the repetitive stress types of injuries.

**Conclusion**

This study investigated the effect of a conservative treatment on IR deficit in a population of overhead athletes as recommended through prior research. We have determined that the prone-passive stretching technique, when performed by a trained clinician, is at least as effective as the cross-body technique at improving glenohumeral internal rotation. The latter is a technique previously shown to be effective at treating GIRD. We suggest further study of the prone-passive technique with a larger sample size as this may demonstrate that this technique could be superior to the cross-body technique, as our data suggested a trend in this regard. We also believe follow-up measurements at periodic intervals, with and without continued treatment, might provide valuable insight for the treatment of GIRD. We believe the prone-passive stretching technique is likely a valuable tool for a clinician treating GIRD, and agree with others that IR stretching should be part of a consistent maintenance program for overhead athletes in the hope that it may decrease the incidence of injury.
REFERENCES


APPENDIX A

Participant Instructions for the Cross-Body Stretching Technique
Instructions for the cross body shoulder stretch

1. Stand with your feet about shoulder width apart and simply bring your dominant arm across your body at about chest-level. Lock that arm in place and stretch by using your other arm to do so.

2. Hold for a 30 second count. Repeat this stretch 5 separate times during each session.

3. For the study, please conduct this stretch three times per week over 4 weeks for a total of 12 sessions.

* You may or may not feel mild stretching discomfort on the back side of the shoulder. Please stretch the dominant shoulder only.
APPENDIX B

Instructions for the Prone-Passive Stretching Technique
Effects of the prone-passive stretching technique on glenohumeral internal rotation study - Boise State University

Instructions for the prone-passive shoulder stretch

1- Participant is positioned on a treatment table in the prone position with the treated extremity off of the table.

2- With the forearm in the pronated position and the elbow flexed to 90° while horizontally abducting the shoulder to 90° (neutral flexion/extension). The upper arm should now be depressed to the table. In some cases the upper arm may need to be off of the table to reduce friction.

3- The following steps must be done while continuously accounting for perpendicular alignment of the shoulder and elbow to the thorax (the shoulder must remain at 90° abduction).

3- Passively stabilize the scapula, as needed, by pressing the lower portion of the scapula toward the posterior thorax. This will be necessary as the arm is now passively internally rotated by the clinician to a firm end point (minimal to mild stretching discomfort should be expected).

4- At the end point of the stretch hold the position statically using manual support for 30 seconds. The stretch is repeated 5 times with appropriate rest given between each stretch.
APPENDIX C

Measurement Tester Procedures
Measurement Procedure for Measurement Tester

1. Prior to measurements medium density foam is placed under the humerus to help keep the humerus in a straight line. The tester will passively rotate the extremity through the total arc to help the participant relax and assimilate to the testing motion and endpoints of motion.

2. The forearm will be secured in a vertical position of GH neutral then passively moved in external and internal motion with one hand on the participant's wrist.

3. The end range of GH motion will be identified when a firm end point is noted, and/or when compensatory movement caused by the scapula flexing forward on the thorax is observed or palpated in the shoulder girdle by the tester.

- The benefit of using this passive technique helps to remove the possibility of muscle insufficiency as a cause of motion difference or compensatory joint movement in the scapulo-thoracic region.
APPENDIX D

Informed Consent
A. PURPOSE AND BACKGROUND

The purpose of the study you have been recruited to participate in is to determine the effectiveness a unique shoulder stretching technique has on shoulder range of motion, specifically internal range of motion.

Voluntary participation in this study will involve subjects in two different groups, a self stretching group and a prone passive stretching group. All participants will have shoulder range of motion measured before beginning the stretching and after the sessions are completed.

You are invited to participate in this study to determine the effectiveness of a unique shoulder stretching technique because of your participation in sport as a competitive athlete, and a healthy volunteer over the age of 18.

This consent form will give you the information you will need to understand why this study is being done and why you are being invited to participate. It will also describe what you will need to do to participate and any known risks, inconveniences or discomforts that you may have while participating. We encourage you to ask questions now and at any time. If you decide to participate, you will be asked to sign this form and it will be a record of your agreement to participate. You will be given a copy of this form.

B. PROCEDURES

As a participant in this study, you will experience the following procedural steps:

- Participants will be screened for differences in range of motion between both shoulders as well as previous history of shoulder injury, current shoulder problems, or surgery within the past year. Participants not meeting these inclusions will be excluded from the study.
- All participants will review the informed consent and have the opportunity to ask questions about participation. Participants will then be randomly assigned to one of two groups for study (self stretching or assisted stretching).
- All participants will be measured in a kinesiology department lab, or
another suitable environment, for shoulder internal and external rotation range of motion using a digital inclinometer. Participants will be instructed on which group they are in and further instructions about what you do next will be given.

- Participants in the self stretching group will administer the cross body shoulder stretch within a 28 day/1 month period and record their performance for each session. Participants in the assisted stretching group will be assigned to a research assistant whom the participant will report to for the stretching sessions over a 28 day/1 month period. Both stretches involve normal stretching of muscular tissue and discomfort would be comparable to other commonly used stretching techniques on the body. Time commitment for each session will be approximately 5 minutes or less. Self stretch participants can perform this stretch on their own accord wherever they wish, and assisted stretch participants will meet with a research assistant in an athletic training facility.

- All participants will be measured for shoulder range of motion after the 28 day period, as in step 3 above, which will allow for comparisons to the pre-test measurement to identify any changes in shoulder range of motion. Participants will be told at this time if there were any changes. No further participation is anticipated after this step. Participants will only be contacted in the future if further research is initiated based on the findings of this study.

C. RISKS AND INCONVENIENCES

- Participants in this study should not experience anything more than normal stretching sensations or discomfort from either of the stretching techniques employed.
- While participation in either of the stretching techniques will require minimal time to complete during each session, participation will require five minutes for each session and approximately fifteen minutes for pre and post measurements and medical history.

BENEFITS

- Participants in this study will receive no direct medical benefits from participation in this study. Although, it is expected that both stretching techniques will improve shoulder range of motion, which in theory may decrease risks of future shoulder injury. The information participants provide may help clinicians better understand the shoulder stretching techniques used in this study.

D. CONFIDENTIALITY

Participation in this research may involve a loss of confidentiality; however, your records will be handled as confidentially as possible. The following procedures
will be used to protect the confidentiality of your data. We will keep all study records (including codes to your data) locked in a secure location. Research records will be labeled with a code. All electronic files (e.g. database, spreadsheet, etc.) containing identifiable information will be password protected. Any computer hosting such files will also have password protection to prevent access by unauthorized users. Only the principal investigator will have access to the passwords. All documents associated with participant information will be stored in a secure office in a locked file cabinet in the principal investigators office and will be maintained for a period of at least three years. At the conclusion of this study, we may publish our findings. Information will be presented in summary format and you will not be identified in any publication or presentation.

E. PAYMENT

You will not be paid for your participation in this study.

F. QUESTIONS

If you have any questions or concerns about your participation in this study, you should first talk with the principal investigator, Dave Hammons, AT, at 870-0921 or co-investigator, Dr. John McChesney, AT, at 426-1481. If for some reason you do not wish to do this, you may contact the Institutional Review Board, which is concerned with the protection of volunteers in research projects. You may reach the board office between 8:00 AM and 5:00 PM, Monday through Friday, by calling (208) 426-5401 or by writing: Institutional Review Board, Office of Research Compliance, Boise State University, 1910 University Dr., Boise, ID 83725-1138.

In the event you become sick or injured during the course of the research study, immediately notify your personal physician and the principal investigator.

G. PARTICIPATION IN RESEARCH IS VOLUNTARY

You do not have to be in this study if you do not want to. You may also refuse to answer any questions you do not want to answer. If you volunteer to be in this study, you may withdraw from it at any time without consequences of any kind or loss of benefits to which you are otherwise entitled.
DOCUMENTATION OF CONSENT

Please initial if you agree to the following:

_____ I give consent to be photographed in this study.

I have read this form and decided that I will participate in the project described above. Its general purposes, the particulars of involvement and possible risks have been explained to my satisfaction. I understand I can withdraw at any time.

_________________________________________  _________________________
Signature of Study Participant  Date

_________________________________________  _________________________
Signature of Person Obtaining Consent  Date
APPENDIX E

Demographic Information and Medical History
Effects of internal rotation stretching techniques on glenohumeral internal rotation

Medical History and Demographic Data
BSU Athletic Training & Motor Control Laboratory. Boise, ID.

Please provide the following demographic information. For this research project we are requesting demographic information. Due to the make-up of Idaho’s population, the combined answers to these questions may make an individual person identifiable. We will make every effort to protect participant’s confidentiality. However, if you are uncomfortable answering any of these questions, you may leave them blank.

Date:
Name: Phone / Email:
Participant Code: Gender: M or F
Age:
Sport: Years you have played this sport:
Sport position / event:
Dominant shoulder (arm): R or L

1- Are you currently experiencing shoulder pain in either shoulder? Y or N
   If yes, please describe

2- Have you sustained a significant shoulder injury to either shoulder in the past year? Y or N
   If yes, please describe

3- Have you had a surgery to either shoulder in the past year? Y or N
   If yes, please describe

4- Do you consider yourself as a healthy athlete? Y or N
   If no, please explain
APPENDIX F

Pre- and Post-Test Measurement Form
Effects of a novel internal rotation stretching technique on glenohumeral internal rotation

Pre Test ROM measurements
BSU Athletic Training & Motor Control Laboratory. Boise, ID.

Participant Code: _______________________________ Date: _______________________________

### INTERNAL Rotation Measurement

<table>
<thead>
<tr>
<th>Pre-Test ROM</th>
<th>Left Shoulder</th>
<th>Right Shoulder</th>
</tr>
</thead>
<tbody>
<tr>
<td>IR ROM 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IR ROM 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IR ROM 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IR Avg.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

IR (nd-d) =°

### EXTERNAL Rotation Measurement

<table>
<thead>
<tr>
<th>Pre-Test ROM</th>
<th>Left Shoulder</th>
<th>Right Shoulder</th>
</tr>
</thead>
<tbody>
<tr>
<td>ER ROM 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ER ROM 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ER ROM 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ER Avg.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Average total GH joint motion [IRavg + ERavg]:
Effects of internal rotation stretching techniques on glenohumeral internal rotation

Post Test ROM measurements
BSU Athletic Training & Motor Control Laboratory. Boise, ID.

<table>
<thead>
<tr>
<th>Participant Code: _______________________________</th>
<th>Date: ________________________________</th>
</tr>
</thead>
</table>

### INTERNAL Rotation Measurement

<table>
<thead>
<tr>
<th>Pre-Test ROM</th>
<th>Left Shoulder</th>
<th>Right Shoulder</th>
<th>IR Avg.</th>
<th>IR (nd-d) =°</th>
</tr>
</thead>
<tbody>
<tr>
<td>IR ROM 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IR ROM 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IR ROM 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IR Avg.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

### EXTERNAL Rotation Measurement

<table>
<thead>
<tr>
<th>Pre-Test ROM</th>
<th>Left Shoulder</th>
<th>Right Shoulder</th>
<th>ER Avg.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ER ROM 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ER ROM 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ER ROM 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ER Avg.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Average total GH joint motion [IRavg + ERavg]: [Blank] [Blank]
APPENDIX G

Cross-Body Treatment Schedule
Effects of internal rotation stretching techniques on glenohumeral internal rotation study

Cross body (self stretch) participant schedule log

Participant code:

<table>
<thead>
<tr>
<th></th>
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<tbody>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 2</td>
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<td></td>
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<td></td>
<td></td>
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<td></td>
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Dominant shoulder being stretched: L R

* Place a check mark and date in the appropriate box when the stretch session is performed. A minimum of 12 separate sessions is required within 28 days, or 1 month. Thank you for your participation. Contact Dave Hammons @ 870-0921 with any questions.
APPENDIX H

Prone-Passive Treatment Schedule
Effects of internal rotation stretching techniques on glenohumeral internal rotation study

Passive Prone IR stretch participant schedule log

Participant code:

Dominant shoulder being stretched:  L  R

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* Research assistant: Place your initials and date in the appropriate box when the stretch session is performed. A minimum of 12 separate sessions is required over the 4 weeks for inclusion in the study, preferably 3 times per week or more. Thank you for your participation. Contact Dave Hammons @ 870-0921 with any questions.