A Prediction of SICK Scapula Syndrome Score from Scapular Stabilizer Muscle Activation Analysis in Overhead Athletes

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ABSTRACT

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A Prediction of SICK Scapula Syndrome Score from Muscle Activation and Kinematic Analysis in Overhead Athletes

(Under the direction of Dr. Joseph B. Myers, Sakiko Oyama, Dr. Steven M. Zinder, and Dr. William E. Prentice)

Objective: To determine if subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] can be predicted from scapular stabilizer muscle activation. **Design:** Quasi-experimental, one group design with a counterbalancing of two functional tasks. **Subjects:** NCAA Division I and/or recreational club overhead athletes (n = 40). **Measurements:** The SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] was used for assessing the severity of scapular malposition. Muscle activation was recorded for the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles. **Results:** Regression analyses revealed that scapular stabilizer muscle activation amplitude did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] **Conclusion:** In overhead athletes, mean muscle activation amplitude of the scapular stabilizers was not found to be valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1]

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CHAPTER I

INTRODUCTION

An Overview

Shoulder pain, regardless of its nature, source, or severity, currently plagues today's overhead athlete.[2-4] The demands of sport require overhead athletes to repetitively endure high amounts of stress and strain to the glenohumeral and scapulothoracic joints. These demands are great, and in turn, so is the risk of shoulder soft tissue injury due to overuse. Warner et al[5] have shown that the vast majority of patients suffering from an overuse shoulder pathology present with scapulothoracic asymmetries during an elevation task. When evaluating a shoulder soft tissue injury, medical professionals face the challenge of determining whether scapular malposition and dyskinesis were the result of a shoulder joint pathology or the source of its development. Current literature suggests that scapular asymmetries may be an objective means of understanding the development of the shoulder pathology itself.[1, 3, 5-16] Scapular malalignment is perhaps one of the most evident signs of shoulder dysfunction that may lead to the initiation of the pathological sequence of events thereafter.[1, 3, 4, 8-10, 13-15, 17, 18]

The upper extremity kinetic chain begins at the shoulder joint complex, with the scapula serving as the base of stability during the performance of overhead functional movement patterns.[19] Because the scapula is connected to the axial skeleton via the small acromioclavicular joint, its stability and mobility are dictated by the numerous muscles

attached to it. The scapular force couple refers specifically to the stability and balance provided to the scapulothoracic joint by the upper trapezius, lower trapezius, rhomboid major, rhomboid minor, levator scapulae, and serratus anterior muscles.[9] Alteration of the activity in any of the muscles contributing to this force couple can lead to the disruption of the force couple, which may decrease both the scapula's stability and overall ability to serve as a muscle attachment site for the scapulothoracic and scapulohumeral muscles. When these muscles surrounding the shoulder girdle lose their stable base of attachment, dynamic glenohumeral joint stabilization may be compromised.

The serratus anterior and trapezius muscles are recognized as two of the critical muscles providing scapular stability as well as serving to upwardly rotate the scapula. Having this critical role in scapular kinematics, various research studies suggest that dysfunction of these muscles are associated with overhead pathologies.[9, 15, 19-23]

Findings by Wadsworth et al[23] revealed that, in a population of swimmers suffering from subacromial impingement syndrome, muscle recruitment of the serratus anterior was delayed when compared to an asymptomatic control group. Results of a study by Ludewig et al[10, 24] indicated that patients suffering from shoulder impingement syndrome displayed altered neuromuscular control of the scapular dynamic stabilizers when compared to an asymptomatic control group. Based on the results of these studies, it can be theorized that altered muscle activation at the shoulder joint complex may be related to soft tissue injury.

Research has repeatedly shown that scapular dyskinesis and postural abnormalities in overhead athletes may be an important risk factor for the development of subacromial impingement syndrome.[1, 3, 4, 8-10, 13-15, 17, 18] Shoulder soft tissue pathology and scapular dyskinesis appear to be intimately related to one another. As a result, asymmetric

malpositioning of the scapula is now being considered a precursor to the development of a number of different shoulder joint pathologies, specifically subacromial impingement syndrome.[1, 3, 4, 8-10, 13-15, 17, 18] Shoulder soft tissue overload and the subsequent injury that results from subacromial impingement syndrome have been linked to decreased scapular upward rotation, decreased scapular posterior tilt, and increased scapular internal rotation with humeral elevation tasks.[10]

Burkhart et al[1] use the acronym SICK when describing a specific form of scapular dyskinesis. SICK scapula refers to the presence of **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement. Overhead athletes diagnosed with SICK scapula syndrome typically present with a unilateral lowered and anteriorly tilted scapula with accompanied anterior shoulder pain on the involved side. In overhead athletes, one initial sign of SICK scapula syndrome is shoulder biomechanical dysfunction and pain. Because athletic skill is dependent on biomechanical ease and efficiency, shoulder pain and altered overhead biomechanics can become extremely debilitating for the competitive overhead athlete.[1, 6, 7, 9, 13, 19, 25] With the ongoing establishment of SICK scapula syndrome, scapular dyskinesis is becoming more objectively assessed for both therapeutic rehabilitation purposes and prophylactic conditioning purposes.

The Problem

Currently there is much speculation among sports medicine professionals regarding the pathologic pathway occurring in the symptomatic overhead athletic shoulder. This pathway is summarized in the above introduction and is a representation of the current status of understanding regarding chronic, overuse overhead shoulder pathologies in the athletic

population. Unfortunately however, both the linkages and the sequence of the events taking place in this pathway remain unclear. More specifically, it is not yet fully understood whether altered EMG muscle activation of the scapular stabilizers is a sign of an existing pathology or a precursor to its development.

Overhead athletes are susceptible to developing postural malalignments due to overhead sport-specific pattern overloads.[13, 26] As a result of these postural malalignments, muscle imbalances may develop at the shoulder joint complex, specifically between the anterior and posterior musculature of the thorax. Whether unilateral or bilateral, muscle imbalance at the scapulothoracic joint can contribute to scapular malpositionings. In overhead athletes exhibiting scapular malpositionings, passive lengthening of the thorax's posterior musculature (i.e. the scapular stabilizers) has occurred in response to the passive shortening of the thorax's anterior musculature (i.e. the pectoralis minor muscle).[13, 26] Altered length-tension relationships occurring within each of the scapular stabilizer muscles can lead to decreased function and force production, and as a result, lead to decreases in scapular stabilizer muscle strength.[9, 19] Scapular force couple muscle imbalances are thought to compromise dynamic joint stabilization, specifically altering the upper trapezius to middle trapezius, lower trapezius, and serratus anterior force couple ratio. Although there is no definitive link between muscle imbalances of the scapular force couple and shoulder subacromial impingement, the influence of such imbalances on scapular kinematics is one of potential injury acquisition.[18] Even the most subtle scapular and/or glenohumeral muscle imbalance can lead to scapular dyskinesis[9, 19], which in turn, can manifest itself into a predictable pattern of tissue overload and dysfunction.[1, 3, 4, 8-10, 13-15, 17, 18]

Whether via special tests (i.e. the Scapular Assistance Test) or the palpation of anatomical landmarks (i.e. the coracoid process of the scapula), one approach to the clinical diagnosis of an underlying shoulder pathology has been the use of patient self-reported pain symptoms. Pain is the common thread interweaving most shoulder disorders, with the source and mechanism of such pain being of extreme variability. Because pain is a person's perception of physical damage, subjective athlete pain reports have the advantage of being athlete-specific and providing an important perspective on athlete status. They cannot, however, accurately represent the presence or the severity of an actual physical impairment with associated soft tissue damage. Currently, there is a need for validated, quantitative measures to improve the reliability of shoulder pathology assessment.

As is the case with the clinical assessment of SICK scapula syndrome, qualitative pain measures provide an incomplete picture of the severity of this scapular malpositioning and dyskinetic disorder. Based upon both the literature and screening guidelines of Burkhart and Morgan[1], the severity of the SICK scapula syndrome is graded on a 0 to 20 point scale, with 0 representing complete shoulder health and 20 representing severe, symptomatic SICK scapula syndrome. It must be noted, however, that both the validity and the reliability of this scale have not yet been established. Typically upon screening, athletes who present with symptomatic SICK scapula syndrome will score somewhere within the range of 10 to 14 on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], but, at this point in time, the scale's diagnostic value is merely anecdotal. This suggests the need for a valid, structured clinical tool that could be utilized by clinicians as a predictor for the potential, presence, and/or severity of SICK scapula syndrome.

Purpose and Clinical Relevance

The purpose of this study was to validate the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale, developed by Burkhart et al[1], by predicting subject score from scapular stabilizer muscle activity. Determining the validity of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] may allow clinicians to more readily utilize this scale as an accurate screening tool toward the identification of SICK scapula syndrome.

The identification of scapular muscle dysfunction in pathologic, SICK scapula syndrome overhead athletes will allow the condition to be more successfully treated. Understanding the adaptive scapular stabilizer activation deficiencies associated with SICK scapula syndrome would allow clinicians to implement specific rehabilitation exercises when treating affected athletes. The rehabilitation exercises that target the muscle identified to be dysfunctional in this study could help restore ideal scapular force couple synchronization, thus allowing the scapula to move harmoniously with the moving humerus. Such fine-tuned, corrective exercise would effectively allow a rehabilitating athlete to have a pain-free return to competition.

Research Question

Is mean electromyographic (EMG) amplitude of scapular stabilizer muscles a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks in overhead athletes?

- **RQ1:** Is mean muscle activation amplitude of the **upper trapezius** a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** in overhead athletes?
- **RQ2:** Is mean muscle activation amplitude of the **middle trapezius** a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** in overhead athletes?
- **RQ3:** Is mean muscle activation amplitude of the **lower trapezius** a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** in overhead athletes?
- **RQ4:** Is mean muscle activation amplitude of the **serratus anterior** a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** in overhead athletes?

Research Design

Quasi-experimental, nonequivalent one group design with a counterbalancing of tasks

Independent Variables (see Table 2)

- Mean muscle activation amplitude (normalized to a maximum voluntary contraction) of the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles during ascending and descending phases of functional movement patterns
- Tasks
    - Glenohumeral elevation in the sagittal plane (flexion)
    - Glenohumeral elevation in scapular plane (scaption)
- Phases of the tasks
    - Ascending phase of glenohumeral elevation
        - 0-120º
    - Descending phase of glenohumeral elevation
        - 120-0º

Dependent Variable (see Table 2)

- Severity of pathology
    - Subject score from the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]

Hypotheses

Mean muscle activation amplitude of the scapular stabilizers will be a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional movement patterns in overhead athletes

- **H1:** An increase in **upper trapezius** mean muscle activation amplitude during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** will be a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] in overhead athletes.
- **H2:** A decrease in **middle trapezius** mean muscle activation amplitude during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** will be a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] in overhead athletes.
- **H3:** A decrease in **lower trapezius** mean muscle activation amplitude during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** will be a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] in overhead athletes.
- **H4:** A decrease in **serratus anterior** mean muscle activation amplitude during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** will be a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] in overhead athletes.

Operational Definitions

- Overhead athletes:
    - NCAA Division I overhead athletes and/or recreational club overhead athletes who participate in a sport that requires their arm to be above their shoulder height on a repetitive basis during throwing or striking activities (i.e. baseball, softball, swimming, tennis, volleyball). Athletes must be active in their overhead sport for a duration of at least 30 minutes per session for at least 3 individual sessions per week
- SICK scapula syndrome:
    - SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] (see Figure 1) will be used to define the subjects with SICK scapula syndrome
    - Subjects who score a 10 or greater on the rating scale will be considered to have SICK scapula syndrome
    - Subjective
        - Coracoid process pain
        - Acromioclavicular joint pain
        - Periscapular pain (superior medial angle)
        - Subacromial pain (proximal lateral arm pain)
        - Radicular pain below the elbow

- Objective
    - Coracoid process tenderness
    - Acromioclavicular joint tenderness
    - Superior medial angle/scapular tenderness
    - (+) Provocative impingement test (Hawkins-Kennedy Impingement Sign)
    - (+) Scapular assistance test
    - (+) Thoracic outlet syndrome test (Allen Test)
- Static Scapular Malposition
    - Infera (i.e. the visual appearance of a dropped scapula due to scapular tilting or protraction)
    - Lateral displacement
    - Abduction

- Functional movement patterns:
    - Glenohumeral elevation in the sagittal plane (flexion) with hand in neutral position
    - Glenohumeral elevation in the scapular plane (scaption) with hand in neutral position
    - Mean EMG amplitude for all functional movement patterns recorded upon both the ascending phase (0^{o}-120^{o}) and the descending phase (120^{o}-0^{o}) of shoulder flexion and scaption
    - Functional movement patterns will be performed in an approximately 2-second ascending phase, 2-second descending phase motion
    - 10 repetitions per each set
    - 2-minute rest periods between all sets

Assumptions

- Sex did not influence the results of this study
- Overhead athletes perform relatively the same functional glenohumeral/scapulothoracic movement patterns, regardless of their specific overhead sport
- Subject symptom report was both honest and unbiased

Delimitations

- Subjects with the following history of injury will be excluded from the study to control for the influence of each injury on scapular muscle activity:
    - Shoulder or neck surgery
    - Rotator cuff tear
    - Cervical spine pathology
    - Acute-onset shoulder pathology within the last six months
    - Adhesive capsulitis
    - Unstable episodes within the last six months (glenohumeral subluxation, dislocation, self-subluxation)
    - Scoliosis

CHAPTER II

REVIEW OF THE LITERATURE

Introduction

There are a number of gaps in the current literature concerning the role that scapular muscle activation, scapular kinematics, and shoulder biomechanics play in the progression of injury to the shoulder joint complex in athletics. Disagreement exists regarding both the etiology and the sequence of events thereafter concerning these chronic, overuse injuries afflicting much of today's athletic population.[2-4]

Currently there is much debate surrounding the topic of scapular dyskinesis as to whether it is a precursor, product, or complication of shoulder soft tissue pathology. Nevertheless, studies have shown that alterations in scapular position and motion occur in 64 to 100% of patients afflicted with shoulder injuries.[5] Kibler et al[9] have shown that scapular dyskinesis is extremely prevalent among individuals suffering from some type of shoulder pathology, revealing its occurrence in 68% of patients with rotator cuff abnormalities, 94% of patients with labral tears, and 100% of patients with diagnosed glenohumeral multi-directional instability. These findings are further supported by Warner et al[5] in a study comparing subjects suffering from overuse shoulder pathologies with that of an asymptomatic control group. Findings of this study revealed that the vast majority of the pathologic population presented with scapulothoracic asymmetries.

With the highly repetitious nature concerning the demands of overhead athletics, any degree of scapular malalignment or dyskinesis could exacerbate an already underlying soft tissue pathology. Future research needs to be focused toward understanding what acts as the catalyst in the progression from sport-specific pattern dominance to injury. Clinically, further understanding regarding scapular asymmetries could lead to the implementation of prophylactic conditioning protocols, effective treatments for pain management, and corrective, therapeutic exercises to help return athletes to their peak of athleticism.

Critical Shoulder Anatomy

Before one can master the art of effectively treating a shoulder pathology, one must first understand both the structural and functional anatomy comprising the shoulder joint complex. A true understanding of shoulder girdle anatomy will reveal how the form of the shoulder's bony and soft tissue structures effectively suits its function.

The shoulder joint complex refers to the intricate arrangement of the humerus, clavicle, scapula, and sternum and their articulations with one another. The joint "complex" itself is actually composed of four distinct joints: the sternoclavicular joint, acromioclavicular joint, glenohumeral joint, and scapulothoracic joint.

Sternoclavicular Joint

The clavicle is commonly referred to as a "strut" capable of supporting the glenohumeral joint in a free-moving, suspended position lateral to the midline of the axial skeleton. The sternoclavicular joint refers to the articulation of the sternal extremity of the clavicle with the clavicular fossa of the manubrium, with an articular disk interposed between

the two. This saddle-type joint allows for clavicular elevation/depression, anterior/posterior rotation, and protraction/retraction throughout shoulder range of motion in three planes of movement.[25]

Static stabilization at the sternoclavicular joint is provided by the costoclavicular ligament, the interclavicular ligament, and the sternoclavicular ligaments. These ligamentous structures provide restraint against distal loads. The costoclavicular ligament is comprised of two portions, an anterior portion and a posterior portion. The anterior portion of the costoclavicular ligament functions to resist both upward rotation and lateral displacement of the medial clavicle on the clavicular fossa of the manubrium, while the posterior portion of the costoclavicular ligament functions to resist both downward rotation and medial displacement of this same articulation. The interclavicular ligament adjoins the right mediosuperior clavicle to the left mediosuperior clavicle, just superior to the manubrium. This ligament is capable of preventing downward rotation of the lateral clavicle upon distal loading. Similar in function to that of the interclavicular ligament are the sternoclavicular ligaments. These ligaments are sectioned thickenings of the sternoclavicular joint capsule, and as indicated, prevent downward rotation of the lateral clavicle upon distal loading.[25]

Acromioclavicular Joint

Laterally the clavicle articulates with the acromion process. Joint congruency between these two bony surfaces is provided by a meniscoid disk located within the acromioclavicular joint capsule. Stability at the acromioclavicular joint comes from the strength provided by its joint capsule, with capsular thickness most substantial at its superior portion. Stability at the acromioclavicular joint is also provided indirectly from two auxiliary

ligaments, the coracoacromial ligament and the coracoclavicular ligament. The coracoacromial ligament runs from the coracoid process to the inferior surface of the acromion process. The coracoclavicular ligament is actually made up of two distinct smaller ligaments, the conoid and the trapezoid. Similar in function to that of the coracoacromial ligament, this ligamentous complex acts to absorb and distribute distal loads that occur at or around the acromioclavicular joint. As previously mentioned, both the coracoacromial ligament and the coracoclavicular ligamentous complex assist the acromioclavicular joint capsule in adjoining the clavicle to the scapula.

Glenohumeral Joint

By far the most intricate articulation of the shoulder joint complex is the glenohumeral joint. This articulation consists of the hemi-spherical humeral head on the glenoid fossa of the scapula. The anatomy of these two articulating surfaces greatly favors joint mobility to joint stability. The mobility of this ball-and-socket joint is remarkable and allows for fine, distal motor skill at both the wrist and fingers.

Static stabilization at the glenohumeral joint is provided by a number of different components. The first component is known as humeral version. Humeral version refers to the 130 to 140 degree posterior angulation of the humeral neck relative to the humeral shaft, as well as the 30 degree retroversion of the humeral head relative to the transepicondylar axis of the elbow.[25] Both of these anatomical angulations help to control anterior humeral head translation by increasing the articular stability of the humeral head on the shallow glenoid fossa in a posteriorly directed position.

The second component of glenohumeral joint static stabilization is known as glenoid version. Differing slightly from humeral version, glenoid version refers the angulation of the glenoid fossa relative to the scapula. In a resting position, the glenoid fossa has been found to have a 5-degree superior tilt relative to the scapular body.[25] This anatomical angulation helps to control inferior humeral head translation by increasing the articular stability of the humeral head on the glenoid fossa in a superiorly directed position.

The third component of glenohumeral joint static stabilization is the presence of a fibrocartilaginous ring known as the glenoid labrum. The nature of the glenohumeral ball-and-socket joint is one of a very large ball relative to a very small, shallow socket. Studies have revealed that only approximately 25% of the humeral head is in contact with the glenoid fossa's center of rotation at any point throughout full humeral range of motion. The glenohumeral index is a ratio, where maximum glenoid diameter is divided by maximum humeral diameter. Calculations reveal the glenohumeral index to be 0.75 in the sagittal plane and 0.76 in the transverse plane, indicating the inherent instability found within the glenohumeral articulation itself. The glenoid labrum functions to deepen the glenoid fossa by 9 mm in the superior-inferior direction and by 5 mm in the anterior-posterior direction.[25] This deepening of the socket results in up to a 50% depth increase of the glenoid fossa and helps to stabilize the humeral head on the glenoid fossa.

The fourth component of glenohumeral joint static stabilization is the presence of intraarticular pressure. Within the glenohumeral joint capsule, there is a slightly negative intraarticular pressure that functions to center and hold the humeral head on the glenoid fossa. This suction effect is extremely effective at decreasing the space between the humeral head and the glenoid cavity when the shoulder is in a resting position but gradually decreases

as the humerus moves from adduction to abduction and from internal rotation to external rotation.

As intraarticular pressure decreases in effectiveness throughout humeral elevation, the glenohumeral joint's static restraints increase in value. These static stabilizers are referred to collectively as the glenohumeral ligament complex and are of increasing importance as the humerus moves from a neutral position to positions of shoulder abduction, flexion, and external rotation. Specifically, this complex consists of the superior glenohumeral ligament, middle glenohumeral ligament, and inferior glenohumeral ligament. The superior glenohumeral ligament runs from the supraglenoid tubercle of the scapula to the lesser tuberosity of the humerus. The primary function of this ligament is to prevent excessive inferior translation of the humerus when the shoulder is in neutral and to prevent excessive shoulder external rotation in the early degrees of frontal plane range of motion. The middle glenohumeral ligament runs from the superior glenoid fossa region of the scapula to the lesser tuberosity of the humerus. The primary function of this ligament is to prevent excessive anterior translation of the humerus when the shoulder is abducted to 45 degrees and to prevent excessive external rotation of the humerus when the shoulder is abducted between 60 and 90 degrees. The inferior glenohumeral ligament is actually comprised of two distinct bands, an anterior and a posterior band. The anterior band of the inferior glenohumeral ligament runs from the superior-anterior glenoid labrum to the inferior humeral head near the subscapularis tendon, and the posterior band of the inferior glenohumeral ligament runs from the superior-posterior glenoid labrum to the inferior humeral head near the triceps long head tendon. The coupling of these two ligament bands allows the inferior glenohumeral ligament to shift positions dependent on shoulder position. When the humerus

is in a position of abduction/external rotation, the inferior glenohumeral ligament shifts anteriorly, hence allowing it to prevent excessive anterior translation of the humerus. When the humerus is in a position of abduction/internal rotation however, the inferior glenohumeral ligament shifts posteriorly, therefore positioning itself to prevent excessive posterior translation of the humerus. One final ligamentous structure of the glenohumeral joint is the coracohumeral ligament. The coracohumeral ligament is extraarticular in nature, running from the lateral coracoid process and inserting on both the lesser and greater tuberosities of the humerus. The primary function of this ligament is to prevent excessive inferior translation when the humerus is in a position of adduction/external rotation.

The glenohumeral joint is dynamically stabilized by both the rotator cuff musculature, specifically the supraspinatus, infraspinatus, teres minor, and subscapularis, and the long head of the biceps brachii muscle. Each of the individual muscles of the rotator cuff and the biceps brachii originate on the scapular body and insert onto the humeral head, thus pulling the humerus closer to the glenoid cavity upon activation. Simultaneous contraction of these five muscles creates a compression effect of the humeral head into the glenoid cavity. As the rotator cuff and biceps brachii musculature contract to pull the humeral head downward and inward, this humeral head compression is coupled by the upward and outward pull of the anterior, middle, and posterior deltoid musculature. This mechanism is commonly referred to as the glenohumeral force couple in the frontal plane. When in balance, this force couple functions to center the humeral head in the glenoid cavity by resisting excessive superior-inferior humeral translation. Another glenohumeral force couple refers to the simultaneous contraction of the infraspinatus, teres minor, and subscapularis muscles. This mechanism is commonly referred to as the glenohumeral force couple in the sagittal plane. When in

balance, this force couple functions to center the humeral head in the glenoid cavity by resisting excessive anterior-posterior humeral translation.

Scapulothoracic Joint

The scapulothoracic joint refers to the non-traditional articulation between the anterior, concave surface of the scapula on the posterior, convex surface of the thorax. This specific joint is non-traditional in nature due to its stabilizing features. The scapulothoracic joint is comprised solely of dynamic stabilizers functioning to suspend the scapula on the posterior thorax wall. Perhaps the most defining characteristic of the scapulothoracic joint is its high degree of mobility. This mobility of the scapula on the thorax wall is made possible due to the influence of the force couple of the muscles attached to the scapula. Although there are numerous periscapular muscles that attach to the scapula, the scapular force couple refers specifically to the stability and balance provided to the scapulothoracic joint by the upper trapezius, middle trapezius, lower trapezius, rhomboid major, rhomboid minor, levator scapulae, and serratus anterior muscles.[9] The scapular stabilizers function both eccentrically and concentrically to position the scapula on the thorax throughout upper extremity range of motion. Eccentrically, the scapular stabilizers undergo controlled lengthening (i.e. deceleration of shoulder internal rotation/protraction/extension). Concentrically, the scapular stabilizers undergo active shortening (i.e. acceleration of shoulder external rotation/retraction/flexion). Optimal scapular force couple synchronization allows for normal scapular rotation.[3]

The upper trapezius has an origin at the external occipital protuberance and an insertion at the posterior-lateral clavicle. Contraction of the upper trapezius creates scapular

upward rotation, elevation, and retraction. The middle trapezius has an origin at spinous processes of the first through fifth thoracic vertebrae and an insertion at the medial margin of the acromion. Contraction of the middle trapezius creates scapular upward rotation and retraction. The lower trapezius has an origin at the spinous processes of C7 through T12 and an insertion at the tubercle crest of the scapular spine. Contraction of the lower trapezius creates scapular upward rotation, depression, and retraction. The rhomboid major has an origin at spinous processes of the T2 through T5 vertebrae and an insertion at the medial scapular border from the scapular spine to the inferior angle. Contraction of the rhomboid major creates scapular retraction, downward rotation, and depression. The rhomboid minor has an origin at the nuchal ligament and the spinous processes of C7 and T1 vertebrae and an insertion at the medial scapular border of scapula just inferior to the scapular spine. Similar to that of the rhomboid major, contraction of the rhomboid minor creates scapular retraction, downward rotation, and depression. The serratus anterior is composed of three distinct divisions. The first division has origins on the first and second ribs and an insertion onto the superior angle of the scapula. The second division has origins on the second, third, and fourth ribs and an insertion onto the anterior-medial border of the scapula. The third division has origins on the fifth through the ninth ribs and an insertion onto the inferior border of the scapula. Contraction of the serratus anterior creates scapular protraction and upward rotation. Most importantly, the serratus anterior acts as the primary scapular stabilizer holding the scapula securely onto the thorax wall, a crucial role in the maintenance of normal scapular kinematics.

Neuromuscular Control of the Shoulder

Coinciding with the dynamic stability provided by the shoulder joint complex's surrounding musculature comes one final mechanism responsible for joint stability, neuromuscular control. Neuromuscular control is defined as the unconscious activation of dynamic restraints occurring in preparation and in response to joint motion and loading for the purpose of maintaining functional joint stability.[27] Neuromuscular control is the preparatory muscle contraction that occurs in response to anticipated external loads as a means of injury prevention. The ability to unconsciously stabilize the shoulder joint complex, especially when in vulnerable positions throughout the range of motion, significantly minimizes the potentially devastating consequences that could result without this mechanism of dynamic stabilization.[27]

Overview of Shoulder Osteokinematics and Arthrokinematics

The kinematics involved at the shoulder joint complex are joint-specific and extremely multifaceted. The sternoclavicular, acromioclavicular, glenohumeral, and scapulothoracic joints are each mechanically unique from one another. At the sternoclavicular joint, the clavicle rotates superiorly with shoulder flexion/abduction and rotates inferiorly with shoulder extension/adduction. The clavicle also rotates anteriorly with scapular protraction and posteriorly with scapular retraction.[25] Shoulder flexion causes the clavicle to posteriorly rotate on its axis approximately 45 degrees.[25]

At the glenohumeral joint, the humeral head glides anteriorly with shoulder external rotation and posteriorly with shoulder internal rotation. The humeral head also glides both posteriorly and inferiorly upon humeral flexion and anteriorly and superiorly upon humeral

extension. Lastly, the humeral head glides inferiorly with abduction and superiorly with adduction.

The kinematics of the scapulothoracic joint are typically described using three sets of rotational descriptives: anterior and posterior tilting, upward and downward rotation, and internal and external rotation. These three rotation sets occur in the following axes: anterior and posterior tilting occurs about an axis that runs through the scapular spine, upward and downward rotation occurs about an axis perpendicular to the plane of the scapula, and internal and external rotation occurs about an axis parallel to the longitudinal axis of the thorax.[28] Humeral elevation involves the simultaneous scapular kinematics of posterior tilting, upward rotation, and external rotation, while humeral depression involves the simultaneous scapular anterior tilting, downward rotation, and internal rotation.[4]

While there is indeed independent movement occurring at each of the four articulations of the shoulder joint complex, there is also an intimate relationship between all four. Such kinematic synchronization is the key to the efficient upper extremity movement.[4, 9, 13, 19, 25, 29] One such relationship is commonly referred to as scapulohumeral rhythm. Scapulohumeral rhythm accounts for the approximately 2:1 ratio between glenohumeral abduction and scapulothoracic rotation occurring throughout overhead activities when the athlete is functioning between 20 and 120 degrees of glenohumeral abduction.[4, 13, 25, 29] Prior to 20 degrees of glenohumeral abduction, the scapula is stationary as it is being held onto the thorax by the scapular stabilizers. After 120 degrees of glenohumeral abduction, scapulohumeral rhythm shifts from an approximately 2:1 ratio between glenohumeral abduction and scapulothoracic rotation to an approximately 1:1 ratio for the remainder of overhead range of motion achievement.[4, 13, 25, 29]

Characteristics of the Athletic Shoulder

The complexity of the shoulder joint complex cannot fully be appreciated until it is looked at kinematically throughout the demands of athletics. In a healthy population of athletes, according to Kibler[19], there are five major functions of the scapula. The first role of the scapula is to provide stability to the glenohumeral joint. In doing so, the scapula moves harmoniously with the moving humerus to ensure that there is always a stable center of rotation for the moving humerus throughout its active range of motion. When the humerus and the scapula move in coordination with one another, the humeral head will remain centered on the glenoid cavity throughout the range of motion, which allows for glenohumeral activity with minimum stress on the surrounding soft tissue structures.

The second role of the scapula is its ability to both protract and retract. As was previously described, the scapula will retract and externally rotate along the thorax as the humerus moves from a position of extension and internal rotation to a position of flexion and external rotation. The position of shoulder flexion, maximum external rotation and abduction to 90 degrees, often referred to as the "cocking" phase of throwing or serving, is crucial for overhead athletes, because it allows the anterior musculature of their shoulder and trunk (i.e. the horizontal adductors – pectoralis major and pectoralis minor) to undergo maximum tension just prior to an explosive, concentric contraction. The acceleration of the limb by the explosive muscle contraction is directly related to the velocity of the pitch or a serve, and therefore is critical for an overhead athlete's performance. Scapular protraction is what is occurring throughout the completion of the acceleration phase of an overhead athlete's functional movement pattern, where the scapula is internally rotating along the thorax as the

humerus moves from a position of flexion and external rotation to a position of extension and internal rotation.

The third role of the scapula regards acromial elevation. Acromial elevation prevents impingement of the rotator cuff tendons in the "cocking" phase of the overhead athlete functional movement pattern. Fleisig et al[17] found that almost all throwing and serving activities occur with the humerus-to-scapular spine angle between 85 to 100 degrees of abduction, thus making rotator cuff impingement seemingly inevitable without acromial elevation. Similar to these findings, Myers et al[13] found an adaptive increase in scapular upward rotation among normal, healthy throwing athletes. This adaptation was believed by the authors to assist in the achievement of subacromial clearance throughout the throwing movement pattern, thus acting as a means of injury prevention (i.e. subacromial impingement).

The fourth role of the scapula concerns its function as a site for muscle origin as well as insertion for both the intrinsic and extrinsic muscles of the shoulder joint complex. Specifically, the scapula is the insertion site for the scapular stabilizers (i.e. the rhomboid major, rhomboid minor, serratus anterior, levator scapulae, and all three divisions of the trapezius). Also, the scapula is the origin for the four rotator cuff muscles, as well as the teres major, biceps brachii, middle and posterior deltoid, and the long head of the triceps brachii.

The fifth and final role of the scapula coincides with the fourth role that the scapula plays in athletic function. The scapula acts as an extremely crucial link in the body's kinetic chain, transferring the power generated from the lower extremity and trunk to the functional movement pattern of an athlete's sport-specific motion. By providing this dynamic link

between the trunk and the humerus, the muscles originating and inserting on the scapula are the driving force behind all scapular and glenohumeral kinematic movement. When there is a disruption of this dynamic link (i.e. scapular muscle activation dysfunction), inefficient energy transfer occurs throughout the kinetic chain, leading to decreased athletic performance.

Pathology of the Athletic Shoulder

When there is harmony between each of the shoulder joint complex's many facets, the shoulder is capable of functioning efficiently. It becomes very clear, however, after understanding both the anatomy and kinematics involved that the potential for dysfunction at the shoulder joint complex is innate to its structural and functional complexity. The demands of sport are remarkable and require overhead athletes to repetitively endure high amounts of stress and strain to the glenohumeral and scapulothoracic joints. Even the most subtle disruption of one of the static or dynamic components of shoulder joint stabilization can lead to kinematic dysfunction, tissue overload, and injury acquisition.[1, 6, 7, 9, 13, 19, 25] Subacromial impingement syndrome describes the pathologic contact of the shoulder rotator cuff tendons, biceps brachii long head tendon, or other glenohumeral joint soft tissue structures with the inferior surface of the acromion process and/or coracoacromial ligament.[17] Due to long-term mechanical overload, the result of subacromial impingement is often abrasion, compression, entrapment, degeneration, or even full thickness rupture of one or a number of the musculotendonous structures lying within the subacromial space.[4] Countless researchers have dedicated themselves toward understanding the progression from sport-specific

functional pattern dominance to injury, and as a result, many different theories currently exist.

Despite the researchers' effort to identify a defining characteristic of individuals with subacromial impingement syndrome, the risk factors toward the development of this injury have not been clearly understood due to the lack of prospective design studies. However, various physical characteristics have been suggested to be associated with the subacromial impingement syndrome. The following five conditions are, perhaps, the most important concerning the progression of shoulder soft tissue pathology: forward head and rounded shoulders posture, posterior shoulder tightness and/or contracture, anterior coracoid musculature tightness and/or contracture, weakness and/or inhibition-based muscle dysfunction of scapular stabilizers, and altered neuromuscular control patterns of glenohumeral or scapular force couple muscles.[1, 6, 7, 15, 27, 30-32]

Forward Head and Rounded Shoulders Posture

The first commonly described predisposing factor contributing to the acquisition of subacromial impingement syndrome is a forward head and rounded shoulders posture (FHRSP). Patients with shoulder pain demonstrate a scapular resting position of increased protraction and downward rotation when compared to those with ideal posture, as well as an increased kyphotic angle of the thoracic spine.[15] Thoracic kyphosis refers to an increased posterior spinal curvature of at least 5 degrees due to an anterior vertebral wedging-effect that involves at least three consecutive vertebrae.[25] Thigpen[15] found that subjects with FHRSP displayed significant alterations in scapular kinematics during humeral elevation in the frontal plane. Specifically, these individuals remained in an increased scapular internally

rotated and anteriorly tilted position throughout humeral elevation when compared to those with ideal posture. FHRSP is thought to decrease the size of the subacromial space, therefore decreasing the space of the supraspinatus outlet and increasing one's potential toward the acquisition of subacromial impingement syndrome.

Posterior Shoulder Tightness and/or Contracture

A second commonly described predisposing factor of subacromial impingement syndrome, as well as internal impingement, is posterior shoulder tightness or contracture. Myers et al[32] found that throwers with pathologic internal impingement demonstrated increased posterior shoulder tightness, with their GIRD to ERG ratio less than or equal to one.[33] GIRD is the acronym used to describe **G**lenohumeral **I**nternal **R**otation **D**eficit based on the difference in internal rotation between the involved and uninvolved shoulder. ERG, on the other hand, is the acronym used to describe **E**xternal **R**otation **G**ain based on the difference in external rotation between the involved and uninvolved shoulder. The authors believe that a GIRD greater than the ERG was a predisposing factor toward the acquisition of shoulder soft tissue pathology, specifically internal impingement. Pathologic internal impingement refers to a condition in which there is an impingement occurring of the supraspinatus and/or the infraspinatus tendons between the greater tuberosity of the humerus and the posterior aspect of the glenoid rim.[6, 7, 32] Typically, this type of impingement will present itself as posterior shoulder pain. Chronic internal impingement, whether due to posterior shoulder tightness or some other etiology, may result in lesion development on the involved tendon(s). Internal impingement may also be related to posteriorsuperior glenoid labrum fraying (i.e. superior labral anterior posterior (SLAP) lesions).

Kibler et al[9] theorized that an inflexibility or contracture of the posterior capsule would increase scapular protraction and scapular depression. Excessive scapular protraction due to a contracture of the posterior joint capsule is thought to cause subacromial impingement as the scapula internally rotates and tilts anteriorly, thereby narrowing the subacromial space.[10, 19, 25]

Anterior Coracoid Musculature Tightness and/or Contracture

Another commonly described possible predisposing factor of subacromial impingement syndrome is anterior musculature tightness or contracture. Kibler et al[9] reported from their clinical observation that subjects with scapular dyskinesis exhibit tightness of the pectoralis minor and short head of the biceps brachii, as well as increased anterior scapular tilt. Tightness of the anterior musculature is thought to cause the coracoid process to be pulled anteriorly due to its increased tension, resulting in increased scapular protraction, scapular depression, and scapular downward rotation. Borstad et al[34] compared subjects within a long pectoralis minor group with those in a short pectoralis minor group and found that the long pectoralis minor group demonstrated significantly more scapular posterior tipping when compared to the short pectoralis minor group at 90 degrees of humeral elevation. Individuals with increased pectoralis minor tension were demonstrated to have increased scapular internal rotation with humeral elevation. Excessive scapular protraction due to tightness of the anterior coracoid muscles is likely to cause subacromial impingement as the scapula internally rotates and tilts anteriorly.[10, 19, 25]

The findings of Borstad et al[34] are consistent with Ludewig et al[35] who concluded that increased pectoralis minor tightness impedes normal scapular posterior tipping motion. In

addition to these findings, Ludewig et al[35] speculated that rhomboid major, rhomboid minor, and levator scapulae tightness may impede the normal scapular upward rotation. These authors theorize that decreased scapular posterior tipping, in conjunction with decreased scapular upward rotation and decreased scapular retraction, may have the potential to contribute to clinical pathology of the shoulder's soft tissue structures.[9, 34, 35]

Weakness and/or Inhibition-based Muscle Dysfunction of Scapular Stabilizers

A fourth commonly described predisposing factor of subacromial impingement syndrome is periscapular muscle weakness and/or inhibition. The upper extremity kinetic chain begins at the shoulder joint complex, with the scapula serving as the base of stability during the performance of overhead functional movement patterns. Overuse of an athlete's sport-specific upper extremity movement system can result in the development of upper extremity muscle imbalances. As was previously mentioned, there is adaptive muscle shortening of the pectoralis minor in some athletes, but there is also adaptive muscle weakening of the middle trapezius, lower trapezius, serratus anterior, rhomboid minor, and rhomboid major in these individuals.[9, 34, 35] These adaptations are known as upper-cross syndrome.[26] Within muscle altered length-tension relationships of scapular force couple muscles compromise dynamic joint stability, specifically altering the upper trapezius to middle trapezius, lower trapezius, and serratus anterior force couple ratio.[13] Most researchers agree that the serratus anterior and the lower trapezius are inherently the most susceptible of the scapular stabilizers to the effects of both weakness and inhibition.[15, 21, 23, 36] When the serratus anterior and lower trapezius suffer weaknesses, there is a significant decrease in scapular upward rotation.[37] Also, inadequate serratus anterior function prevents the

anchoring of the inferior scapular angle to the wall of the thorax, thus also preventing smooth scapular movement.[3] This is commonly referred to as a "winging" effect of the involved scapula.

Ludewig et al[10] compared both shoulder electromyographic (EMG) activity and kinematics of subjects with symptoms of subacromial impingement syndrome to an asymptomatic control group as each group performed humeral elevation in the scapular plane (scaption) under three hand-held load conditions: no load, 2.3-kg load, and 4.6-kg load. It was revealed that subjects in the subacromial impingement syndrome group demonstrated a statistically significant 9% reduction in serratus anterior muscle activity across load and phase conditions as well as a very subtle increase in the upper trapezius muscle activity. These findings are consistent with those demonstrated by Wadsworth et al[23], where the activation of the serratus anterior muscle of swimmers suffering from subacromial impingement syndrome was found to be delayed by three times when performing a scapular plane elevation task as compared to the asymptomatic control group.

Myers et al[13] stated that poor scapular positioning and movement can lead to altered length-tension relationships of the periscapular musculature, thus adversely affecting their ability to generate force. Similarly, Kibler[19] found that subjects displaying scapular dyskinesis demonstrated a lack of stability regarding their periscapular musculature, leading the author to theorize that dysfunctional scapular muscle performance is indeed a contributing factor for scapular dyskinesis. Weaknesses of the scapular stabilizers resulted in force production alterations, and thus an overall decrease in their development of maximal torque. Kibler[19] stated that if the scapula became a truly unstable base, a compensatory reversal of origin and insertion characteristics result. In the pathologic population of

individuals suffering from scapular dyskinesis, the authors observed the scapula actually being pulled laterally into a position of scapular protraction and scapular external rotation by the humeral distal insertion sites, which were consequently acting as the more stable base.

Altered Neuromuscular Control of Glenohumeral and Scapular Force Couples

Altered neuromuscular control patterns act very similar in effect to that of muscle imbalances due to weakness and/or inhibition. These alterations of the shoulder joint complex's neuromuscular control patterns are the fifth commonly described predisposing factor of subacromial impingement syndrome. As defined by Myers et al[27], neuromuscular control refers to the unconscious control of dynamic restraints occurring in preparation and in response to joint motion and loading for the purpose of maintaining functional joint stability. One component of neuromuscular control is proprioception – the specialized variation of sensory modality of touch that encompasses the sensation of joint movement (kinesthesia) and joint position – which is transmitted via intrafusal muscles spindles to the central nervous system.[5] In a pre-test/post-test research design with a fatigue intervention, Myers et al[31] required subjects to perform either an active angle-reproduction test or a single-arm dynamic stability test both before and after performing a fatigue protocol. Fatigue tasks utilized continuous concentric shoulder internal and external rotation. Myers et al[31] concluded that muscle fatigue desensitized muscle spindle threshold, thereby possibly decreasing afferent feedback to the central nervous system. The authors theorized that fatigue had decreased proprioception by affecting the mechanoreceptors present within the musculature of the shoulder, thus hindering the neuromuscular control of joint stability.

Similar to previous findings, Myers et al[27] found that decreased proprioception and altered neuromuscular control resulted in functional instabilities of the glenohumeral joint. Myers et al[38] stated that more sensitive muscle spindles are capable of detecting stretch caused by perturbation, thus producing rapid reflexive responses. Myers et al[24] also concluded that there exists altered neuromuscular control patterns in subjects with anterior glenohumeral instability. Specifically, these subjects displayed a suppression of pectoralis major and biceps brachii mean activation, increased peak activation of the rotator cuff muscles, a slower biceps brachii reflex latency, and suppression of both the supraspinatus and subscapularis muscles. Any loss of dynamic joint stability, whether acute or chronic in mechanism, can potentially lead to subacromial impingement of the shoulder's soft tissue structures. As laxity increases about the glenohumeral joint, in turn so does the potential for subacromial impingement symptoms secondary to unstable episodes (i.e. glenohumeral dislocation/subluxation).[5] In general glenohumeral instabilities are thought to manifest themselves as shoulder pain due to subacromial impingement syndrome.[39]

Scapular Dyskinesis

One final, and possibly the most significant, predisposing factor of subacromial impingement syndrome is the presence of scapular dyskinesis. By definition scapular dyskinesis is an alteration in the normal position or motion of the scapula during coupled scapulohumeral movements.[9] Scapular dyskinesis is associated with shoulder pain and typically coincides with any combination of the above possible predisposing factors of subacromial impingement syndrome.[1, 6-10, 13, 18, 19] Any combination of these conditions can provide an adequate mechanism of injury for the initial development of subacromial

impingement syndrome.[9] It is speculated that the result of subacromial impingement is inhibition or disorganization of scapular muscle activation patterns, and ultimately, scapular dyskinesis.[9] Scapular kinematic alterations have been previously identified in subjects with shoulder impingement syndrome.[4, 5, 8, 9, 11, 12, 18] Studies comparing the scapular kinematics of healthy subjects and subjects with subacromial impingement have revealed that the pathologic subjects demonstrated increased anterior tilt, increased internal rotation, and decreased upward rotation of the scapula during humeral elevation. Researchers concluded that such altered scapular kinematics may decrease the size of the subacromial space, thus increasing one's susceptibility for soft tissue impingement.

Ebaugh et al[29], Tsai et al[16], and Su et al[14] utilized fatigue tasks to compare subject scapular kinematics under pre-fatigue and post-fatigue conditions. The authors theorized that fatigue was capable of producing scapular kinematic changes, specifically a decrease of scapular posterior tilt following completion of the fatigue protocol. Such altered kinematic patterns parallel those that exist in populations with shoulder girdle muscle weaknesses and imbalances. Specifically, Ludewig et al[10] found that the subacromial impingement group demonstrated a significant 4.1 degree decrease in scapular upward rotation at 60 degrees of abduction in the scapular plane, a significant 5.8 degree increase in scapular anterior tilting at 120 degrees of abduction in the scapular plane, and statistically significant increases in scapular internal rotation under all loaded conditions.

Kibler et al[9] stated that scapular dyskinesis is the result of both altered muscle activation patterns of the scapular force couples due to painful conditions around the shoulder as well as excessive thoracic kyphosis. Pink et al[3] theorized that improper scapulohumeral positioning (i.e. thoracic kyphosis) that presented as an adaptation of

swimming place increased stress on the anterior capsular structures of the shoulder joint complex, causing an increase of anterior humeral translation with secondary impingement of the rotator cuff tendons.

McClure et al[4] found that subjects within a subacromial impingement group presented with compensatory strategies to avoid the pain associated with subacromial impingement syndrome. These compensatory strategies included both greater upward rotation and clavicular elevation in midrange positions (90 to 120 degrees) of flexion and greater posterior tilt, upward rotation, and clavicular retraction at midrange positions (90 to 120 degrees) of scaption. These authors also found significantly less range of motion (i.e. flexion and scaption) and less isometric force production (i.e. rotator cuff musculature force production) for all measures when compared with an asymptomatic control group.

In a study comparing construction workers with and without symptoms of shoulder impingement, Ludewig et al[18] results contrast those found by McClure et al[4] in regards to altered scapular kinematics in the presence of an underlying pathology. Unlike the findings of McClure et al[4] where the subacromial impingement syndrome group displayed increased scapular upward rotation and increased scapular posterior tilt when compared to a healthy, control group, Ludewig et al[18] found the subacromial impingement syndrome group to have decreased scapular upward rotation and decreased scapular posterior tilt when compared to a healthy, control group. It should be noted that Ludewig et al[18] implemented loaded conditions, whereas McClure et al[4] did not in an attempt to prevent inducing or increasing symptoms of pain.

Bandholm et al[40] supported the findings of McClure et al[4] regarding pain's effects on force production. Using healthy subjects and experimental pain, the researchers

demonstrated pain's ability to inhibit maximal force steadiness and related muscle activity while having no effect on the shoulder musculature's contractile properties. The authors believed that the excitation of the muscle nociceptor afferents facilitate inhibitory pathways during muscle agonist activity.[40]

Regardless of what acts as the precursor toward the acquisition of scapular dyskinesis, its presence is problematic until both treated and corrected. Burkhart et al[1] use the acronym SICK when describing a specific form of scapular dyskinesis. SICK scapula syndrome refers to the presence of **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement. Based upon both the literature and screening guidelines of Burkhart and Morgan[1], the inclusion criteria for the SICK scapula group is based on a 0 to 20 point scale, with 0 representing complete shoulder health and 20 representing severe, symptomatic SICK scapula syndrome. Typically upon screening, athletes who present with symptomatic SICK scapula syndrome will score somewhere within the range of 10 to 14 on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] Athletes diagnosed with SICK scapula syndrome typically present with a unilateral lowered and anteriorly tilted scapula with accompanied anterior shoulder pain on the involved side. While the validity and the reliability of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] has not been established, the scale is used in clinical settings to aid in the objective shoulder evaluation process.

Burkhart et al[1], as well as Kibler et al[9], have presented their view on the association between scapular dysfunction and shoulder pathology based on their clinical observation. The authors theorize that such scapular asymmetries are a sign of underlying alteration in scapular muscle activation, resulting in altered scapular kinematics during overhead activity.

The authors also theorized that altered scapular kinematics are strongly associated with various shoulder pathologies such as subacromial impingement syndrome, rotator cuff lesion, and labral tear. However, it must be mentioned that a limitation of such findings is the lack of a comparison to the asymptomatic population. Future research should focus on case-control comparisons between healthy subjects and subjects suffering from some form of scapular biomechanical dysfunction, such as SICK scapula syndrome.

Researchers agree that alterations of scapulothoracic kinematics can stress the shoulder girdle's soft tissues. The high prevalence of such kinematic dysfunction found at clinical settings demands the development of effective therapeutic exercises designed specifically to correct scapular dyskinesis by both stretching and strengthening the appropriate tissues. Identification of the dysfunctional tissues associated with the dyskinesis will allow clinicians to prescribe rehabilitation specific to the needs of the patients with the condition. Corrective exercise protocols play an essential role in the rehabilitation of athletes with scapular impairments. Furthermore, it can be assumed that the implementation of adequate prehabilitation exercise protocols is the essential step toward the prevention of scapular dyskinesis in the athletic population.

Methodological Considerations for Electromyographic (EMG) Analysis

Electromyographic (EMG) analysis has traditionally been utilized as a dependable source of data collection in medical research, typically demonstrating both good reliability and validity[10, 15, 16, 20-23, 36, 38, 40-45], with $ICC_{(2,1)}$ values in an acceptable range.[15, 46] In sports medicine research, surface EMG is utilized to study neuromuscular activation in targeted muscles during both postural tasks and functional movements. When a research design is

focused on the study of kinematics pertaining to a specific population, muscle activation can be an extremely telling dependent variable. Because kinematic function is predominately influenced by the muscle activity surrounding a given articulation, it can be theorized that kinematic dysfunction may be the result of altered periarticular muscle activity.

In regards to the study of scapular kinematics however, there is a weak body of literature concerning the analysis of scapular stabilizer EMG. Researchers have shown in comparison studies that dysfunctional kinematics do exist in individuals suffering from shoulder pathology when compared to healthy, control subjects[4, 5, 8-12, 14, 23, 32], and authors have also qualitatively assessed and described specific dyskinetic syndromes (i.e. SICK scapula syndrome) in similar pathological populations.[1, 3, 6, 7, 9, 19] Currently however, researchers are limited in their understanding of the muscle deficiencies present in individuals suffering from scapular dyskinesis, and as a result, must speculate on the associated scapular force couple dysfunction occurring in afflicted individuals. This speculation is the driving force behind the implementation of therapeutic exercises designed specifically to restore normal scapular kinematics via scapular force couple re-education.

In healthy individuals, overhead functional movement requires scapular force couples to stabilize the scapula, allowing for both the absorption and transference of forces and moments from the upper extremity to and from the trunk and lower extremity.[19] McMahon et al[47] describe a synergistic relationship existing between each of the scapular force couple muscles. This synergistic relationship is of paramount importance regarding normal scapular kinematics. Researchers have found significant group differences in scapular muscle activity between healthy and patient populations[3-5, 10, 12, 21, 23, 24, 40, 43, 45, 47-50], but variation exists among each study's EMG dependent variable. When studying the muscle activity of the

scapular stabilizer muscles, researchers typically manipulate their usage of EMG to analyze one of three specific variables: muscle activation recruitment sequence, peak muscle activation amplitude, or mean muscle activation amplitude.

When comparing healthy overhead athletes with those who presented with impingement symptoms during an isokinetic perturbation test, Cools et al[21] found that athletes with impingement showed a delay in muscle activation for both the middle and lower trapezius muscles. From these findings, the authors concluded that overhead athletes with impingement symptoms show abnormal muscle recruitment patterns for both the middle and lower trapezius muscles.[21]

In a similar research design with an implemented fatigue protocol and no patient population, Cools et al[48] found that, following fatigue, the upper trapezius, middle trapezius, and lower trapezius muscles were recruited secondary to the onset of the deltoid muscle. The authors then theorized that shoulder muscle recruitment patterns are delayed following fatigue but not altered.[48] The implications of fatigue on muscle recruitment could be detrimental regarding its effects on normal scapular kinematics for athletes already suffering from scapular stabilizer insufficiency.

Wadsworth et al[23] also utilized muscle recruitment EMG analysis. In a comparison study of healthy swimmers and swimmers suffering from subacromial impingement syndrome, the researchers found the activation of the serratus anterior muscle to be delayed by three times when compared to healthy swimmers while performing a scaption task.

When analyzing peak EMG for the scapular stabilizers, Pink et al[3] found the serratus anterior muscle to have decreased activation in a population of swimmers with painful shoulders when compared to healthy swimmers while swimming. These researchers also

found that the rhomboid major and rhomboid minor muscles of the impingement swimmers displayed increased peak EMG when compared to the healthy swimmers. As a result, the authors concluded that the already failing serratus anterior muscle was being forced to work in direct opposition to the rhomboid muscles as they function to downwardly rotate the scapula.[3]

Peak EMG analysis has also been utilized to assess the efficacy of specific scapular strengthening exercises implemented in therapeutic exercise protocols. In a study designed to examine the activity of scapular muscles throughout each of 16 different exercises, Moseley et al[20] concluded there to be four most effective scapular stabilizer strengthening exercises: scaption, rowing, push-up with a plus, and press-up.

Utilizing mean EMG activation, Ludewig et al[10] compared subjects with symptoms of subacromial impingement syndrome to an asymptomatic control group as each group performed a scaption task under three loaded conditions. Significant findings revealed a reduction in the serratus anterior muscle activity in the impingement subjects across load and phase conditions when compared to healthy subjects. The researchers also found there to be a very subtle increase in the upper trapezius muscle activity in impingement subjects.

In a comparison study conducted by Cools et al[49] between overhead athletes with and without impingement symptoms, mean EMG activity of the upper trapezius, middle trapezius, and lower trapezius were measured during isokinetic glenohumeral abduction and external rotation. The results showed a significant increase in upper trapezius activity during both glenohumeral abduction and glenohumeral external rotation in the patient group. The findings of this study also revealed decreased activity in the lower trapezius during the

glenohumeral abduction task and decreased activity in the middle trapezius during the glenohumeral external rotation task when compared to healthy subjects.

Implications for rehabilitation can also be addressed with the usage of mean amplitude EMG. In a study conducted by Cools et al[51], the activation of the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles were analyzed during twelve commonly used shoulder therapeutic exercises. The researchers then calculated both intermuscular and intramuscular balance ratios. Based on the results of this study, the authors suggest the usage of side-lying external rotation, side-lying forward flexion, prone horizontal abduction with external rotation, and prone extension exercises as the most effective exercises at promoting lower trapezius and middle trapezius activity while minimizing the activation of the upper trapezius.[51]

The purpose of this study is to validate the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale, developed by Burkhart et al[1], by predicting subject score from scapular stabilizer muscle activity. For the purposes of this study, mean amplitude EMG was selected as the most appropriate dependent variable for analyzing scapular muscle activation occurring during both the ascending and descending phases of a functional task.

CHAPTER III

METHODOLOGY

Clinical Relevance

The purpose of this study was to validate the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] by predicting subject score from scapular stabilizer muscle activity. Determining the validity of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] may allow clinicians to more readily utilize this scale as an accurate screening tool toward the identification of SICK scapula syndrome. Understanding the mean electromyographic (EMG) activity of the scapular muscles in overhead athletes with SICK scapula syndrome will help sports medicine professionals to prescribe these patients with rehabilitation exercises specific to their deficits, which may lead to better treatment outcome.

With the highly repetitious nature of overhead movement in athletics, even a small degree of scapular malalignment or dyskinesis may overload the shoulder's soft tissue structures over time. As was previously mentioned, the union between the scapula and the axial skeleton is extremely limited to the surface area of the acromioclavicular joint. As a result of this minimal articulation, scapular stability and mobility are largely dictated by the numerous muscles originating and inserting on the scapular surface. Understanding the adaptive scapular muscle activation deficiencies associated with SICK scapula syndrome, as well as the condition's affect on scapular force couple synchronization, will allow clinicians to implement specific rehabilitation exercises when dealing with affected overhead athletes. Corrective exercises implemented to target the muscles identified to be dysfunctional in this

study can help restore ideal scapular force couple synchronization, thus allowing the scapula to move harmoniously with the moving humerus.

Population and Recruitment

Subjects were recruited from a university population of NCAA Division I overhead athletes and/or recreational club overhead athletes at The University of North Carolina at Chapel Hill. Subjects were both male and female and were between the ages of 18 and 25 years old.

Based upon a recent study performed at UNC-CH with similar dependent variables, forty subjects were required to achieve a statistical significance level of 0.05 with a power of 0.80 (see Table 1).[49, 52] Utilizing both the mean difference and standard deviation of these similar research studies, the effect size index for each dependent variable was calculated (see Figure 2). A priori power was determined for each dependent variable using a standard Power Table and the calculated effect size index.

Subject Inclusion Criteria

Subjects qualifying for this study were NCAA Division I overhead athletes and/or recreational club overhead athletes who participate in an overhead sport for a duration of at least 30 minutes per session for at least 3 individual sessions per week.

Subject Exclusion Criteria

Individuals with a history of shoulder and/or neck surgery, rotator cuff tear, cervical spine pathology, history of acute-onset shoulder pathology within the past six months, adhesive capsulitis, history of unstable episodes within the past six months (glenohumeral subluxation, dislocation, self-subluxation), or scoliosis were excluded from the study.

Research Design

The selected research design was quasi-experimental in nature, specifically a nonequivalent one group design with a counterbalancing of tasks. The study took place in an approximately 90 minute session. Prior to testing, subjects were screened for both inclusion and exclusion criteria as well as skill and activity level.

Procedure and Description of Tasks

Upon entering the lab, each subject was briefed on testing procedures and signed and received a personal copy of the Consent to Act as a Human Subject form. Subjects then filled out a medical history form, underwent screening based on the SICK scapula, Static Measurements, 0 to 20 Point Rating Scale (see Figure 1), and were measured for both height and weight. Limb selection was dictated either by 1) the subject's involved side in those who had reported symptoms of shoulder pain or 2) the dominant side in those who had reported to be asymptomatic for shoulder pain. Subjects were screened a total of twice (one screening per principle investigator), and an average of the two scores was calculated and utilized for later statistical analysis.

Counterbalancing for functional tasks was implemented following the subject's briefing session, where subjects selected a random task completion order of one (glenohumeral elevation in the sagittal plane) and two (glenohumeral elevation in the scapular plane).

After the setup for both EMG and kinematic analysis (see Measurement and Instrumentation section) was completed, subjects completed the following tasks: glenohumeral elevation in the sagittal plane (flexion) and glenohumeral elevation in the scapular plane (scaption). Each of the two humeral elevation tasks required the subject to lift their arm for ten repetitions per task. The sagittal plane was defined as the plane parallel to the sagittal plane of the thorax. The scapular plane was defined as the plane 30 degrees anterior to the frontal plane of the thorax.

Subjects completed their full range of motion at a controlled movement velocity by moving in time with a digital metronome set at 1 beat per second. Each functional task required ten continuous repetitions, with each repetition lasting approximately four seconds (two-second ascending phase, two-second descending phase). A guiding pole made of PVC pipe was used as a guide for both flexion and scaption. For the flexion task, the guide pole was placed in the sagittal plane, parallel to the sagittal plane of the thorax. For the scaption task, the guide pole was placed in the scapular plane, 30 degrees anterior to the frontal plane.

Description of Tasks

Glenohumeral flexion tasks were performed through a range of motion of approximately 0 degrees humeral elevation to approximately 180 degrees of humeral elevation in the sagittal plane. The subject elevated their arm until they were able to reach

their terminal end point in their available range of motion. The subject then returned to the starting position. Subjects maintained a neutral hand position throughout the ten-repetition task.

Glenohumeral scaption tasks were performed through a range of motion of approximately 0 degrees humeral elevation to approximately 180 degrees of humeral elevation in the scapular plane. The subject elevated their arm until they were able to reach their terminal end point in their available range of motion. The subject then returned to the starting position. Subjects maintained a neutral hand position throughout the ten-repetition task.

Measurement and Instrumentation

SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale

The SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale is a grading system used for assessing the severity of scapular malposition based on 1) infera (i.e. the visual appearance of a dropped scapula due to scapular tilting or protraction), 2) lateral displacement, and 3) abduction. All measurements were made statically with the patients standing erect with arms relaxed at their side. The measurement of infera is the difference in vertical height of the superomedial scapular angle of the dropped scapula in centimeters compared with the contralateral superomedial angle.[1] While limited in both reliability and external validity, the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale was used to give a quantitative sense of SICK scapula syndrome severity.

Prior to the study, reliability and precision of this scale were established from a small pilot study by the chief investigators using intraclass correlation coefficient (ICC) and

standard error of measurement (SEM). The inter-session reliability and precision were calculated to yield an ICC of 0.682 and SEM of 1.44 points, respectively. The inter-tester reliability and precision were calculated to yield an ICC of 0.684 and SEM of 1.18 points, respectively.

Kinematic Analysis

A Motion Star (Ascension Technologies Inc, Burlington, Vt) electromagnetic motion analysis mini bird system controlled by the Motion Monitor (Innovative Sports Training Inc, Chicago, Ill) software was used to assess shoulder complex kinematics at a sampling rate of 100 Hz. Previous research demonstrates that electromagnetic tracking systems provide valid, accurate, and reliable measures of dynamic motion that are comparable to camera based systems.[53-57] The Motion Star system has been shown to be accurate within 1.8 mm for linear displacements and 0.5º for angular displacements.[15] Separate electromagnetic receivers were attached to the thorax, scapula, and humerus. The thorax sensor was placed over the spinous process of the seventh cervical vertebrae (C7), and the scapula receiver was placed over the broad, flat surface of the posterolateral acromion. The humeral receiver was placed over the posterior aspect of the humerus, distal to the triceps muscle belly. The humeral kinematic data were used to define the ascending and descending phases during each humeral elevation task. All receivers were attached using double-sided tape. An elastic wrap was used to further secure the humeral receiver. Before receiver application, the skin was dried and sprayed with an adhesive spray to improve adherence.

Electromyographic Analysis

Electromyographic (EMG) muscle activation analyses were performed to measure the mean amplitude of the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles using a Delsys Bagnoli-8 EMG (Boston, MA) with differential amplication, CMRR >80 dB, input impedance >1015//0.2 ohm//pF, SNR >40 dB using an 8 channel amplifier. The EMG signal was amplified by a factor of 1000 over a bandwidth of 0.01 to 2000 Hz, passed via an A/D converter (National Instruments, Austin, TX) sampling at 1000 Hz then corrected for DC bias. Raw EMG data were collected by the Motion Monitor software and stored for analysis. The electrodes were 19.8 mm wide and 35 mm long with approximately 10 mm between contacts.

Before applying surface electrodes, the subject's skin was shaved, cleaned with alcohol, and lightly abraded to ensure good electrode contact and transmission. We fixed a bar Ag/AgCl single differential surface electrode (Delsys Inc., Boston, MA) on the midpoint of each muscle belly perpendicular to the muscle fiber direction using surgical tape and adhesive stickers. The specified electrode placement has been used in a number of studies.[15, 21, 43] Electrodes were placed according to previously published guidelines on the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles' fibers in the following arrangement.[15, 21, 43]

Upper trapezius: one half the distance from the mastoid process to the root of the spine of the scapula approximately at the angle of the neck and shoulder

Middle trapezius: one half the distance from the spine of the scapula to the spinous process in a position perpendicular to the spine.

Lower trapezius: two finger widths medial to the inferior angle of the scapula on 45-degree angle towards T10

Serratus anterior: below the axilla, anterior to latissimus dorsi, placed over 4th through 6th ribs angled at 30 degrees above the nipple line

A carbon reference electrode was placed over the non-involved olecranon process. Isometric manual muscle tests were performed to ensure accurate placement of electrodes and to measure and record maximal voluntary isometric contraction (MVIC) EMG. Subjects performed each MVIC measure for five seconds.

Manual muscle tests to determine MVIC were randomized and performed according to the procedures described by Kendall et al.[58] Prior to testing, subjects performed one sub-maximal contraction to familiarize themselves with proper form for each manual muscle test. Following this warm-up and learning session, subjects performed three maximal voluntary isometric contractions measured for each muscle with a one-minute rest period between each muscle and a thirty-second rest period between each trial. The peak mean force for a five-second period was recorded. Mean amplitude value for the three trials was used to express muscle activity during tasks as a percentage of muscle activity during the maximal isometric contraction (%MVIC).

Upper Trapezius MVIC Assessment

Testing of the upper trapezius was performed with the subject seated with their arms at their side. The tester stood behind the subject and gave the instructions to "shrug your shoulders straight up and turn your face in the opposite direction" and hold that position.

The tester provided a downward force on the superior aspect of the acromion and back of the head for five seconds. The subject was then instructed to "relax".[15, 22]

Middle Trapezius MVIC Assessment

Testing of the middle trapezius was performed with the subject lying prone on a table with their shoulder at the edge of the table and both arms externally rotated and extended at their sides to approximately 90 degrees of abduction. The tester stood on the dominant shoulder side and gave the instructions to "lift both arms up placing your shoulder blades in your opposite back pocket". The tester provided a downward force on the forearm for five seconds. The subject was then instructed to "relax".[15, 22, 43]

Lower Trapezius MVIC Assessment

Testing of the lower trapezius was performed with the subject lying prone on a table with their shoulder at the edge of the table and both arms externally rotated and extended overhead to approximately 130 degrees of abduction. The tester stood on the dominant shoulder side and gave the instructions to "lift both arms up placing your shoulder blades in your opposite back pocket". The tester provided a downward force on the forearm for five seconds. The subject was then instructed to "relax".[15, 22, 43]

Serratus Anterior MVIC Assessment

Testing of the serratus anterior was performed with the subject in a seated position with their arm internally rotated and elevated in the scapular plane to approximately 120 degrees. The tester was positioned standing beside the subject and gave the instructions to

"lift your arm out and up; don't let me push you down". The tester provided a downward force on the superior aspect of the arm at the elbow while providing pressure at the lateral, inferior angle of the scapula inwards for five seconds. The subject was then instructed to "relax". This position has been shown to yield the most reliable and highest MVIC values for the serratus anterior.[15, 22, 43]

Data Reduction

Kinematic Data Reduction

Raw kinematic data were low pass filtered with a fourth-order zero-phase shift at a 6.6 Hz cut off frequency.[10, 13, 52, 59] [10, 13, 60, 61] Glenohumeral elevation angles of 0º and 120º were identified for the purposes of analyzing mean EMG amplitude between those points during the ascending and descending phases of glenohumeral elevation.

The local coordinate system for each segment were defined according to the recommendations established by the International Shoulder Group of the International Society of Biomechanics.[62] Two points were first defined as the segment's longitudinal axis with a third point defining the plane. A second axis was determined perpendicular to the plane, and the third axis was defined as perpendicular to both of the first two axes. When standing in a neutral stance, the orthogonal coordinate system for each segment was vertical (y-axis), horizontal to the right (x-axis), and posterior (z-axis). Matrix transformations for each of the segments were used to move from the global to local coordinate systems, producing a 4 x 4 position and orientation matrix.

Euler-angle decompositions were used to describe humeral orientation with respect to the thorax. Humeral orientation was determined as rotation about the y-axis of the humerus

(plane of elevation), rotation about the z-axis of the humerus (elevation), and rotation about the y-axis of the humerus (axial rotation). Each of these rotations was chosen based on the recommendations of the International Shoulder Group.[62] The Euler-angle sequences were used to most closely represent clinical definitions of movements and to decrease mathematical inconsistencies.[63]

Electromyographic Data Reduction

All electromyographic (EMG) data reduction was done using the Motion Monitor software. EMG data were filtered using a band-pass filter (10-350 Hz) and a Butterworth filter (4^{th} order, recursive, zero-phase lag). The root mean square (RMS) of the EMG signal over a 50 ms time constant was taken to further smooth the data. The MVIC was calculated as the mean of the EMG activity during the 5-second trial.

Mean EMG amplitude was calculated, with the ascending phase (0^{o}-120^{o}) and the descending phase (120-0^{o}) being determined by the humeral elevation angle data. For each repetition, the lowest point of humeral elevation represented the initiation of the ascending phase, and the highest point of humeral elevation represented the cessation of the ascending phase and the initiation of the descending phase. The subsequent lowest point of humeral elevation represented the cessation of the descending phase. The EMG activity during both the ascending and descending phases were calculated as an average of the mean EMG amplitude over each phase of motion for the middle 5 repetitions of each muscle tested. The mean EMG activity independent variables were normalized to the MVICs obtained prior to the trials, and were expressed as a percentage of the MVIC (%MVIC). Variables were

calculated and processed using Matlab R2007a (The MathWorks Inc., Natick, Massachusetts).

Statistical Analysis

Mean EMG amplitude data were used to represent muscle activation over each phase of humeral elevation for the upper trapezius, middle trapezius, lower trapezius, and serratus anterior.

Multiple linear regressions were used to predict the scores derived from the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] using the Statistical Package for Social Sciences (SPSS 15.0, Inc, Chicago, IL)(see Table 2). Statistical significance for all comparisons was set a priori at alpha-level of 0.05.

CHAPTER IV

RESULTS

Descriptive Statistics

Forty Division I overhead athletes and/or recreational club overhead athletes (10 softball players, 20 swimmers, 9 volleyball players, and 1 water polo player; 33 right arm dominant, 7 left arm dominant) participated in this study. Due to errors in data, three subjects were dropped from this study. Out of the remaining 37 participants, 20 reported to be currently experiencing shoulder pain. The descriptive statistics on demographics and subject SICK scapula syndrome score are presented in Table 3. The breakdown of SICK scapula syndrome score for all subjects is presented in Table 4. Mean amplitude EMG was calculated for the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles (see Table 5).

Upper Trapezius

A simple linear regression was performed to assess how the upper trapezius mean muscle activation amplitude during a sagittal and scapular plane elevation task was able to predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] A simple linear regression analysis for the ascending phase of the flexion task revealed that the mean upper trapezius muscle activation amplitude did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating

Scale[1], (F = 0.568, df = 35, p = 0.456), with an r^2 of 0.016 (see FIGURE 3). Regression analysis for the descending phase of the flexion task also revealed no significant findings, (F = 0.092, df = 35, p = 0.764), with an r^2 of 0.003 (see FIGURE 4). Regression analysis for the ascending phase of the scaption task revealed no significant findings, (F = 0.010, df = 35, p = 0.921), with an r^2 of less than 0.001 (see FIGURE 5). Regression analysis for the descending phase of the scaption task also revealed no significant findings, (F = 0.329, df = 35, p = 0.570), with an r^2 of 0.009 (see FIGURE 6).

Middle Trapezius

A simple linear regression was performed to assess how the middle trapezius mean muscle activation amplitude during a sagittal and scapular plane elevation task was able to predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] A simple linear regression analysis for the ascending phase of the flexion task revealed that the mean middle trapezius muscle activation amplitude did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], (F = 0.026, df = 35, p = 0.874), with an r^2 of 0.001 (see FIGURE 7). Regression analysis for the descending phase of the flexion task also revealed no significant findings, (F = 0.021, df = 35, p = 0.886), with an r^2 of 0.001 (see FIGURE 8). Regression analysis for the ascending phase of the scaption task revealed no significant findings, (F = 0.278, df = 35, p = 0.601), with an r^2 of 0.008 (see FIGURE 9). Regression analysis for the descending phase of the scaption task also revealed no significant findings, (F = 2.116, df = 35, p = 0.154), with an r^2 of 0.056 (see FIGURE 10).

Lower Trapezius

A simple linear regression was performed to assess how the lower trapezius mean muscle activation amplitude during a sagittal and scapular plane elevation task was able to predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] A simple linear regression analysis for the ascending phase of the flexion task revealed that the mean lower trapezius muscle activation amplitude did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], ($F = 0.026$, $df = 35$, $p = 0.873$), with an r^2 of 0.001 (see FIGURE 11). Regression analysis for the descending phase of the flexion task also revealed no significant findings, ($F = 0.010$, $df = 35$, $p = 0.919$), with an r^2 of less than 0.001 (see FIGURE 12). Regression analysis for the ascending phase of the scaption task revealed no significant findings, ($F = 0.000$, $df = 35$, $p = 0.998$), with an r^2 of less than 0.001 (see FIGURE 13). Regression analysis for the descending phase of the scaption task also revealed no significant findings, ($F = 0.196$, $df = 35$, $p = 0.660$), with an r^2 of 0.005 (see FIGURE 14).

Serratus Anterior

A simple linear regression was performed to assess how the serratus anterior mean muscle activation amplitude during a sagittal and scapular plane elevation task was able to predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] A simple linear regression analysis for the ascending phase of the flexion task revealed that the mean serratus anterior muscle activation amplitude task did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], ($F = 0.214$, $df = 35$, $p = 0.646$), with an r^2 of 0.006 (see FIGURE 15). Regression

analysis for the descending phase of the flexion task also revealed no significant findings, ($F = 0.000$, $df = 35$, $p = 0.998$), with an r^2 of less than 0.001 (see FIGURE 16). Regression analysis for the ascending phase of the scaption task revealed no significant findings, ($F = 0.087$, $df = 35$, $p = 0.770$), with an r^2 of 0.002 (see FIGURE 17). Regression analysis for the descending phase of the scaption task also revealed no significant findings, ($F = 0.000$, $df = 35$, $p = 0.982$), with an r^2 of less than 0.001 (see FIGURE 18).

CHAPTER V

DISCUSSION

The purpose of this study was to determine if subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] can be predicted from scapular stabilizer muscle activation. If muscle activation is found to be predictive, the hope is that some level of validation can be established for the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], theoretically allowing sports medicine professionals to better prescribe affected athletes with the most effective rehabilitation exercises when addressing their specific scapular movement and stabilizer strength deficits. Ultimately the goal clinically is to improve treatment outcome. The rationale for the validation of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] was to test its clinical efficacy as a predictor for the potential, presence, and/or severity of SICK scapula syndrome.

Our results indicate that mean muscle activation amplitude of the scapular stabilizers was not found to be a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] Our research hypothesis anticipated an increase in upper trapezius muscle activity, as well as decreases in middle trapezius, lower trapezius, and serratus anterior muscle activity as a valid predictor of symptomatic SICK scapula syndrome (i.e. 10 or greater on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]). Based on our findings however, we conclude that neither upper trapezius muscle over-activation nor middle trapezius, lower trapezius, or serratus anterior muscle under-

activation can reliably or accurately predict subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1]

Scapular stabilizer muscles dictate both scapular position and motion and are also considerably inhibited by the presence of pain.[40] Theoretically, EMG analysis of the scapular stabilizers should be an accurate predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], with the scale designed specifically to assess the presence of scapular malpositioning and shoulder pain.

The muscles originating and inserting on the scapula are the driving force behind all scapular and glenohumeral kinematic movement. Therefore, it was inferred that scapular positioning and motion are notably impacted when there is a disruption of this dynamic link via scapular muscle activation dysfunction.

Scapular muscle activation dysfunction refers specifically to strength deficits of the middle trapezius, lower trapezius, and serratus anterior muscles in relation to the upper trapezius muscle.[13] Most researchers agree that the serratus anterior and the lower trapezius are inherently the most susceptible of the scapular stabilizers to the effects of both weakness and inhibition.[15, 21, 23, 36] When the serratus anterior and lower trapezius suffer weaknesses, there is a significant decrease in scapular upward rotation.[37] Also, inadequate serratus anterior function prevents the anchoring of the inferior scapular angle to the wall of the thorax, thus also preventing smooth scapular movement.[3] Essentially, scapular malpositioning and dyskinesis have a certain dependency on the presence of scapular stabilizer strength deficits.

Bandholm et al[40] studied the effects of pain on force steadiness and related muscle activity. Using healthy subjects and experimental pain, the researchers demonstrated pain's

ability to inhibit maximal force production while having no effect on musculature contractile properties. The authors believed that the excitation of the muscle nociceptor afferents facilitate inhibitory pathways during muscle agonist activity.[40]

Both Pink et al[3] and Ludewig et al[10] compared muscle activity in subjects with symptoms of subacromial impingement syndrome to an asymptomatic control group. Significant findings revealed decreased activation of the serratus anterior muscle in the impingement subjects when compared to healthy, control subjects. Similarly, Wadsworth et al[23] demonstrated that the activation of the serratus anterior muscle of swimmers suffering from subacromial impingement syndrome was found to be delayed by three times when performing a scapular plane elevation task as compared to the asymptomatic control group.

Cools et al[49] also utilized a comparison study to observe the differences in scapular muscle activity between overhead athletes with and without impingement symptoms. Here researchers measured the mean EMG activity of the upper trapezius, middle trapezius, and lower trapezius during isokinetic glenohumeral abduction and external rotation. Their results showed significantly decreased activity in the lower trapezius during the glenohumeral abduction task and decreased activity in the middle trapezius during the glenohumeral external rotation task in individuals with impingement symptoms when compared to healthy, control subjects.

In the presence of shoulder pain, the literature clearly supports the linkage of altered scapular stabilizer muscle activity and the initiation of some degree of scapular malpositioning and/or dyskinesis. Over half of the subjects tested in this study self-reported as having a painful shoulder, yet only four scored higher than 10 out of 20 points on the scale, which is a criteria clinically used to diagnose individual as having SICK scapula

syndrome. If subjects are categorically symptomatic for self-reported shoulder pain, yet qualify as "healthy" upon assessment, one must question both the sensitivity and accuracy of the instrumentation being utilized for pathology detection.

In attempting to explain why altered EMG of the scapular stabilizers was unable to predict SICK scapula syndrome score in this study, we must acknowledge flaws in both the structural and theoretic framework of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] Pilot work, an a priori reliability study, and the thesis project itself allowed researchers to screen over 100 athletes utilizing the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] Out of all the athletes who were screened, only four were clinically diagnosed as having a SICK scapula syndrome. The principal investigator suggests two structural flaws within the scale's framework. The first flaw is the ambiguity of the questionnaire and the second is the threshold scoring.

Questionnaire ambiguity was encountered in the form of subject confusion regarding the subjective portion of the screening process. One common example of such uncertainty often came in response to the question, "do you ever have pain on, in between, or around your shoulder blades?" Subjects seemed reluctant to answer yes but would later test positive for tenderness to palpation of scapular soft tissue structures. Athletes seemingly had the perception that muscular pain in the form of myofascial trigger points was unrelated to the subacromial and/or internal impingement symptoms they may have been experiencing. We believe this misunderstanding among athletes to be extremely reasonable considering the nature, source, and severity of their pain is so inherently different. Question clarification by the principal investigator may have allowed athletes to give a more appropriate response to a

subjective symptom question; however, we felt that doing so may affect the athlete responses.

Threshold scoring was utilized for the scapular malpositioning portion of subject screening. While this method was deemed the most appropriate to maintain adequate inter-tester reliability, it often resulted in a lower score than the amount of malposition would suggest. For example, while measuring scapular abduction, a subject may have a four-degree abduction discrepancy between scapulae in resting position but would receive no points, because it failed to reach the five-degree threshold that would have allotted one point.

As was previously mentioned, there are definitive links between shoulder pain, scapular malpositioning, and altered scapular stabilizer muscle activity, however we must acknowledge that combinations of the three can be mutually exclusive to one another. Shoulder pain can exist without the presence of scapular malpositioning and/or dyskinesis. Myers et al[32] found that throwers with pathologic internal impingement typically present with posterior shoulder pain. If screened utilizing the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], these athletes may receive up to 11 subjective and objective points but display no scapular malpositioning. In such instances, these athletes would be clinically diagnosed as having SICK scapula syndrome without the defining characteristics of **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement.

Suggesting some flaws with the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], we also recognize the asymptomatic scapular malpositionings demonstrated as healthy adaptations in overhead athletes. Myers et al[13] found an adaptive increase in scapular upward rotation among normal, healthy throwing athletes. This adaptation was believed by

the authors to assist in the achievement of subacromial clearance throughout the throwing movement pattern, thus acting as a means of preventing subacromial impingement. Possessing marked scapular malpositioning does not always mandate an involved symptomatic shoulder.

Lastly, scapular stabilizer deficits can exist without the presence of either shoulder pain or scapular malpositioning. Weakness and/or inhibition-based muscle dysfunction of the scapular stabilizers is a common trend among overhead athletes, where there is adaptive muscle shortening of the pectoralis minor in conjunction with an adaptive muscle weakening of the middle trapezius, lower trapezius, serratus anterior, rhomboid minor, and rhomboid major.[9, 34, 35] The presence of upper extremity muscle imbalances is generally accepted to be the result of sport-specific pattern dominance and can be entirely asymptomatic in nature.[9, 34, 35]

While one criticism regarding the theoretic framework of the scale refers to inflated scores without the true presence of scapular malpositioning, an inverse criticism is the relative difficulty of scoring in general, even in the presence of both shoulder pain and scapular malpositioning.

The subjective and objective portions of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] dually assess an athlete for a wide range of chronic shoulder pathologies, from AC joint sprain to thoracic outlet syndrome to subacromial impingement syndrome. While an athlete may possess all of the classic characteristics of SICK scapula syndrome (i.e. **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement), he or she may not score any points for AC joint irritation, TOS parathesias, or subacromial impingement syndrome. As a result,

subject score would be rather low on both the subjective and objective portions of the scale, thus allowing the subject to appear seemingly healthy. Because total subject score is heavily reliant on reports of pain in these two sections, only gross scapular malpositioning (i.e. > 15 degrees of scapular abduction) would result in a clinical diagnosis of SICK scapula syndrome. Studies comparing healthy subjects and subjects experiencing shoulder pain found only modest differences (i.e. < 5 degrees) in scapular kinematics between groups.[4, 5, 8, 9, 11, 12, 18] A 15-degree scapular asymmetry is not only uncommon among ill-maintenance shoulders, it is relatively non-existent among Division I overhead athletes.

The SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] was designed as a structured clinical tool that could be utilized by clinicians as a predictor for the potential, presence, and/or severity of SICK scapula syndrome. However, it appears to have rather poor predictive value. In this study, SICK scapula syndrome and other chronic shoulder pathologies proved to be far too multi-factorial in nature for one all encompassing number.

Limitations

Perhaps the greatest limitation of this study was the narrow and low-ended range of subject scores collected utilizing the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] While subject recruitment made no distinction regarding a need for either symptomatic or asymptomatic shoulders, upon screening, only four subjects scored higher than 11 out of a possible 20 points, which is a minimum cutoff score commonly used to clinically diagnose individual as having SICK scapula syndrome. Again, we attribute unexpected subject scoring to flaws of both the structural and theoretic framework of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1]

Due to in-season compliance issues, this study was subject to an unintentional exclusion of baseball players. Baseball players, specifically baseball pitchers, often experience shoulder or elbow pain that prevents them from participation in sports at some point in their careers.[44] Therefore, caution must be used in extrapolating our findings to a baseball pitcher population.

Future Research

Future research should seek to further identify scapular muscle dysfunction in symptomatic overhead athletes, specifically those suffering with SICK scapula syndrome. Based on our findings, we believe that it is necessary to conduct a large-scale study of overhead athletes, perhaps focusing on ill-maintained shoulders to better exemplify the condition in its most exaggerated form.

Another potential avenue regarding future research would be the construction of a more theoretically sound screening instrument, similar in principle to the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] SICK scapula syndrome represents a collection of signs and symptoms commonly seen in individuals with various shoulder pathologies. A more fine-tuned, pathology-specific screening tool may allow clinicians to more reliably identify and quantify shoulder injuries.

We acknowledge the findings listed in Table 4 when offering sound recommendations regarding the development of a new and theoretically improved screening instrument. By dissecting SICK scapula syndrome score for each of the forty subjects screened, we were able to tease out exactly where the bulk of point allotment occurred, specifically among those with self-reported shoulder pain. Based on score breakdown, we

conclude that ten characteristics of SICK scapula syndrome best exemplify the condition's signs and symptoms, thus serving as the most accurate predictors regarding its presence and severity. We recommend the following subjective questions for the presence of pain: coracoid process, periscapular, proximal lateral arm, and radicular symptoms. We recommend the following objective palpations and/or special tests for the presence of pain: coracoid process, superior medial scapular angle, and Hawkins-Kennedy Impingement Test. We recommend the following measurements for the determination of scapular malpositioning: infera 0 to 1 cm, lateral protraction 0 to 1 cm, and abduction 0 to 5 degrees.

We also recommend the implementation of a more detailed objective screening process; one which includes a postural assessment, observation and measurement of dynamic scapular positioning, soft-tissue mobility assessment, and scapular muscle strength assessment. Postural assessments should seek to identify and grade the presence of cervical lordosis, thoracic kyphosis, lumbar lordosis, pelvic rotations, and abnormal hip rotations that may affect scapular kinematics as energy is transferred through the kinetic chain from the lower extremity and core to the thorax and upper extremity.[9] Clinicians may perform a quick and effective postural assessment utilizing a plumb-line while observing the patient from a side-view.

Scapular position should be observed at rest and during loaded and unloaded humeral elevation. While in resting position, clinicians should observe the scapulae for signs of winging (i.e. excessive scapular internal rotation, scapular anterior tilt, and scapular elevation). Dynamic scapular motion should be assessed in both loaded and unloaded conditions. Johnson et al.[64] developed a protocol to detect abnormal scapular motion via the repetitive challenging of the scapulae under loaded conditions. The authors data indicated

that three tests were able to detect abnormal scapular motion: 1) observation of bilateral scapular motion during five to ten repetitions of unloaded humeral elevation in the scapular plane (scaption) to establish a baseline of scapular movement, 2) observation of bilateral scapular motion during five to ten repetitions of loaded (0.5-5 kg) scaption, and 3) observation of unilateral scapular motion during resisted isometric external rotation with the arm at the side in neutral rotation (i.e. scapular flip sign).[64]

The scapular lateral slide test is a semi-dynamic, quantitative assessment of scapular position. This test has been shown to be reliable in assessing the bilateral position of the scapulae in relation to a fixed point on the spine as varying loads are placed on the supporting scapular musculature.[9] The test involves a series of three measurement positions.

Evaluation of the mobility of the posterior glenohumeral joint capsule, the posterior shoulder musculature, and the anterior coracoid musculature provides critical information regarding the pathomechanic assessment of scapular dysfunction. Posterior glenohumeral joint capsule contracture has been shown to produce excessive superior and anterior humeral head translation, thereby compromising the size of the subacromial space and altering glenohumeral and scapular kinematics.[10, 19, 25] Posterior shoulder tightness is an additional commonly described flexibility characteristic of scapular dysfunction. [1, 3, 8,9, 13, 17, 32] Myers et al. quantify posterior shoulder tightness utilizing supine and side-lying horizontal adduction assessments.[65] One final flexibility measurement to consider during scapular evaluation is pectoralis minor mobility. Due to its proximal attachment on the coracoid process of the scapula, inflexibility of the pectoralis minor muscle may manifest as excessive scapular anterior tilt and internal rotation, thus resulting in coracoid process pain and scapular dysfunction.

Manual muscle testing of the scapular stabilizing muscles is critical in determining the presence of or potential for scapular dysfunction. Strength of the middle and lower trapezius, rhomboids major and minor, and the serratus anterior muscles should be assessed through manual muscle testing techniques. Additional scapular muscle strength and endurance tests include the isometric scapular retraction pinch and wall push up tests. Typically, patients are able to hold an isometric pinch of the scapulae in retraction for 15 to 20 seconds without the onset of burning pain or muscle weakness. An inability to hold this position due to pain or weakness provocation is a positive sign indicating scapular muscle dysfunction.[9] The ability of the serratus anterior muscle to stabilize the scapula on the thorax is easily evaluated with the wall push-up test. The patient performs 5-10 wall push-ups while the clinicians observes for abnormalities in scapular position and motion, specifically scapular winging.[9]

Conclusion

This study is the first to assess the validity of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] In overhead athletes, mean muscle activation amplitude of the scapular stabilizers was not found to be valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] However, the results of this study need to be interpreted with caution, because the majority of subjects scored lower than the commonly accepted threshold score for the clinical diagnosis of SICK scapula syndrome.

APPENDIX A: TABLES

Table 1. Estimated Study Power for Each Dependent Variable (n = 40)

	Standard Deviation (s)	Mean Difference ($X_{control} - X_{experimental}$)	Effect Size Index (d)	Power
Upper Trapezius (% max muscle activity)	80%	60%	.750	.94
Middle Trapezius (% max muscle activity)	12.9%	24%	1.884	.99
Lower Trapezius (% max muscle activity)	80%	50%	.625	.87
Serratus Anterior (% max muscle activity)	80%	55%	.688	.90

(Cools et al.[49], Thigpen et al.[15])

Table 2. Individual Study Research Questions

RQ1: Is mean electromyographic (EMG) amplitude of the **upper trapezius** a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** in overhead athletes?	**IV:** Glenohumeral flexion task Glenohumeral scaption task **Upper trapezius** mean EMG amplitude muscle activation **DV:** Subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]	**Multiple linear regressions** will be used to predict the scores derived from the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]
RQ2: Is mean electromyographic (EMG) amplitude of the **middle trapezius** a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** in overhead athletes?	**IV:** Glenohumeral flexion task Glenohumeral scaption task **Middle trapezius** mean EMG amplitude muscle activation **DV:** Subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]	
RQ3: Is mean electromyographic (EMG) amplitude of the **lower trapezius** a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** in overhead athletes?	**IV:** Glenohumeral flexion task Glenohumeral scaption task **Lower trapezius** mean EMG amplitude muscle activation **DV:** Subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]	
RQ4: Is mean electromyographic (EMG) amplitude of the **serratus anterior** a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] during functional tasks **(glenohumeral elevation in the sagittal plane and glenohumeral elevation in the scapular plane)** in overhead athletes?	**IV:** Glenohumeral flexion task Glenohumeral scaption task **Serratus anterior** mean EMG amplitude muscle activation **DV:** Subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]	

Table 3. Study Participant Demographics

	Male Participants (n = 7)		Female Participants (n = 30)	
	Mean	**±SD**	**Mean**	**±SD**
Age (years)	19.14	1.07	19.97	1.08
Height (cm)	181.43	2.48	173.79	8.42
Mass (kg)	73.87	3.59	69.00	7.90
SICK Score [a]	5.29	2.23	4.32	3.44
Subjective [b]	2.14	1.25	1.66	1.64
Objective [c]	1.43	0.79	1.45	1.62
Malpositioning [d]	1.71	0.64	1.23	0.84

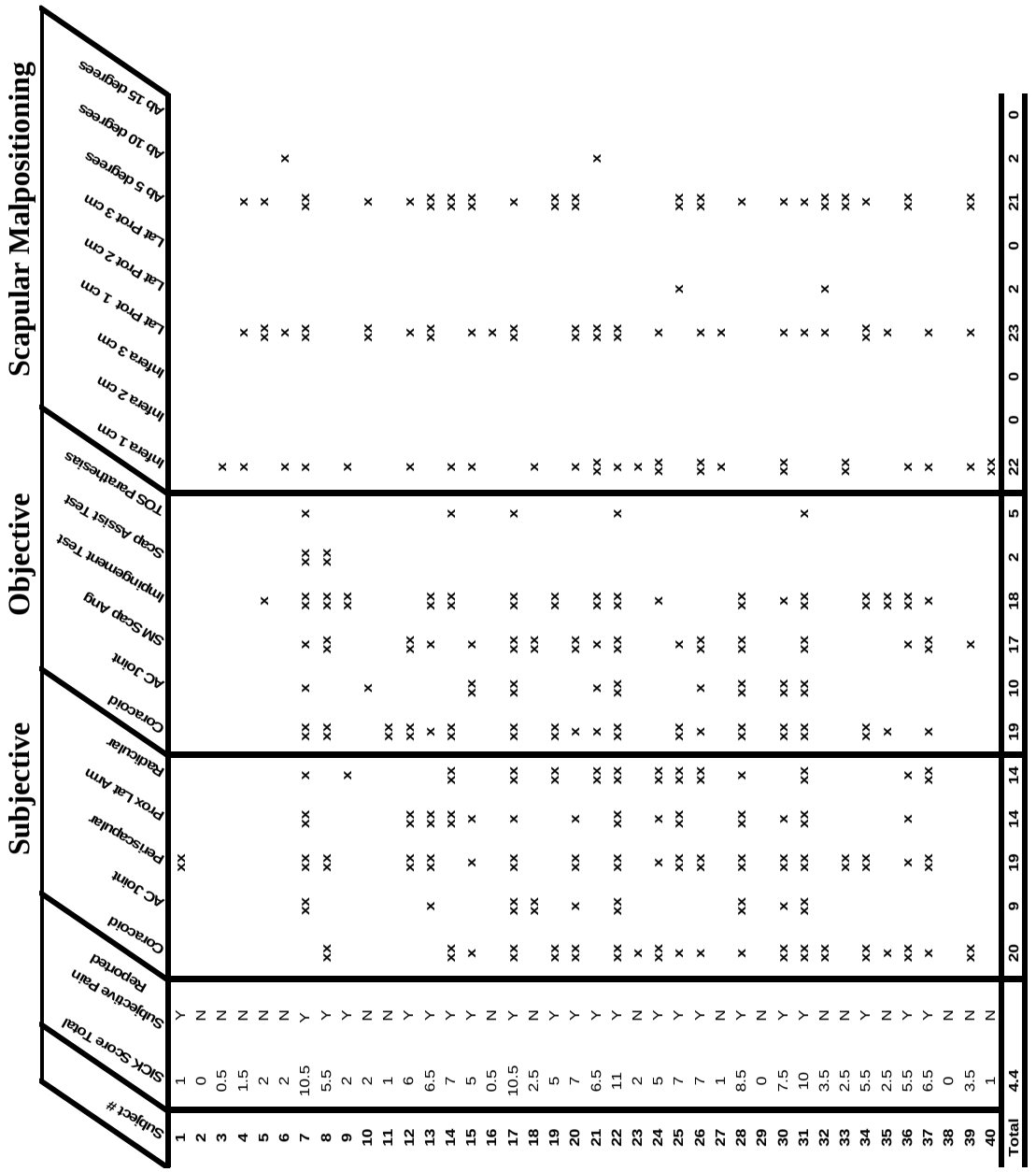
[a] SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]

[b] Self-reported pain of the coracoid process, AC joint, periscapular soft tissue, proximal lateral arm, and/or elbow (possible 5 points)

[c] Self-reported tenderness to palpation of the coracoid process, AC joint, superior medial angle; (+) provocative impingement test (Hawkins-Kennedy Impingement Sign), (+) scapular assistance test, and/or (+) thoracic outlet syndrome test (Allen Test) (possible 6 points)

[d] Scapular malposition based on 1) infera (i.e. the visual appearance of a dropped scapula due to scapular tilting or protraction), 2) lateral displacement, and 3) abduction (possible 9 points

Table 4. Breakdown of SICK Scapula Syndrome Score



Subject #	SICK Score Total	Subjective Painful Reported	Subjective Coracoid	Subjective AC Joint	Subjective Periscapular	Subjective Prox Lat Arm	Subjective Radicular	Objective Coracoid	Objective AC Joint	Objective SM Scap Ang	Objective Impingement Test	Objective Scap Assist Test	Objective TOS Paresthesias	Scapular Malpositioning Infera 1 cm	Infera 2 cm	Infera 3 cm	Lat Prot 1 cm	Lat Prot 2 cm	Lat Prot 3 cm	Ab 5 degrees	Ab 10 degrees	Ab 15 degrees
1	1	Y			xx																	
2	0	N																				
3	0.5	N												x								
4	1.5	N												x			x			x		
5	2	N									x						xx			x		
6	2	N												x			x				x	
7	10.5	Y		xx	xx	xx	x	xx	x	x	xx	xx	x	x			xx			xx		
8	5.5	Y	xx		xx			xx		xx	xx	xx										
9	2	Y					x							x								
10	2	N							x		xx						xx			x		
11	1	N						xx														
12	6	Y			xx	xx		xx		xx				x			x			x		
13	6.5	Y		x	xx	xx		x		x	xx						xx			xx		
14	7	Y	xx			xx	xx	xx			xx		x	x						xx		
15	5	Y	x		x	x			xx	x				x			x			xx		
16	0.5	N												x			x					
17	10.5	Y	xx	xx	xx	x	xx	xx	xx	xx	xx		x				xx			x		
18	2.5	N		xx					xx	xx				x								
19	5	Y	xx				xx	xx			xx									xx		
20	7	Y	xx	x	xx	x		x		xx				x			xx			xx		
21	6.5	Y					xx	x	x	x	xx			xx			xx				x	
22	11	Y	xx	xx	xx	xx	xx	xx	xx	xx	xx		x	x			xx					
23	2	N	x											x								
24	5	Y	xx		x	x	xx				x			xx			x			xx		
25	7	Y	x		xx	xx	xx	xx		x								x		xx		
26	7	Y	x		xx		xx	x	x	xx				xx			x			xx		
27	1	N												x			x					
28	8.5	Y	x	xx	xx	xx	x	xx	xx	xx	xx									x		
29	0	N																				
30	7.5	Y	xx	x	xx	x		xx	xx		x			xx			x			x		
31	10	Y	xx	xx	xx	xx	xx	xx	xx	xx	xx		x				x			x		
32	3.5	N	xx														x	x		xx		
33	2.5	N			xx									xx						xx		
34	5.5	Y	xx		xx			xx			xx						xx			x		
35	2.5	N	x					x			xx						x					
36	5.5	Y	xx		x	x	x			x	xx			x						xx		
37	6.5	Y	x		xx		xx	x		xx	x			x			x					
38	0	N																				
39	3.5	N	xx							x				x			x			xx		
40	1	N												xx								
Total	4.4		20	9	19	14	14	19	10	17	18	2	5	22	0	0	23	2	0	21	2	0

x = identified by one investigator
xx = identified by two investigators
Prox lat arm = proximal lateral arm
SM scap ang = superior medial scapular angle

Scap assist test = scapular assistance test
Lat prot = lateral protraction
Ab = abduction

Table 5: Mean and Standard Deviation Muscle Amplitude (%MVIC)

	Flexion Elevation Task			
	Ascending Phase		Descending Phase	
	Mean	±SD	Mean	±SD
Upper Trapezius	54.01	28.53	28.91	16.75
Middle Trapezius	20.61	22.07	16.02	19.58
Lower Trapezius	26.91	24.15	20.18	24.11
Serratus Anterior	53.57	40.26	26.33	19.86
	Scaption Elevation Task			
	Ascending Phase		Descending Phase	
	Mean	±SD	Mean	±SD
Upper Trapezius	62.70	30.25	33.92	22.35
Middle Trapezius	31.24	35.39	24.53	33.03
Lower Trapezius	25.05	22.99	20.51	23.65
Serratus Anterior	51.06	38.12	29.87	25.03

APPENDIX B: FIGURES

Figure 1. SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale. Dr. Craig D. Morgan, MD.[1]

DATE _____ SPORT _____
NAME _____ POSITION _____
AGE _____ PRESENTING SX's _____

SUBJECTIVE	PAIN	YES	NO	SCORE
	Coracoid	1	0	
	AC Joint	1	0	
	Periscapular	1	0	
	Prox. Lat. Arm	1	0	
	Radicular	1	0	
OBJECTIVE				
	Coracoid	1	0	
	AC Joint	1	0	
	Sup. Med. Scap. Angle	1	0	
	Impingement Test	1	0	
	Scapular Asst. Test	1	0	
	Tos Paresthesias	1	0	

SCAP. MALPOSITION	0cm	1cm	2cm	3cm	SCORE
Infera	0	1	2	3	
Lateral Protraction	0	1	1	3	
Abduction	0°	5°	10°	15°	
	0	1	2	3	
			TOTAL SCORE		

Figure 2. Effect Size Index Calculations (Cools et al.[49], Thigpen et al.[15])

Upper Trapezius

$$\mathbf{d = (X_{control} - X_{experimental})/(s)}$$

$$\mathbf{d = (60)/(80)}$$

$$\mathbf{d = 0.75}$$

Middle Trapezius

$$\mathbf{d = (X_{control} - X_{experimental})/(s)}$$

$$\mathbf{d = (58)/(80)}$$

$$\mathbf{d = 0.73}$$

Lower Trapezius

$$\mathbf{d = (X_{control} - X_{experimental})/(s)}$$

$$\mathbf{d = (50)/(80)}$$

$$\mathbf{d = 0.625}$$

Serratus Anterior

$$\mathbf{d = (X_{control} - X_{experimental})/(s)}$$

$$\mathbf{d = (55)/(80)}$$

$$\mathbf{d = 0.688}$$

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d, effect size index
s, standard deviation
X, group mean

Figure 3. Upper Trapezius Muscle Activity during Sagittal Plane Elevation (Ascending Phase)

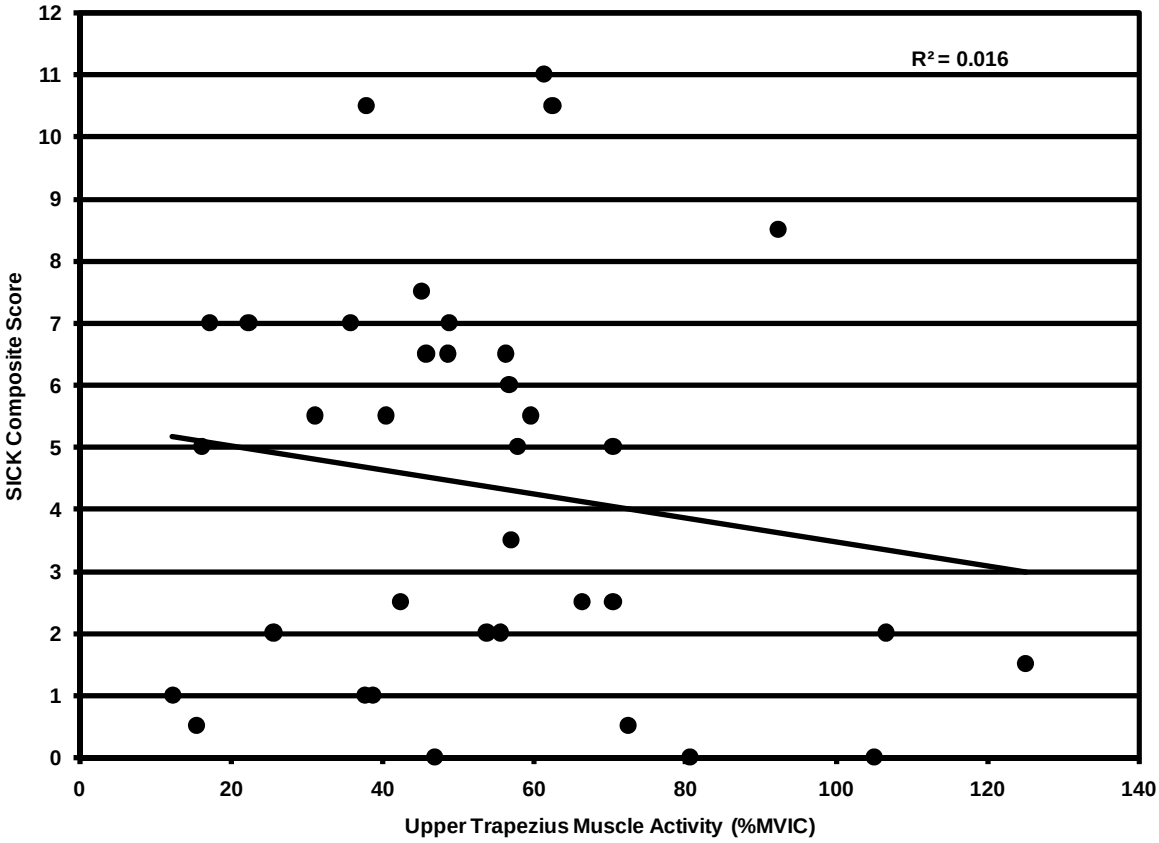


Figure 4. Upper Trapezius Muscle Activity during Sagittal Plane Elevation (Descending Phase)

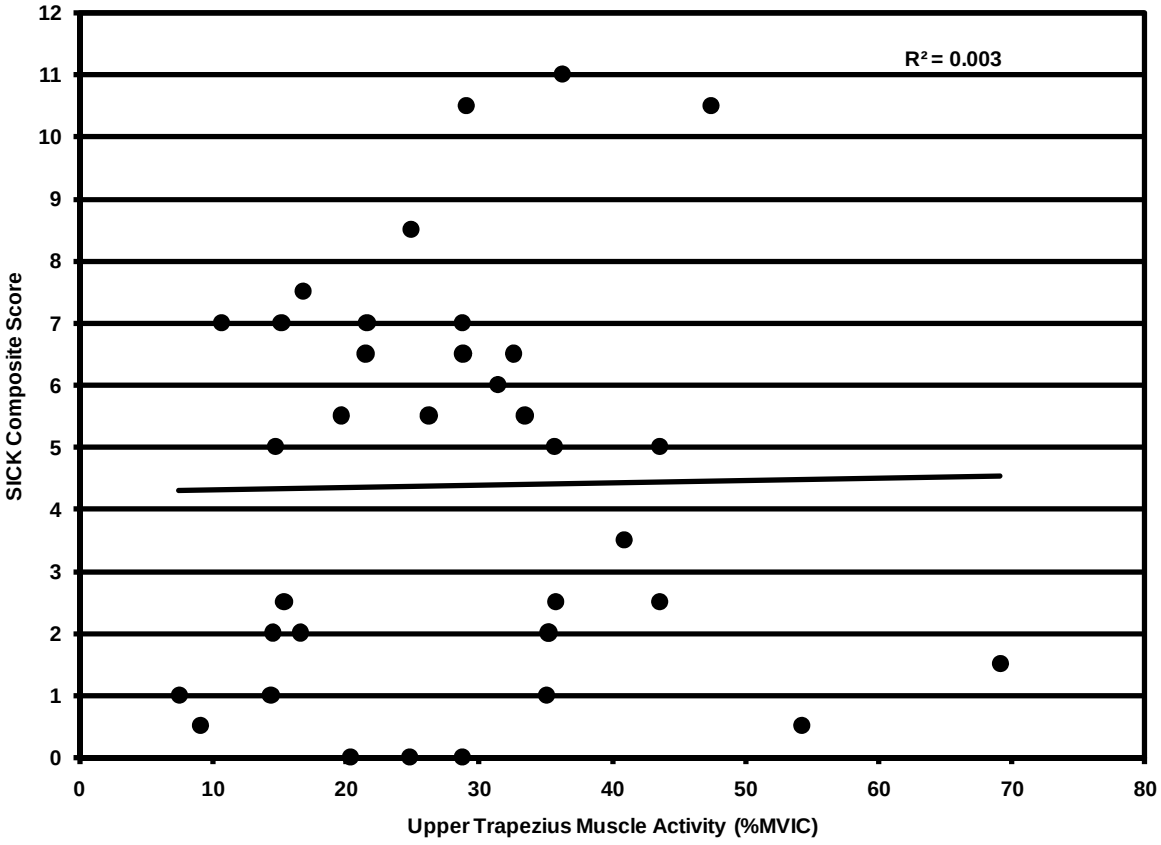


Figure 5. Upper Trapezius Muscle Activity during Scapular Plane Elevation (Ascending Phase)

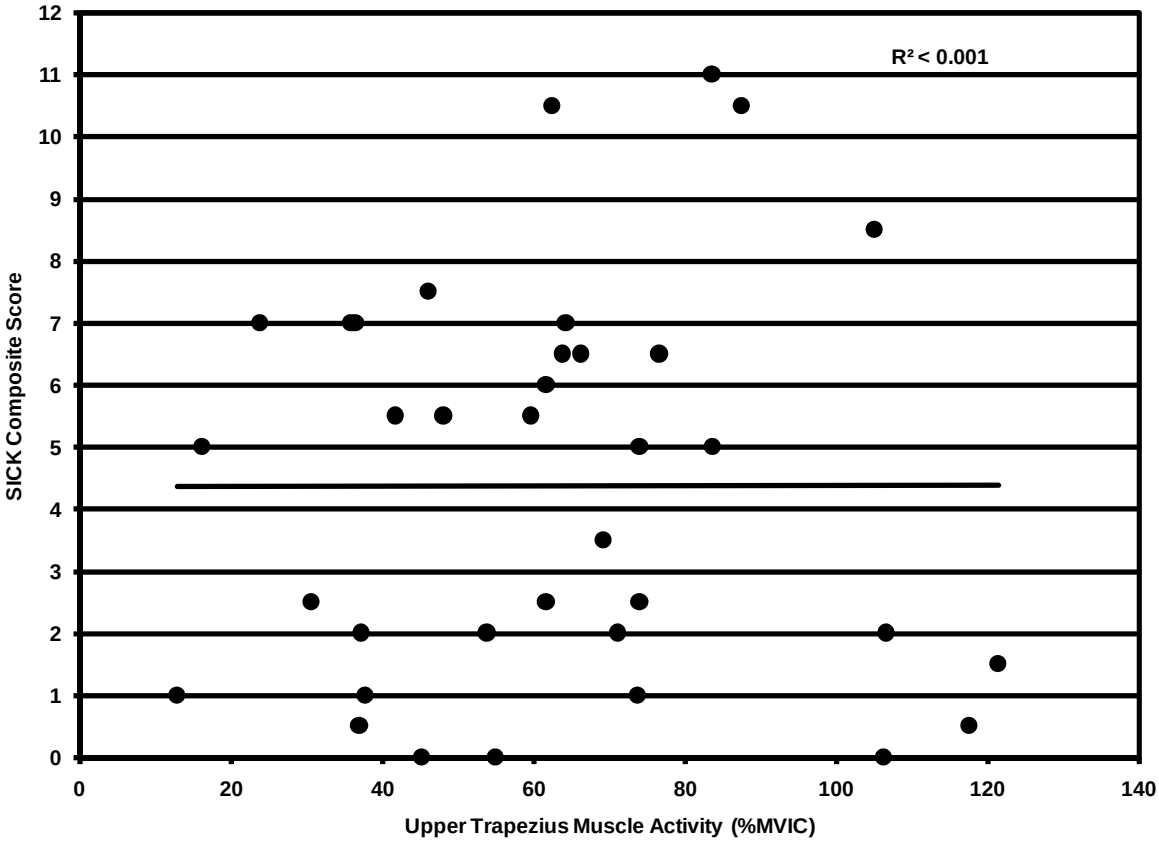


Figure 6. Upper Trapezius Muscle Activity during Scapular Plane Elevation (Descending Phase)

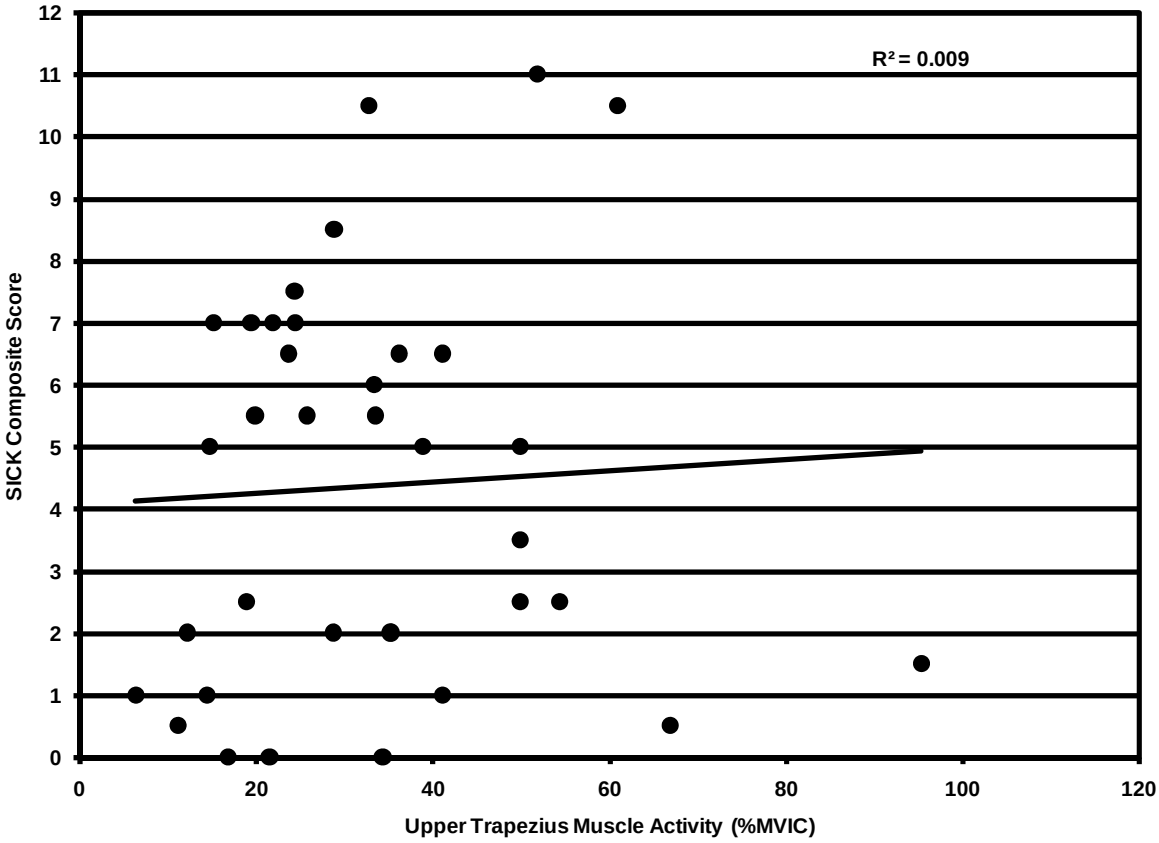


Figure 7. Middle Trapezius Muscle Activity during Sagittal Plane Elevation (Ascending Phase)

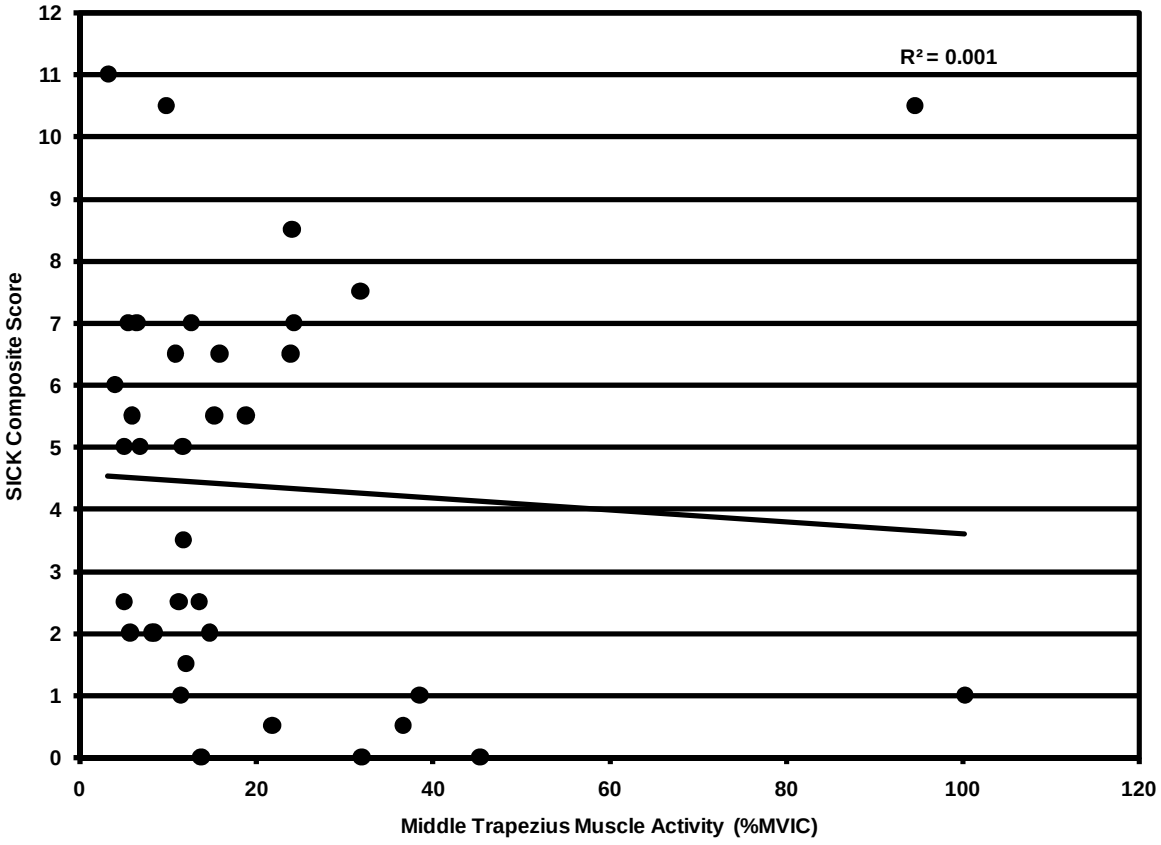


Figure 8. Middle Trapezius Muscle Activity during Sagittal Plane Elevation (Descending Phase)

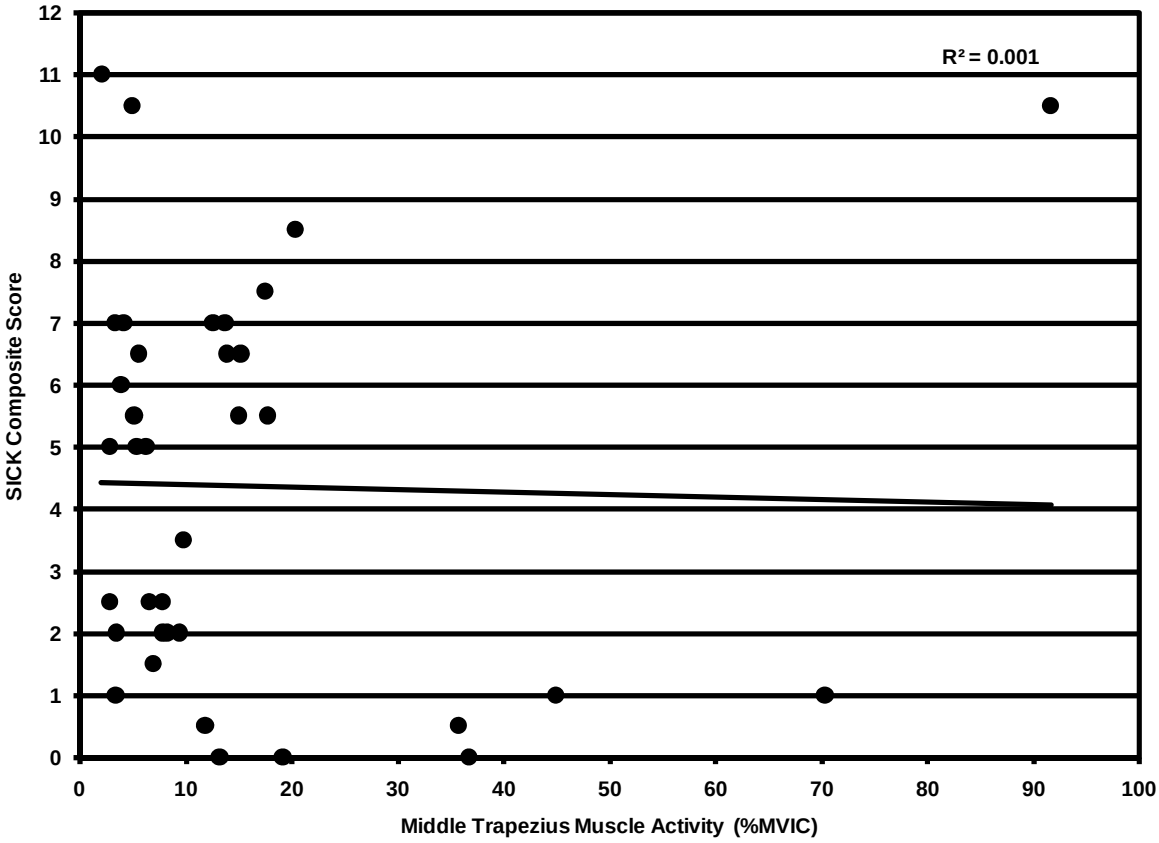


Figure 9. Middle Trapezius Muscle Activity during Scapular Plane Elevation (Ascending Phase)

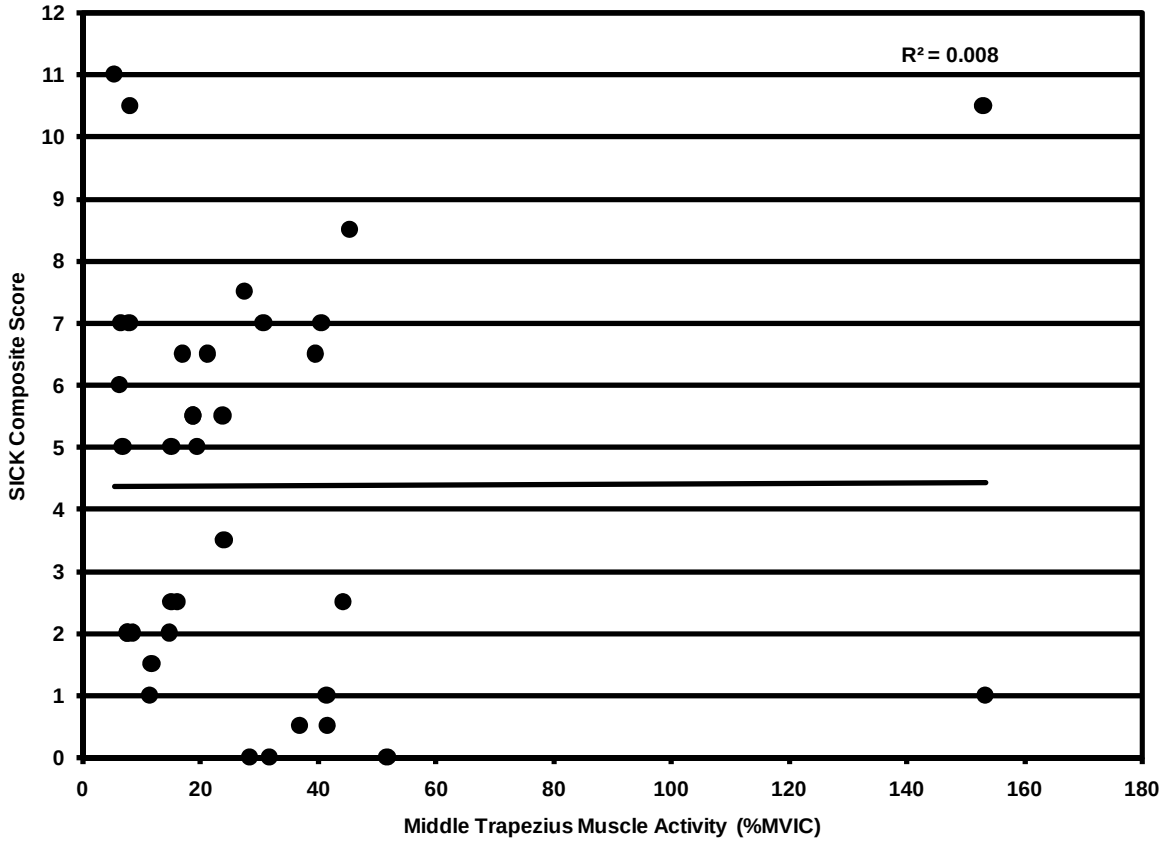


Figure 10. Middle Trapezius Muscle Activity during Scapular Plane Elevation (Descending Phase)

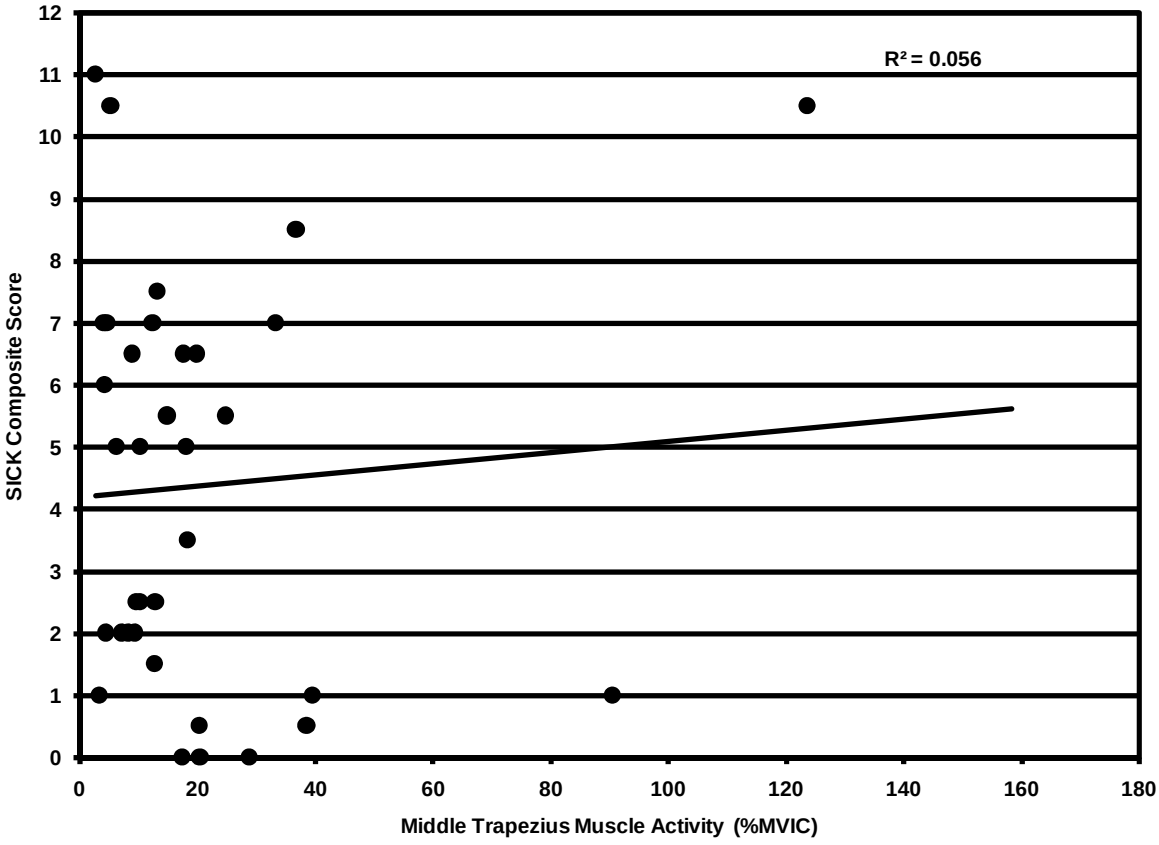


Figure 11. Lower Trapezius Muscle Activity during Sagittal Plane Elevation (Ascending Phase)

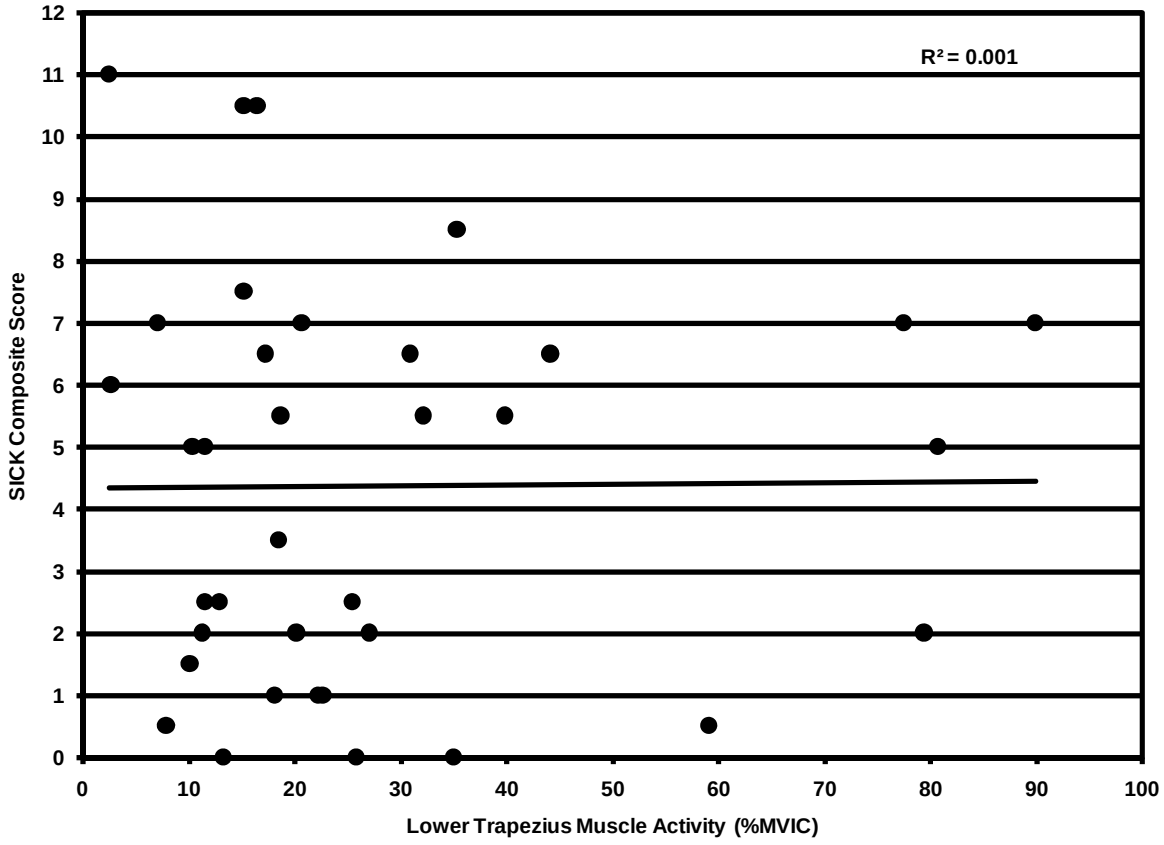


Figure 12. Lower Trapezius Muscle Activity during Sagittal Plane Elevation (Descending Phase)

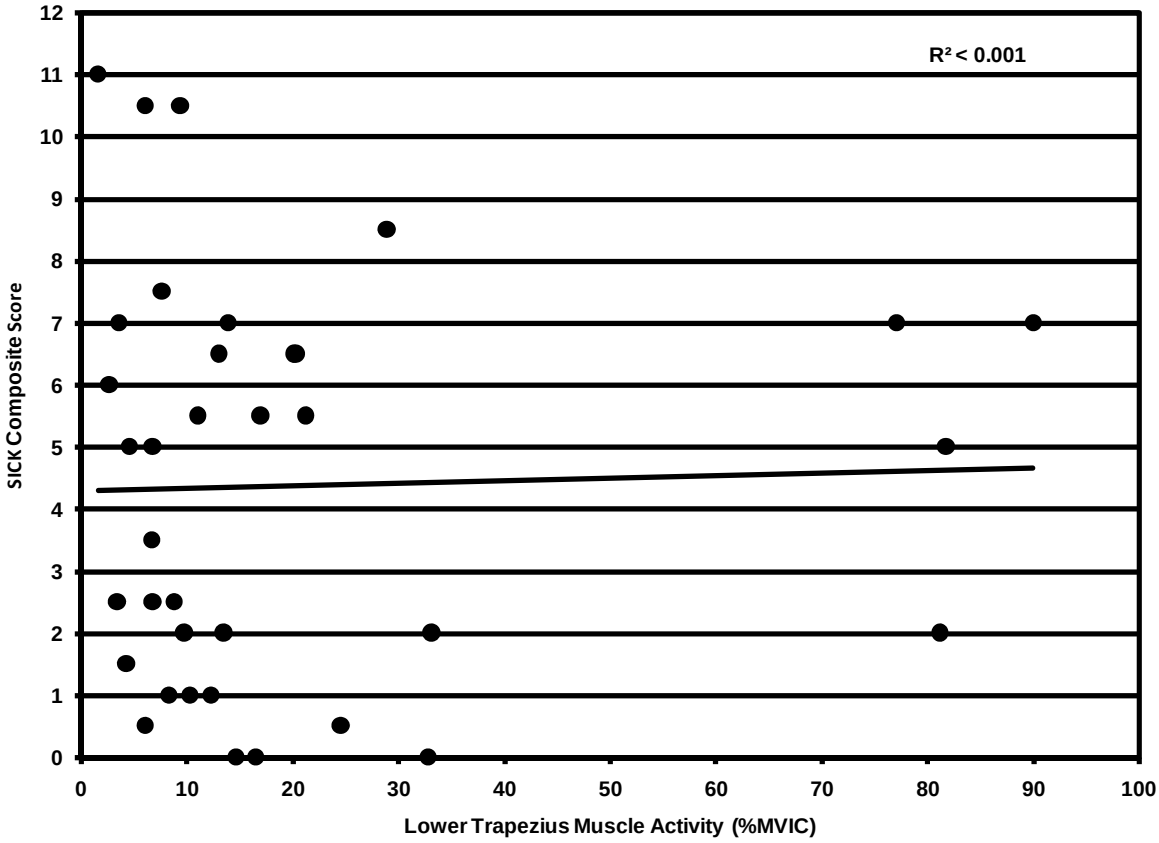


Figure 13. Lower Trapezius Muscle Activity during Scapular Plane Elevation (Ascending Phase)

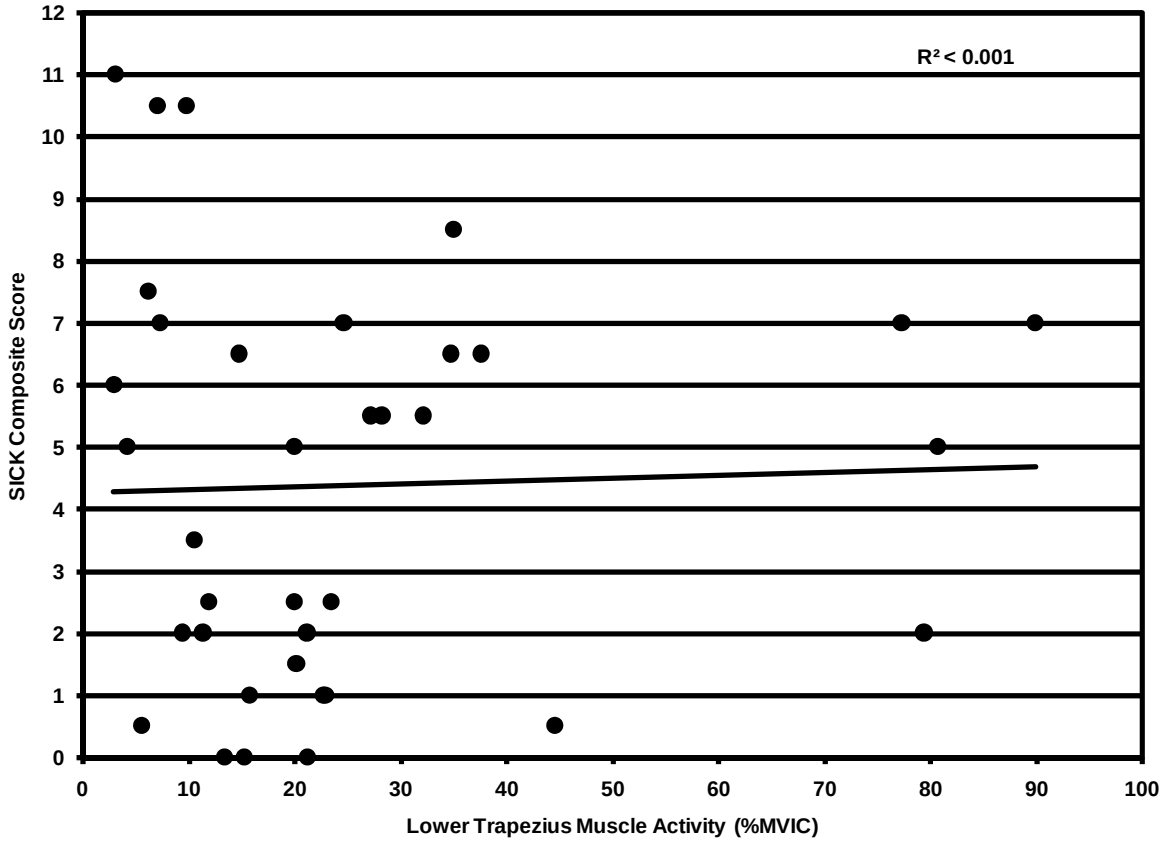


Figure 14. Lower Trapezius Muscle Activity during Scapular Plane Elevation (Descending Phase)

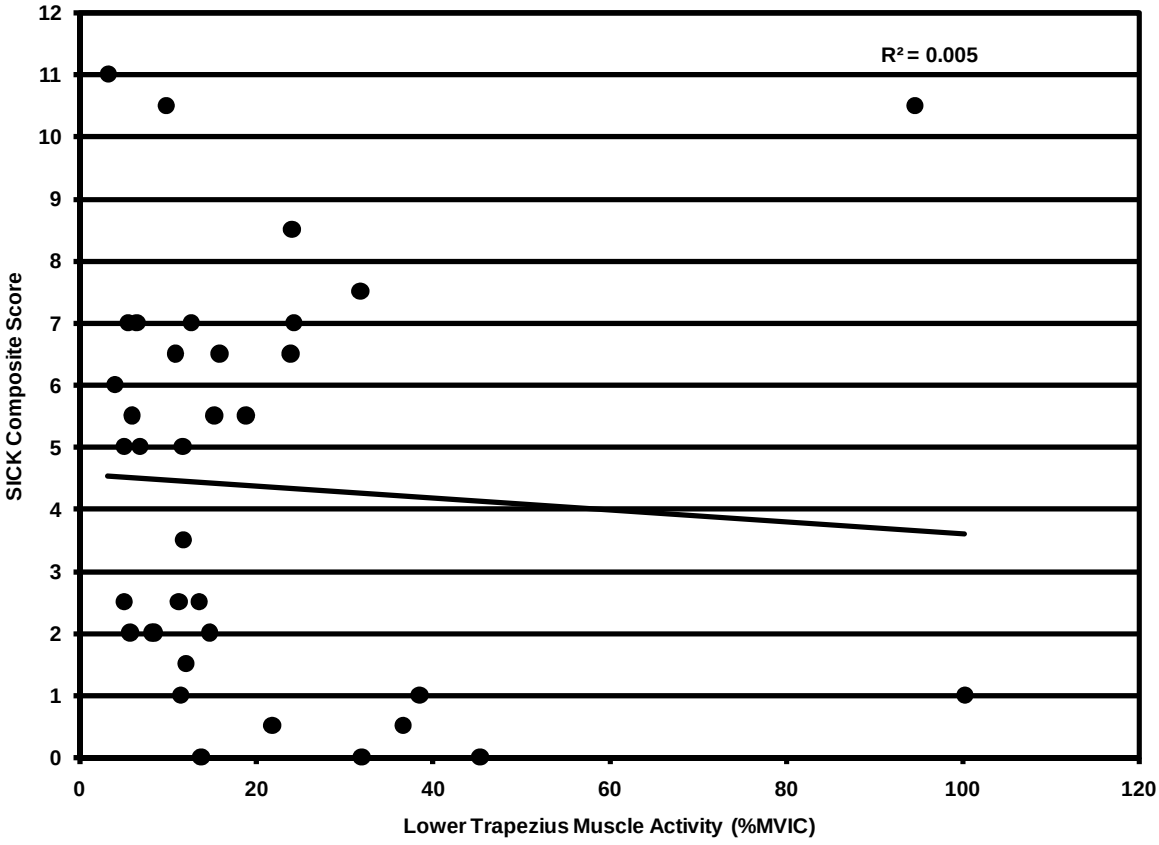


Figure 15. Serratus Anterior Muscle Activity during Sagittal Plane Elevation (Ascending Phase)

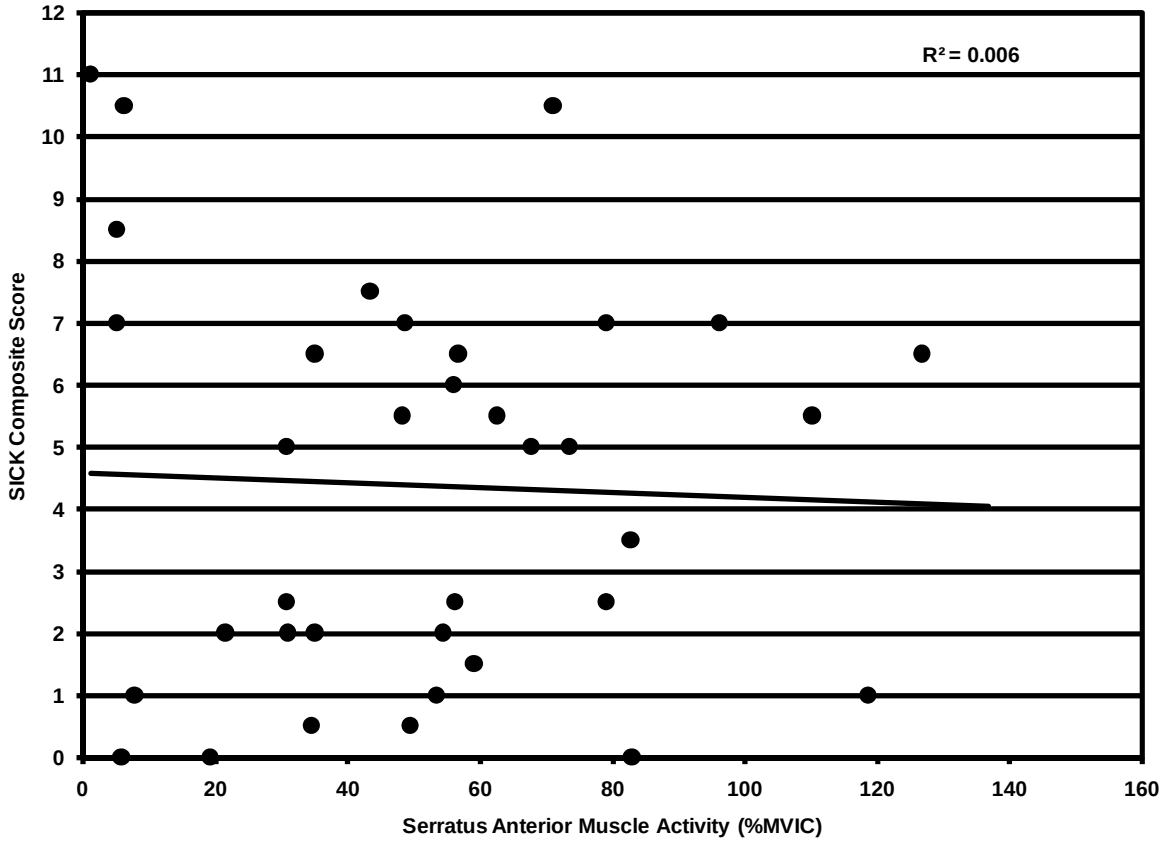


Figure 16. Serratus Anterior Muscle Activity during Sagittal Plane Elevation (Descending Phase)

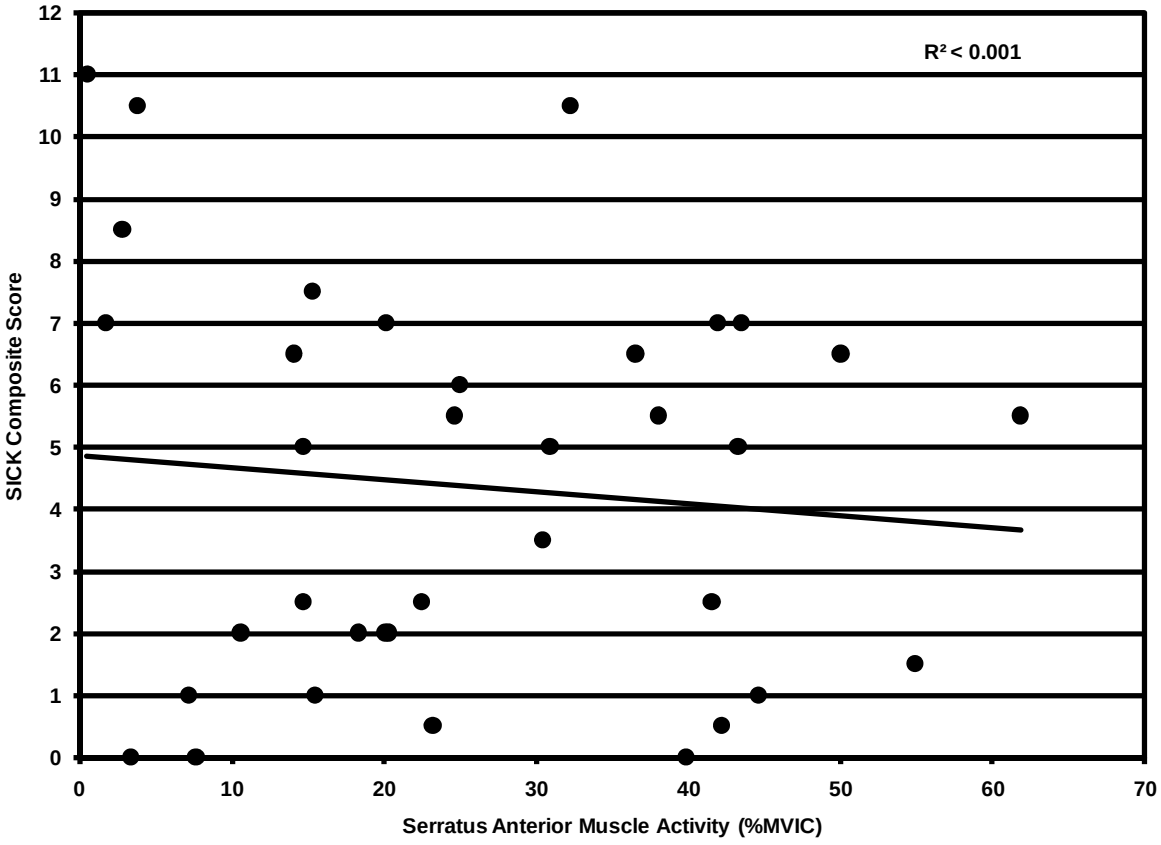


Figure 17. Serratus Anterior Muscle Activity during Scapular Plane Elevation (Ascending Phase)

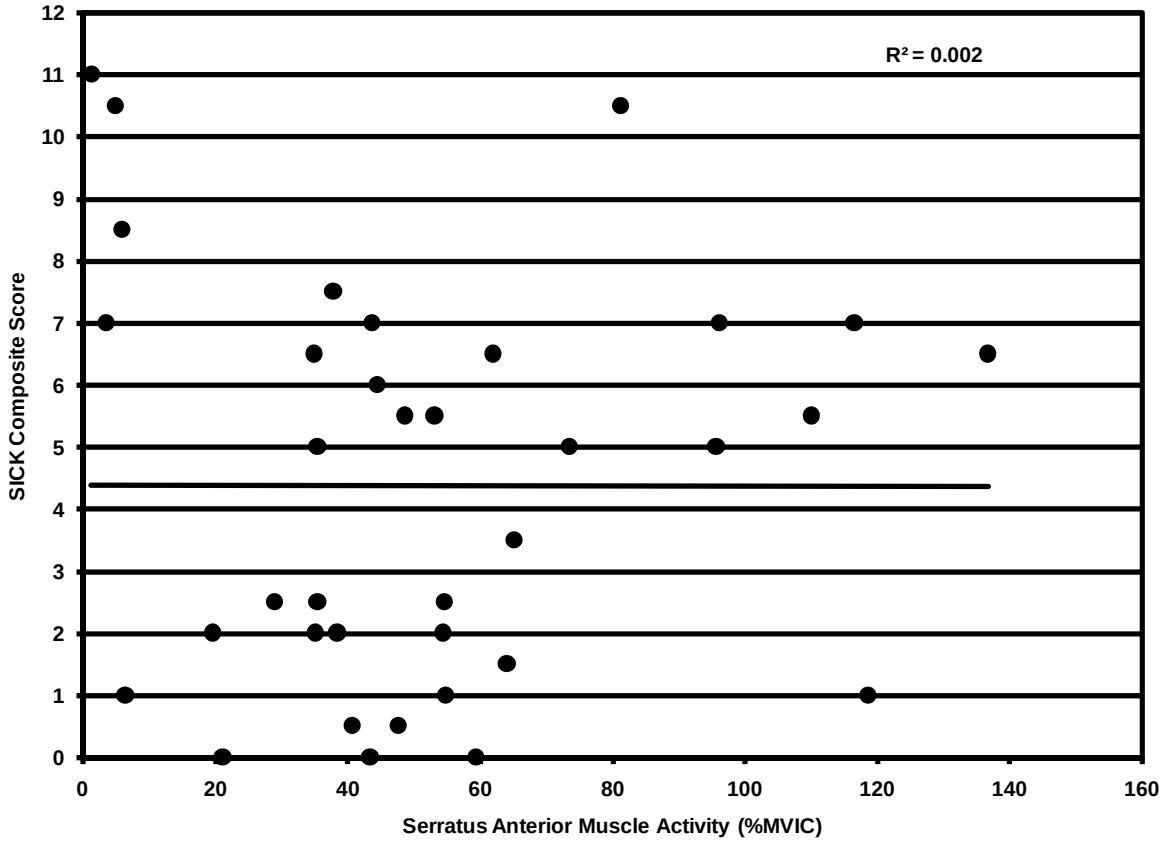
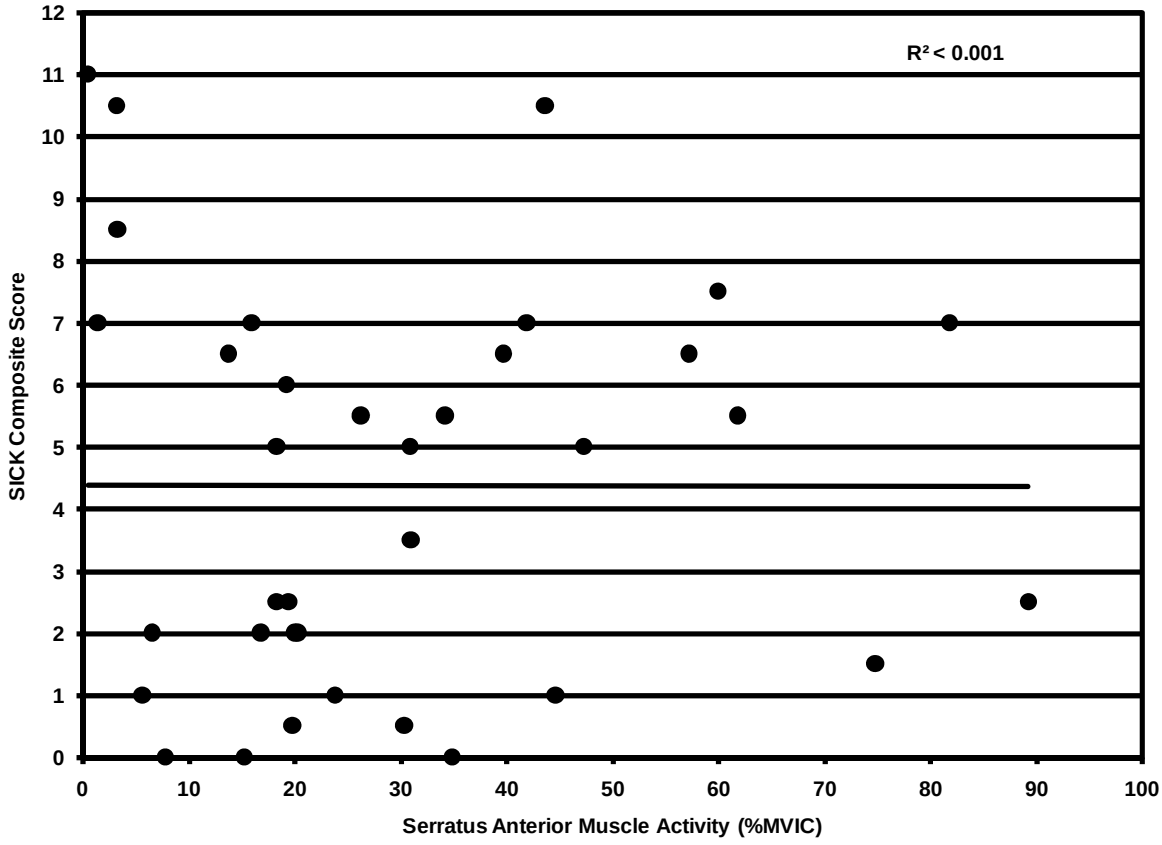


Figure 18. Serratus Anterior Muscle Activity during Scapular Plane Elevation (Descending Phase)



APPENDIX C: MANUSCRIPT

ABSTRACT

A Prediction of SICK Scapula Syndrome Score from Muscle Activation and Kinematic Analysis in Overhead Athletes

Context: Scapular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement (SICK) are associated with shoulder injury. In overhead athletes, one initial sign of SICK scapula syndrome is shoulder biomechanical dysfunction and pain, yet it is not fully understood whether altered muscle activation of the scapular stabilizers is a sign of SICK scapula syndrome or a precursor to its development. Currently, there is a need for validated, quantitative measures to improve the reliability SICK scapula syndrome assessment. **Objective:** To determine if subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] can be predicted from scapular stabilizer muscle activation. **Design:** Quasi-experimental, one group design with a counterbalancing of two functional tasks. **Setting:** Research laboratory. **Patients or Other Participants:** NCAA Division I and/or recreational club overhead athletes (n = 40). **Data Collection and Analysis:** The SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale was used for assessing the severity of scapular malposition. Muscle activation was recorded for the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles. Using mean EMG amplitude data to represent muscle activation over each phase of humeral elevation, multiple linear regressions were used to predict subject score derived from the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] **Results:** Regression analyses revealed that scapular stabilizer muscle activation amplitude did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] **Conclusion:** In overhead athletes, mean muscle activation amplitude of

the scapular stabilizers was not found to be valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] **Key Words:** shoulder, scapula, muscle activation, dyskinesis, validity.

INTRODUCTION

Shoulder pain, regardless of its nature, source, or severity, currently plagues today's overhead athlete.[2-4] The demands of sport require overhead athletes to repetitively endure high amounts of load to the glenohumeral and scapulothoracic joints. These demands are great, and in turn, so is the risk of shoulder soft tissue injury due to overuse. Warner et al[5] have shown that the vast majority of patients suffering from an overuse shoulder pathology present with scapulothoracic asymmetries during an elevation task. When evaluating a shoulder soft tissue injury, medical professionals face the challenge of determining whether scapular malposition and dyskinesis were the result of a shoulder joint pathology or the source of its development. Current literature suggests that scapular asymmetries may be an objective means of understanding the development of the shoulder pathology itself.[1, 3, 5-16] Scapular malalignment is perhaps one of the most evident signs of shoulder dysfunction that may lead to the initiation of the pathological sequence of events thereafter.[1, 3, 4, 8-10, 13-15, 17, 18]

Research has repeatedly shown that scapular dyskinesis and postural abnormalities in overhead athletes may be an important risk factor for the development of shoulder pain .[1, 3, 4, 8-10, 13-15, 17, 18] As a result, shoulder soft tissue pathology and scapular dyskinesis appear to be intimately related to one another. Shoulder soft tissue overload and the subsequent injury that results from shoulder pain, specifically subacromial impingement syndrome, have been linked to decreased scapular upward rotation, decreased scapular posterior tilt, and increased scapular internal rotation with humeral elevation tasks.[10] Burkhart et al[1] use the acronym SICK when describing a specific form of scapular dyskinesis. SICK scapula refers to the presence of **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement. Overhead athletes diagnosed with SICK

scapula syndrome typically present with a unilateral lowered and anteriorly tilted scapula with accompanied anterior shoulder pain on the involved side. In overhead athletes, one initial sign of SICK scapula syndrome is shoulder biomechanical dysfunction and pain. Because athletic skill is dependent on biomechanical ease and efficiency, shoulder pain and altered overhead biomechanics can become extremely debilitating for the competitive overhead athlete.[1, 6, 7, 9, 13, 19, 25] With the ongoing establishment of SICK scapula syndrome, scapular dyskinesis is becoming more objectively assessed for both therapeutic rehabilitation purposes and prophylactic conditioning purposes.

Whether via special tests (i.e. the Scapular Assistance Test) or the palpation of anatomical landmarks (i.e. the coracoid process of the scapula), one approach to the clinical diagnosis of an underlying shoulder pathology has been the use of patient self-reported pain symptoms. Pain is the common thread interweaving most shoulder disorders, with the source and mechanism of such pain being variable. Because pain is a person's perception of physical damage, subjective athlete pain reports have the advantage of being athlete-specific and providing an important perspective on athlete status. They cannot, however, accurately represent the presence or the severity of an actual physical impairment with associated soft tissue damage. Currently, there is a need for a validated, reliable, quantitative measure of scapular dyskinesis for shoulder pathology assessment.

As is the case with the clinical assessment of SICK scapula syndrome, qualitative pain measures provide an incomplete picture of the severity of this scapular malpositioning and dyskinetic disorder. Based upon both the literature and screening guidelines of Burkhart & Morgan[1], the severity of the SICK scapula syndrome is graded on a 0 to 20 point scale, with 0 representing complete shoulder health and 20 representing severe, symptomatic SICK

scapula syndrome. It must be noted, however, that both the validity and the reliability of this scale have not yet been established. Typically upon screening, athletes who present with symptomatic SICK scapula syndrome will score somewhere within the range of 10 to 14 on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], but at this point in time, the scale's diagnostic value is merely anecdotal. This suggests the need for a valid, structured clinical tool that could be utilized by clinicians as a predictor for the potential, presence, and/or severity of SICK scapula syndrome.

The purpose of this study was to validate the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale, developed by Burkhart et al[1], by predicting subject score from scapular stabilizer EMG activity. Determining the validity of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] may allow clinicians to more readily utilize this scale as an accurate screening tool toward the identification of SICK scapula syndrome.

The identification of scapular muscle dysfunction in pathologic, SICK scapula syndrome overhead athletes will allow the condition to become more successfully treated. Understanding the adaptive scapular stabilizer activation deficiencies associated with SICK scapula syndrome would allow clinicians to implement specific rehabilitation exercises when treating affected athletes. The rehabilitation exercises that target the muscle identified to be dysfunctional in this study could help restore ideal scapular force couple synchronization, thus allowing the scapula to move harmoniously with the moving humerus. Such fine-tuned, corrective exercise would effectively allow a rehabilitating athlete to have a pain-free return to competition.

METHODS

Subjects

Subjects were recruited from a university population of NCAA Division I overhead athletes and/or recreational club overhead athletes at The University of North Carolina at Chapel Hill. Subjects were both male and female and were between the ages of 18 and 25 years old.

Overhead athletes were operationally defined as those who participate in a sport that requires their arm to be above their shoulder height on a repetitive basis during throwing or striking activities (i.e. baseball, softball, swimming, tennis, volleyball, and water polo) for a duration of at least 30 minutes per session for at least 3 individual sessions per week.

Instrumentation

The SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale is a grading system used for assessing the severity of scapular malposition based on 1) infera (i.e. the visual appearance of a dropped scapula due to scapular tilting or protraction), 2) lateral displacement, and 3) abduction. All measurements were made statically with the patients standing erect with arms relaxed at their side. The measurement of infera is the difference in vertical height of the superomedial scapular angle of the dropped scapula in centimeters compared with the contralateral superomedial angle.[1] The SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale was used to give a quantitative sense of SICK scapula syndrome severity.

Prior to the study, reliability and precision of this scale were established from a small pilot study by the chief investigators using intraclass correlation coefficient (ICC) and

standard error of measurement (SEM). The inter-session reliability and precision were calculated to yield an ICC of 0.682 and SEM of 1.44 points, respectively. The inter-tester reliability and precision were calculated to yield an ICC of 0.684 and SEM of 1.18 points, respectively.

A Motion Star (Ascension Technologies Inc, Burlington, Vt) electromagnetic motion analysis mini bird system controlled by the Motion Monitor (Innovative Sports Training Inc, Chicago, Ill) software was used to assess shoulder complex kinematics at a sampling rate of 100 Hz. The humeral kinematic data were used to define the ascending and descending phases during each humeral elevation task.

Electromyographic (EMG) muscle activation analyses were performed to measure the mean amplitude of the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles using a Delsys Bagnoli-8 EMG (Boston, MA). The EMG signal was amplified by a factor of 1000 over a bandwidth of 0.01 to 2000 Hz, passed via an A/D converter (National Instruments, Austin, TX) sampling at 1000 Hz then corrected for DC bias.

Procedures

Upon entering the lab, each subject was briefed on testing procedures and signed and received a copy of the Consent to Act as a Human Subject form, that was approved by the University Institution Review Board. Subjects then completed a medical history form, underwent a screening by two trained clinicians using the SICK scapula, Static Measurements, 0 to 20 Point Rating Scale, and were measured for both height and weight.

Limb selection was dictated either by 1) the subject's involved side in those who had reported symptoms of shoulder pain or 2) the dominant side in those who had reported to be asymptomatic for shoulder pain. Subjects were screened twice (one screening per trained clinician/investigator), and an average of the two scores was calculated and utilized for later statistical analysis.

Isometric manual muscle tests were performed to ensure accurate placement of electrodes and to measure and record maximal voluntary isometric contraction (MVIC) EMG. Subjects performed each MVIC measure for five seconds. The MVIC measures were taken for three trials averaged for normalization of muscle activity during each task. The peak mean force for a five-second period was recorded. Mean amplitude values for the three trials were expressed as a percentage (%MVIC) for each functional task.

After the setup for both EMG and kinematic analysis was completed, subjects completed the following tasks in a counterbalanced order: glenohumeral elevation in the sagittal plane (flexion) and glenohumeral elevation in the scapular plane (scaption). Each of the two humeral elevation tasks required the subject to lift their arm for ten repetitions per task. The sagittal plane was defined as the plane parallel to the sagittal plane of the thorax. The scapular plane was defined as the plane 30 degrees anterior to the frontal plane of the thorax.

Subjects completed their full range of motion at a controlled movement velocity by moving in time with a digital metronome set at one beat per second. Each functional task required ten continuous repetitions, with each repetition lasting approximately four seconds (two-second ascending phase, two-second descending phase). A PVC guide pole was used as a guide for both flexion and scaption.

Data Reduction

All raw trial and MVIC data EMG data were filtered using a fourth order Butterworth band-pass filter (10-350 Hz). The root mean square (RMS) of the EMG signal over a 50 ms time constant was taken to further smooth the data.

Mean EMG amplitude was calculated, with the ascending phase (0^{o}-120^{o}) and the descending phase (120-0^{o}) being determined by the humeral elevation angle data. For each repetition, the lowest point of humeral elevation represented the initiation of the ascending phase, and the highest point of humeral elevation represented the cessation of the ascending phase and the initiation of the descending phase. The subsequent lowest point of humeral elevation represented the cessation of the descending phase.

The EMG activity during both the ascending and descending phases were calculated as an average of the mean EMG amplitude over each phase of motion for the middle five repetitions of each muscle tested. The mean EMG activity independent variables were normalized to the MVICs obtained prior to the trials, and were expressed as a percentage of the MVIC (%MVIC). Variables were calculated and processed using Matlab R2007a (The MathWorks Inc., Natick, Massachusetts).

Statistical Analyses

Mean EMG amplitude data were used to represent muscle activation over each phase of humeral elevation for the upper trapezius, middle trapezius, lower trapezius, and serratus anterior.

Multiple linear regressions were used to predict the scores derived from the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] using the Statistical Package for

Social Sciences (SPSS 15.0, Inc, Chicago, IL)(see Table 2). Statistical significance for all comparisons was set a priori at alpha-level of 0.05.

RESULTS

Descriptive Statistics

Forty Division I overhead athletes and/or recreational club overhead athletes (10 softball players, 20 swimmers, 9 volleyball players, and 1 water polo player; 33 right arm dominant, 7 left arm dominant) participated in this study. Due to errors in data, three subjects were dropped from this study. Out of the remaining 37 participants, 20 reported to be currently experiencing shoulder pain. The descriptive statistics on demographics and the SICK Score is presented in Table 3. The breakdown of SICK scapula syndrome score for all subjects is presented in Table 4. Mean amplitude EMG was calculated for the upper trapezius, middle trapezius, lower trapezius, and serratus anterior muscles (see Table 5).

Table 3. Study Participants Demographics

	Male Participants (n = 7)		Female Participants (n = 30)	
	Mean	**±SD**	**Mean**	**±SD**
Age (years)	19.14	1.07	19.97	1.08
Height (cm)	181.43	2.48	173.79	8.42
Mass (kg)	73.87	3.59	69.00	7.90
SICK Score [a]	5.29	2.23	4.32	3.44
Subjective [b]	2.14	1.25	1.66	1.64
Objective [c]	1.43	0.79	1.45	1.62
Malpositioning [d]	1.71	0.64	1.23	0.84

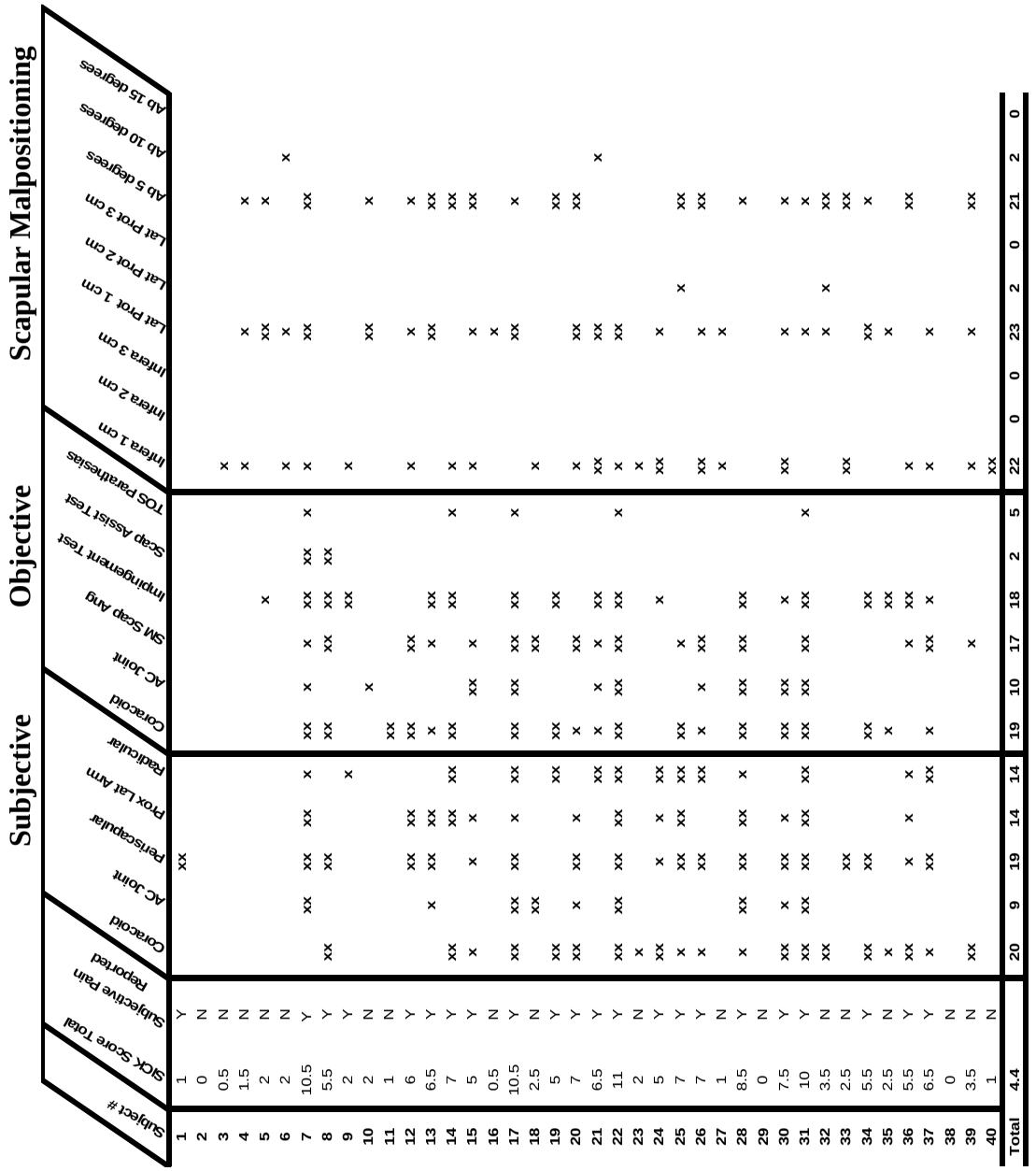
[a] SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]

[b] Self-reported pain of the coracoid process, AC joint, periscapular soft tissue, proximal lateral arm, and/or elbow (possible 5 points)

[c] Self-reported tenderness to palpation of the coracoid process, AC joint, superior medial angle; (+) provocative impingement test (Hawkins-Kennedy Impingement Sign), (+) scapular assistance test, and/or (+) thoracic outlet syndrome test (Allen Test) (possible 6 points)

[d] Scapular malposition based on 1) infera (i.e. the visual appearance of a dropped scapula due to scapular tilting or protraction), 2) lateral displacement, and 3) abduction (possible 9 points)

Table 4. Breakdown of SICK Scapula Syndrome Score



Subject #	SICK Score Total	Subjective Pain Reported	Coracoid	AC Joint	Periscapular	Prox Lat Arm	Radicular	Coracoid	AC Joint	SM Scap Ang	Impingement Test	Scap Assist Test	TOS Paratheisis	Objective Total	Scapular Malpositioning Intera 1 cm	Intera 2 cm	Intera 3 cm	Lat Prot 1 cm	Lat Prot 2 cm	Lat Prot 3 cm	Ab 5 degrees	Ab 10 degrees	Ab 15 degrees
1	1	Y			xx																		
2	0	N																					
3	0.5	N													x								
4	1.5	N													x			x			x		
5	2	N									x							xx			x		
6	2	N													x			x				x	
7	10.5	Y		xx	xx	xx	x	xx	x	x	xx	xx	x		x			xx			xx		
8	5.5	Y	xx		xx			xx		xx	xx	xx											
9	2	Y					x								x								
10	2	N							x		xx							xx			x		
11	1	N						xx															
12	6	Y			xx	xx		xx		xx					x			x			x		
13	6.5	Y		x	xx	xx		x		x	xx							xx			xx		
14	7	Y	xx		xx	xx		xx			xx		x		x						xx		
15	5	Y	x		x	x			xx	x			x		x			x			xx		
16	0.5	N																x					
17	10.5	Y	xx	xx	xx	x	xx	xx	xx	xx	xx		x					xx			x		
18	2.5	N		xx						xx					x								
19	5	Y	xx				xx	xx			xx										xx		
20	7	Y	xx	x	xx	x		x		xx					x			xx			xx		
21	6.5	Y					xx	x	x	x	xx				xx			xx				x	
22	11	Y	xx	xx	xx	xx	xx	xx	xx	xx	xx		x		x			xx					
23	2	N	x												x								
24	5	Y	xx		x	x	xx				x				xx			x			xx		
25	7	Y	x		xx	xx	xx	xx		x									x		xx		
26	7	Y	x		xx		xx	x	x	xx					xx			x			xx		
27	1	N													x			x					
28	8.5	Y	x	xx	xx	xx	x	xx	xx	xx	xx										x		
29	0	N																					
30	7.5	Y	xx	x	xx	x		xx	xx		x				xx			x			x		
31	10	Y	xx	xx	xx	xx	xx	xx	xx	xx	xx		x					x			x		
32	3.5	N	xx															x	x		xx		
33	2.5	N			xx										xx								
34	5.5	Y	xx		xx			xx			xx							xx			x		
35	2.5	N	x						x		xx							x					
36	5.5	Y	xx		x	x	x			x	xx				x						xx		
37	6.5	Y	x		xx		xx	x		xx	x				x			x					
38	0	N																					
39	3.5	N	xx							x					x			x			xx		
40	1	N													xx								
Total	4.4		20	9	19	14	14	19	10	17	18	2	5	22	0	0	23	2	0	21	2	0

x = identified by one investigator
xx = identified by two investigators
Prox lat arm = proximal lateral arm
SM scap ang = superior medial scapular angle

Scap assist test = scapular assistance test
Lat prot = lateral protraction
Ab = abduction

Table 5: Mean and Standard Deviation Muscle Amplitude (%MVIC)

	Flexion Elevation Task			
	Ascending Phase		Descending Phase	
	Mean	**±SD**	**Mean**	**±SD**
Upper Trapezius	54.01	28.53	28.91	16.75
Middle Trapezius	20.61	22.07	16.02	19.58
Lower Trapezius	26.91	24.15	20.18	24.11
Serratus Anterior	53.57	40.26	26.33	19.86
	Scaption Elevation Task			
	Ascending Phase		Descending Phase	
	Mean	**±SD**	**Mean**	**±SD**
Upper Trapezius	62.70	30.25	33.92	22.35
Middle Trapezius	31.24	35.39	24.53	33.03
Lower Trapezius	25.05	22.99	20.51	23.65
Serratus Anterior	51.06	38.12	29.87	25.03

A simple linear regression was performed to assess how the upper trapezius mean muscle activation amplitude during a sagittal and scapular plane elevation task was able to predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] A simple linear regression analysis for the ascending phase of the flexion task revealed that the mean upper trapezius muscle activation amplitude did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], ($F = 0.568$, $df = 35$, $p = 0.456$), with an r^2 of 0.016. Regression analysis for the descending phase of the flexion task also revealed no significant findings, ($F = 0.092$, $df = 35$, $p = 0.764$), with an r^2 of 0.003. Regression analysis for the ascending phase of the scaption task revealed no significant findings, ($F = 0.010$, $df = 35$, $p = 0.921$), with an r^2 of less than 0.001. Regression analysis for the descending phase of the scaption task also revealed no significant findings, ($F = 0.329$, $df = 35$, $p = 0.570$), with an r^2 of 0.009.

A simple linear regression was performed to assess how the middle trapezius mean muscle activation amplitude during a sagittal and scapular plane elevation task was able to predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] A simple linear regression analysis for the ascending phase of the flexion task revealed that the mean middle trapezius muscle activation amplitude did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], ($F = 0.026$, $df = 35$, $p = 0.874$), with an r^2 of 0.001. Regression analysis for the descending phase of the flexion task also revealed no significant findings, ($F = 0.021$, $df = 35$, $p = 0.886$), with an r^2 of 0.001. Regression analysis for the ascending phase of the scaption task revealed no significant findings, ($F = 0.278$, $df = 35$, $p = 0.601$), with an r^2 of 0.008. Regression analysis for the descending phase of the scaption task also revealed no significant findings, ($F = 2.116$, $df = 35$, $p = 0.154$), with an r^2 of 0.056.

A simple linear regression was performed to assess how the lower trapezius mean muscle activation amplitude during a sagittal and scapular plane elevation task was able to predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] A simple linear regression analysis for the ascending phase of the flexion task revealed that the mean lower trapezius muscle activation amplitude did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], ($F = 0.026$, $df = 35$, $p = 0.873$), with an r^2 of 0.001. Regression analysis for the descending phase of the flexion task also revealed no significant findings, ($F = 0.010$, $df = 35$, $p = 0.919$), with an r^2 of less than 0.001. Regression analysis for the ascending phase of the scaption task revealed no significant findings, ($F = 0.000$, $df = 35$, $p = 0.998$), with an r^2

of less than 0.001. Regression analysis for the descending phase of the scaption task also revealed no significant findings, ($F = 0.196$, $df = 35$, $p = 0.660$), with an r^2 of 0.005.

A simple linear regression was performed to assess how the serratus anterior mean muscle activation amplitude during a sagittal and scapular plane elevation task was able to predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] A simple linear regression analysis for the ascending phase of the flexion task revealed that the mean serratus anterior muscle activation amplitude task did not significantly predict the subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], ($F = 0.214$, $df = 35$, $p = 0.646$), with an r^2 of 0.006. Regression analysis for the descending phase of the flexion task also revealed no significant findings, ($F = 0.000$, $df = 35$, $p = 0.998$), with an r^2 of less than 0.001. Regression analysis for the ascending phase of the scaption task revealed no significant findings, ($F = 0.087$, $df = 35$, $p = 0.770$), with an r^2 of 0.002. Regression analysis for the descending phase of the scaption task also revealed no significant findings, ($F = 0.000$, $df = 35$, $p = 0.982$), with an r^2 of less than 0.001.

DISCUSSION

The purpose of this study was to determine if subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] can be predicted from scapular stabilizer muscle activation. If muscle activation is found to be predictive, the hope is that some level of validation can be established for the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], theoretically allowing sports medicine professionals to better prescribe affected athletes with the most effective rehabilitation exercises when addressing their specific scapular movement and stabilizer strength deficits. Ultimately the goal clinically is

to improve treatment outcome. The rationale for the validation of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] was to test its clinical efficacy as a predictor for the potential, presence, and/or severity of SICK scapula syndrome.

Our results indicate that mean muscle activation amplitude of the scapular stabilizers was not found to be a valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] Our research hypothesis anticipated an increase in upper trapezius muscle activity, as well as decreases in middle trapezius, lower trapezius, and serratus anterior muscle activity as a valid predictor of symptomatic SICK scapula syndrome (i.e. 10 or greater on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1]). Based on our findings however, we conclude that neither upper trapezius muscle over-activation nor middle trapezius, lower trapezius, or serratus anterior muscle under-activation can reliably or accurately predict subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1]

Scapular stabilizer muscles dictate both scapular position and motion and are also considerably inhibited by the presence of pain.[40] Theoretically, EMG analysis of the scapular stabilizers should be an accurate predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], with the scale designed specifically to assess the presence of scapular malpositioning and shoulder pain.

The muscles originating and inserting on the scapula are the driving force behind all scapular and glenohumeral kinematic movement. Therefore, it was inferred that scapular positioning and motion are notably impacted when there is a disruption of this dynamic link via scapular muscle activation dysfunction.

Scapular muscle activation dysfunction refers specifically to strength deficits of the middle trapezius, lower trapezius, and serratus anterior muscles in relation to the upper trapezius muscle.[13] Most researchers agree that the serratus anterior and the lower trapezius are inherently the most susceptible of the scapular stabilizers to the effects of both weakness and inhibition.[15, 21, 23, 36] When the serratus anterior and lower trapezius suffer weaknesses, there is a significant decrease in scapular upward rotation.[37] Also, inadequate serratus anterior function prevents the anchoring of the inferior scapular angle to the wall of the thorax, thus also preventing smooth scapular movement.[3] Essentially, scapular malpositioning and dyskinesis have a certain dependency on the presence of scapular stabilizer strength deficits.

Bandholm et al[40] studied the effects of pain on force steadiness and related muscle activity. Using healthy subjects and experimental pain, the researchers demonstrated pain's ability to inhibit maximal force production while having no effect on musculature contractile properties. The authors believed that the excitation of the muscle nociceptor afferents facilitate inhibitory pathways during muscle agonist activity.[40]

Both Pink et al[3] and Ludewig et al[10] compared muscle activity in subjects with symptoms of subacromial impingement syndrome to an asymptomatic control group. Significant findings revealed decreased activation of the serratus anterior muscle in the impingement subjects when compared to healthy, control subjects. Similarly, Wadsworth et al[23] demonstrated that the activation of the serratus anterior muscle of swimmers suffering from subacromial impingement syndrome was found to be delayed by three times when performing a scapular plane elevation task as compared to the asymptomatic control group.

Cools et al[49] also utilized a comparison study to observe the differences in scapular muscle activity between overhead athletes with and without impingement symptoms. Here researchers measured the mean EMG activity of the upper trapezius, middle trapezius, and lower trapezius during isokinetic glenohumeral abduction and external rotation. Their results showed significantly decreased activity in the lower trapezius during the glenohumeral abduction task and decreased activity in the middle trapezius during the glenohumeral external rotation task in individuals with impingement symptoms when compared to healthy, control subjects.

In the presence of shoulder pain, the literature clearly supports the linkage of altered scapular stabilizer muscle activity and the initiation of some degree of scapular malpositioning and/or dyskinesis. Over half of the subjects tested in this study self-reported as having a painful shoulder, yet only four scored higher than 10 out of 20 points on the scale, which is a criteria clinically used to diagnose individual as having SICK scapula syndrome. If subjects are categorically symptomatic for self-reported shoulder pain, yet qualify as "healthy" upon assessment, one must question both the sensitivity and accuracy of the instrumentation being utilized for pathology detection.

In attempting to explain why altered EMG of the scapular stabilizers was unable to predict SICK scapula syndrome score in this study, we must acknowledge flaws in both the structural and theoretic framework of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1]

Pilot work, an a priori reliability study, and the thesis project itself allowed researchers to screen over 100 athletes utilizing the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] Out of all the athletes who were screened, only four were clinically

diagnosed as having a SICK scapula syndrome. The principal investigator suggests two structural flaws within the scale's framework. The first flaw is the ambiguity of the questionnaire and the second is the threshold scoring.

Questionnaire ambiguity was encountered in the form of subject confusion regarding the subjective portion of the screening process. One common example of such uncertainty often came in response to the question, "do you ever have pain on, in between, or around your shoulder blades?" Subjects seemed reluctant to answer yes but would later test positive for tenderness to palpation of scapular soft tissue structures. Athletes seemingly had the perception that muscular pain in the form of myofascial trigger points was unrelated to the subacromial and/or internal impingement symptoms they may have been experiencing. We believe this misunderstanding among athletes to be extremely reasonable considering the nature, source, and severity of their pain is so inherently different. Question clarification by the principal investigator may have allowed athletes to give a more appropriate response to a subjective symptom question; however, we felt that doing so may affect the athlete responses.

Threshold scoring was utilized for the scapular malpositioning portion of subject screening. While this method was deemed the most appropriate to maintain adequate inter-tester reliability, it often resulted in a lower score than the amount of malposition would suggest. For example, while measuring scapular abduction, a subject may have a four-degree abduction discrepancy between scapulae in resting position but would receive no points, because it failed to reach the five-degree threshold that would have allotted one point.

As was previously mentioned, there are definitive links between shoulder pain, scapular malpositioning, and altered scapular stabilizer muscle activity, however we must acknowledge that combinations of the three can be mutually exclusive to one another.

Shoulder pain can exist without the presence of scapular malpositioning and/or dyskinesis. Myers et al[32] found that throwers with pathologic internal impingement typically present with posterior shoulder pain. If screened utilizing the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], these athletes may receive up to 11 subjective and objective points but display no scapular malpositioning. In such instances, these athletes would be clinically diagnosed as having SICK scapula syndrome without the defining characteristics of **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement.

Suggesting some flaws with the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1], we also recognize the asymptomatic scapular malpositionings demonstrated as healthy adaptations in overhead athletes. Myers et al[13] found an adaptive increase in scapular upward rotation among normal, healthy throwing athletes. This adaptation was believed by the authors to assist in the achievement of subacromial clearance throughout the throwing movement pattern, thus acting as a means of preventing subacromial impingement. Possessing marked scapular malpositioning does not always mandate an involved symptomatic shoulder.

Lastly, scapular stabilizer deficits can exist without the presence of either shoulder pain or scapular malpositioning. Weakness and/or inhibition-based muscle dysfunction of the scapular stabilizers is a common trend among overhead athletes, where there is adaptive muscle shortening of the pectoralis minor in conjunction with an adaptive muscle weakening

of the middle trapezius, lower trapezius, serratus anterior, rhomboid minor, and rhomboid major.[9, 34, 35] The presence of upper extremity muscle imbalances is generally accepted to be the result of sport-specific pattern dominance and can be entirely asymptomatic in nature.[9, 15, 21, 23, 34-36]

While one criticism regarding the theoretic framework of the scale refers to inflated scores without the true presence of scapular malpositioning, an inverse criticism is the relative difficulty of scoring in general, even in the presence of both shoulder pain and scapular malpositioning.

The subjective and objective portions of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] dually assess an athlete for a wide range of chronic shoulder pathologies, from AC joint sprain to thoracic outlet syndrome to subacromial impingement syndrome. While an athlete may possess all of the classic characteristics of SICK scapula syndrome (i.e. **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and malposition, and dys**K**inesis of scapular movement), he or she may not score any points for AC joint irritation, TOS parathesias, or subacromial impingement syndrome. As a result, subject score would be rather low on both the subjective and objective portions of the scale, thus allowing the subject to appear seemingly healthy. Because total subject score is heavily reliant on reports of pain in these two sections, only gross scapular malpositioning (i.e. > 15 degrees of scapular abduction) would result in a clinical diagnosis of SICK scapula syndrome. Studies comparing healthy subjects and subjects experiencing shoulder pain found only modest differences (i.e. < 5 degrees) in scapular kinematics between groups.[4, 5, 8, 9, 11, 12, 18] A 15-degree scapular asymmetry is not only uncommon among ill-maintenance shoulders, it is relatively non-existent among Division I overhead athletes.

The SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale[1] was designed as a structured clinical tool that could be utilized by clinicians as a predictor for the potential, presence, and/or severity of SICK scapula syndrome. However, it appears to have rather poor predictive value. In this study, SICK scapula syndrome and other chronic shoulder pathologies proved to be far too multi-factorial in nature for one all encompassing number.

Limitations

Perhaps the greatest limitation of this study was the narrow and low-ended range of subject scores collected utilizing the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] While subject recruitment made no distinction regarding a need for either symptomatic or asymptomatic shoulders, upon screening, only four subjects scored higher than 11 out of a possible 20 points, which is a minimum cutoff score commonly used to clinically diagnose individual as having SICK scapula syndrome. Again, we attribute unexpected subject scoring to flaws of both the structural and theoretic framework of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1]

Due to in-season compliance issues, this study was subject to an unintentional exclusion of baseball players. Baseball players, specifically baseball pitchers, often experience shoulder or elbow pain that prevents them from participation in sports at some point in their careers.[44] Therefore, caution must be used in extrapolating our findings to a baseball pitcher population.

Future Research

Future research should seek to further identify scapular muscle dysfunction in symptomatic overhead athletes, specifically those suffering with SICK scapula syndrome. Based on our findings, we believe that it is necessary to conduct a more large-scale study of overhead athletes, perhaps focusing on ill-maintained shoulders to better exemplify the condition in its most exaggerated form.

Another potential avenue regarding future research would be the construction of a more theoretically sound screening instrument, similar in principle to the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] SICK scapula syndrome represents a collection of signs and symptoms commonly seen in individuals with various shoulder pathologies. A more fine-tuned, pathology-specific screening tool may allow clinicians to more reliably identify and quantify shoulder injuries for clinical purpose.

We acknowledge the findings listed in Table 4 when offering sound recommendations regarding the development of a new and theoretically improved screening instrument. By dissecting SICK scapula syndrome score for each of the forty subjects screened, we were able to tease out exactly where the bulk of point allotment occurred, specifically among those with self-reported shoulder pain. Based on score breakdown, we conclude that ten characteristics of SICK scapula syndrome best exemplify the condition's signs and symptoms, thus serving as the most accurate predictors regarding its presence and severity. We recommend the following subjective questions for the presence of pain: coracoid process, periscapular, proximal lateral arm, and radicular symptoms. We recommend the following objective palpations and/or special tests for the presence of pain: coracoid process, superior medial scapular angle, and Hawkins-Kennedy Impingement Test.

We recommend the following measurements for the determination of scapular malpositioning: infera 0 to 1 cm, lateral protraction 0 to 1 cm, and abduction 0 to 5 degrees.

We also recommend the implementation of a more detailed objective screening process; one which includes a postural assessment, observation and measurement of dynamic scapular positioning, soft-tissue mobility assessment, and scapular muscle strength assessment. Postural assessments should seek to identify and grade the presence of cervical lordosis, thoracic kyphosis, lumbar lordosis, pelvic rotations, and abnormal hip rotations that may affect scapular kinematics as energy is transferred through the kinetic chain from the lower extremity and core to the thorax and upper extremity.[9] Clinicians may perform a quick and effective postural assessment utilizing a plumb-line while observing the patient from a side-view.

Scapular position should be observed at rest and during loaded and unloaded humeral elevation. While in resting position, clinicians should observe the scapulae for signs of winging (i.e. excessive scapular internal rotation, scapular anterior tilt, and scapular elevation). Dynamic scapular motion should be assessed in both loaded and unloaded conditions. Johnson et al.[64] developed a protocol to detect abnormal scapular motion via the repetitive challenging of the scapulae under loaded conditions. The authors data indicated that three tests were able to detect abnormal scapular motion: 1) observation of bilateral scapular motion during five to ten repetitions of unloaded humeral elevation in the scapular plane (scaption) to establish a baseline of scapular movement, 2) observation of bilateral scapular motion during five to ten repetitions of loaded (0.5-5 kg) scaption, and 3) observation of unilateral scapular motion during resisted isometric external rotation with the arm at the side in neutral rotation (i.e. scapular flip sign).[64]

The scapular lateral slide test is a semi-dynamic, quantitative assessment of scapular position. This test has been shown to be reliable in assessing the bilateral position of the scapulae in relation to a fixed point on the spine as varying loads are placed on the supporting scapular musculature.[9] The test involves a series of three measurement positions.

Evaluation of the mobility of the posterior glenohumeral joint capsule, the posterior shoulder musculature, and the anterior coracoid musculature provides critical information regarding the pathomechanic assessment of scapular dysfunction. Posterior glenohumeral joint capsule contracture has been shown to produce excessive superior and anterior humeral head translation, thereby compromising the size of the subacromial space and altering glenohumeral and scapular kinematics.[10, 19, 25] Posterior shoulder tightness is an additional commonly described flexibility characteristic of scapular dysfunction. [1, 3, 8,9, 13, 17, 32] Myers et al. quantify posterior shoulder tightness utilizing supine and side-lying horizontal adduction assessments.[65] One final flexibility measurement to consider during scapular evaluation is pectoralis minor mobility. Due to its proximal attachment on the coracoid process of the scapula, inflexibility of the pectoralis minor muscle may manifest as excessive scapular anterior tilt and internal rotation, thus resulting in coracoid process pain and scapular dysfunction.

Manual muscle testing of the scapular stabilizing muscles is critical in determining the presence of or potential for scapular dysfunction. Strength of the middle and lower trapezius, rhomboids major and minor, and the serratus anterior muscles should be assessed through manual muscle testing techniques. Additional scapular muscle strength and endurance tests include the isometric scapular retraction pinch and wall push up tests. Typically, patients are able to hold an isometric pinch of the scapulae in retraction for 15 to

20 seconds without the onset of burning pain or muscle weakness. An inability to hold this position due to pain or weakness provocation is a positive sign indicating scapular muscle dysfunction.[9] The ability of the serratus anterior muscle to stabilize the scapula on the thorax is easily evaluated with the wall push-up test. The patient performs 5-10 wall push-ups while the clinicians observes for abnormalities in scapular position and motion, specifically scapular winging.[9]

Conclusion

This study is the first to assess the validity of the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] In overhead athletes, mean muscle activation amplitude of the scapular stabilizers was not found to be valid predictor of subject score on the SICK Scapula, Static Measurements, 0 to 20 Point Rating Scale.[1] However, the results of this study need to be interpreted with caution, because the majority of subjects scored lower than the commonly accepted threshold score for the clinical diagnosis of SICK scapula syndrome.

APPENDIX D: CONSENT FORM

University of North Carolina-Chapel Hill
Consent to Participate in a Research Study
Adult Subjects Biomedical Form

IRB Study #_____07-1689_____
Consent Form Version Date: _____11/19/07_____
Title of Study: A Validation of the SICK Scapula Rating Scale in Overhead Athletes: Prediction of Score from Strength, Flexibility, Muscle Activation, and Kinematic Analysis
Principal Investigator: Karen Tankersley, BS, ATC-L ; Sarah Vizza, BS, ATC-L
UNC-Chapel Hill Department: Exercise and Sport Science
UNC-Chapel Hill Phone number: 919-962-2067
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Faculty Advisor: Joseph Myers, PhD, ATC
Funding Source:
Study Contact telephone number: 919-962-2067
Study Contact email: ktankers@email.unc.edu; svizza@email.unc.edu

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What are some general things you should know about research studies?
You are being asked to take part in a research study. To join the study is voluntary. You may refuse to join, or you may withdraw your consent to be in the study, for any reason.

Research studies are designed to obtain new knowledge that may help other people in the future. You may not receive any direct benefit from being in the research study. There also may be risks to being in research studies.

Deciding not to be in the study or leaving the study before it is done will not affect your relationship with the researcher, your health care provider, or the University of North Carolina-Chapel Hill. If you are a patient with an illness, you do not have to be in the research study in order to receive health care.

Details about this study are discussed below. It is important that you understand this information so that you can make an informed choice about being in this research study. You will be given a copy of this consent form. You should ask the researchers named above, or staff members who may assist them, any questions you have about this study at any time.

What is the purpose of this study?
The purpose of this study is to validate a clinical shoulder assessment tool called the SICK Scapula Rating Scale. This study is designed to look at shoulder strength, flexibility, shoulder blade movement, and shoulder blade muscle activity in athletes who use their arms over their heads.

You are being asked to volunteer for this study because you actively participate in a physical activity at least 3 times per week for a minimum of 30 minutes each session, one in which your arms are required to be over your head for a significant period of time within each session. It is believed that physically active individuals participating in repetitive overhead activities are at greatest risk for exhibiting alterations of normal position or motion of the shoulder blades.

Are there any reasons you should not be in this study?
You should not be in this study if you have a history of shoulder or neck surgery, rotator cuff tear, cervical spine pathology, history of acute-onset shoulder pathology within the last six months, adhesive capsulitis, history of unstable episodes within the past six months (glenohumeral subluxation, dislocation, self-subluxation), or scoliosis.

How many people will take part in this study?
If you decide to be in this study, you will be one of approximately 60 people in this research study.

How long will your part in this study last?
If you participate in this study, you will spend approximately 90 minutes during one testing session. A follow up session is not required.

What will happen if you take part in the study?
You will be asked to report to the Motor Control Lab located in 123 Fetzer on the UNC-CH campus. Male subjects will be asked to remove their shirt, and female subjects will be asked to wear either an athletic bra or tank-top. You will be asked questions regarding your shoulder history to ensure that you meet this study's criteria. You will then be measured for both height and weight and briefed on testing procedures. Your shoulder will then be evaluated by two Certified Athletic Trainers. They will ask you questions regarding your shoulder pain and take measurements around your shoulder. Following your briefing session, you will select a random task completion order for two shoulder elevation tasks.

During testing, male subjects will be required to take off their shirt and female subjects will be in a tank-top or wearing an athletic bra. This is to allow exposure of your shoulder blades and arms for strength testing and sensor/electrode placement.

Band-aid like electrodes that measure muscle activity will be attached over muscles on back of your neck, below your shoulder blade, and on the side of your trunk, just below your armpit. Sensors that measure joint motion will be placed on back of your neck, your shoulder, and close to your elbow. All of these sensors will then be secured with tape.

Prior to testing, you will perform one sub-maximal contraction for each of the previously mentioned muscles around the shoulder and upper back to adequately familiarize yourself with proper form for each manual muscle test. Following this warm-up and learning session, an investigator will apply a small force to your forearm, and you will be asked to hold your arm as still as possible for approximately five seconds. This process will be repeated in four different arm positions and three trials will be recorded for each position.

After the setup and baseline measurement has been completed, you will complete two lifting tasks. The first lifting task will require you to raise your arms above your head while they're directly in front of you. The second lifting task will require you to raise your arms above your head while they're off to the side of your body. You will lift your upper arm at shoulder while keeping your elbow straight over your head as far as possible. This will be done at a controlled movement velocity while keeping in time with a digital metronome. Each lifting task will require ten continuous repetitions, with each repetition lasting approximately four seconds. You will be given a 2 minute rest period between each lifting task. Lastly, your shoulder flexibility will be measured.

What are the possible benefits from being in this study?

Research is designed to benefit society by gaining new knowledge. You may not benefit personally from participating in this study.

What are the possible risks or discomforts involved with being in this study?

If you are selected for participation in this study, there is a risk of common discomfort that may be experienced during and following each of the two functional tasks. You may potentially experience mild discomfort during and following each of the two functional tasks, which can be attributed to the onset of muscle soreness due to temporary overuse. The discomfort that may be experienced with participation is similar to that associated with overhead athletic participation and/or activities of daily living in which your arms are being used over your head. In addition, there may be uncommon or previously unknown risks that might occur. You should report any problems to the researchers. If such problems occur, the researchers will assist you in obtaining medical care. However, any costs for the medical care will be billed to you or your insurance company. The University of North Carolina at Chapel Hill has not set aside funds to pay for any such reactions or injuries, or for the related medical care. However, by signing this consent form, you do not give up any legal rights.

What if we learn about new findings or information during the study?

You will be given any new information gained during the course of the study that might affect your willingness to continue your participation.

How will your privacy be protected?

You will not be identified in any report or publication about this study. Although every effort will be made to keep research records private, there may be times when federal or state law requires the disclosure of such records, including personal information. This is very unlikely, but if disclosure is ever required, UNC-Chapel Hill will take steps allowable by law to protect the privacy of personal information. In some cases, your information in this research study could be reviewed by representatives of the University, research sponsors, or government agencies for purposes such as quality control or safety.

What will happen if you are injured by this research?

All research involves a chance that something bad may happen to you. This may include the risk of personal injury. In spite of all safety measures, you might develop a reaction or injury

from being in this study. If such problems occur, the researchers will help you get medical care, but any costs for the medical care will be billed to you and/or your insurance company. The University of North Carolina at Chapel Hill has not set aside funds to pay you for any such reactions or injuries, or for the related medical care. However, by signing this form, you do not give up any of your legal rights.

What if you want to stop before your part in the study is complete?
You can withdraw from this study at any time, without penalty. The investigators also have the right to stop your participation at any time. This could be because you have had an unexpected reaction, or have failed to follow instructions, or because the entire study has been stopped.

Will you receive anything for being in this study?
You will not receive anything for taking part in this study.

Will it cost you anything to be in this study?
No cost will be required of you for this study.

What if you are a UNC student?
You may choose not to be in the study or to stop being in the study before it is over or at any time. This will not affect your class standing or grades at UNC-Chapel Hill. You will not be offered or receive any special consideration if you take part in this research. You may choose not to participate or withdrawal from the study at any time or for any reason without jeopardizing your relationship with your coach, athletic trainer, or physician and without being penalized in any way. If you are an athlete, there will be no benefit or consequence to your standing on your athletic team in any way.

What if you have questions about this study?
You have the right to ask, and have answered, any questions you may have about this research. If you have questions, or if a research-related injury occurs, you should contact the researchers listed on the first page of this form.

What if you have questions about your rights as a research subject?
All research on human volunteers is reviewed by a committee that works to protect your rights and welfare. If you have questions or concerns about your rights as a research subject you may contact, anonymously if you wish, the Institutional Review Board at 919-966-3113 or by email to IRB_subjects@unc.edu.

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IRB Study # 07-1689
Title of Study: A Validation of the SICK Scapula Rating Scale in Overhead Athletes: Prediction of Score from Strength, Flexibility, Muscle Activation, and Kinematic Analysis
Principal Investigators: Karen Tankersley, BS, ATC-L ; Sarah Vizza, BS, ATC-L

Subject's Agreement:

I have read the information provided above. I have asked all the questions I have at this time. I voluntarily agree to participate in this research study.

_____ _____
Signature of Research Subject Date

_____
Printed Name of Research Subject

_____ _____
Signature of Person Obtaining Consent Date

_____
Printed Name of Person Obtaining Consent

REFERENCES

1. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology Part III: The SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. *Arthroscopy.* Jul-Aug 2003;19(6):641-661.

2. Fayad F HG, Yazbeck C, Lefevre-colau MM, Poiraudeau S, Revel M, Roby-Brami A. 3-D Scapular Kinematics During Arm Elevation: Effect of Motion Velocity. *Journal of Clinical Biomechanics.* 2006;21:932-941.

3. Pink MM, Tibone JE. The painful shoulder in the swimming athlete. *Orthop Clin North Am.* Apr 2000;31(2):247-261.

4. McClure PW, Michener LA, Karduna AR. Shoulder function and 3-dimensional scapular kinematics in people with and without shoulder impingement syndrome. *Phys Ther.* Aug 2006;86(8):1075-1090.

5. Warner JJ, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Scapulothoracic motion in normal shoulders and shoulders with glenohumeral instability and impingement syndrome. A study using Moire topographic analysis. *Clin Orthop Relat Res.* Dec 1992(285):191-199.

6. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part II: evaluation and treatment of SLAP lesions in throwers. *Arthroscopy.* May-Jun 2003;19(5):531-539.

7. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology Part I: pathoanatomy and biomechanics. *Arthroscopy.* Apr 2003;19(4):404-420.

8. Hebert LJ, Moffet H, McFadyen BJ, Dionne CE. Scapular behavior in shoulder impingement syndrome. *Arch Phys Med Rehabil.* Jan 2002;83(1):60-69.

9. Kibler WB, McMullen J. Scapular dyskinesis and its relation to shoulder pain. *J Am Acad Orthop Surg.* Mar-Apr 2003;11(2):142-151.

10. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther.* Mar 2000;80(3):276-291.

11. Lukasiewicz AC MP, Michener L et al. Comparison of 3-Dimensional Scapular Position and Orientation Between Subjects With and Without Shoulder Impingement. *J Orthop Sports Phys Ther.* 1999;29:574-583.

12. McClure PW, Bialker J, Neff N, Williams G, Karduna A. Shoulder function and 3-dimensional kinematics in people with shoulder impingement syndrome before and after a 6-week exercise program. *Phys Ther.* Sep 2004;84(9):832-848.

13. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Scapular position and orientation in throwing athletes. *Am J Sports Med.* Feb 2005;33(2):263-271.

14. Su KP, Johnson MP, Gracely EJ, Karduna AR. Scapular rotation in swimmers with and without impingement syndrome: practice effects. *Med Sci Sports Exerc.* Jul 2004;36(7):1117-1123.

15. Thigpen CA PDea. Effects of Forward Head and Rounded Shoulder Posture on Scapular Kinematics, Muscle Activity, and Shoulder Coordination. In Press.

16. Tsai NT, McClure PW, Karduna AR. Effects of muscle fatigue on 3-dimensional scapular kinematics. *Arch Phys Med Rehabil.* Jul 2003;84(7):1000-1005.

17. Fleisig GS, Barrentine SW, Escamilla RF, Andrews JR. Biomechanics of overhand throwing with implications for injuries. *Sports Med.* Jun 1996;21(6):421-437.

18. Ludewig PM CT. Alterations in Shoulder Kinematics and Associated Muscle Activity in People With Symptoms of Shoulder Impingement. *Phys Ther.* 2000;80:276-291.

19. Kibler WB. The role of the scapula in athletic shoulder function. *Am J Sports Med.* Mar-Apr 1998;26(2):325-337.

20. Moseley JB, Jr., Jobe FW, Pink M, Perry J, Tibone J. EMG analysis of the scapular muscles during a shoulder rehabilitation program. *Am J Sports Med.* Mar-Apr 1992;20(2):128-134.

21. Cools AM, Witvrouw EE, Declercq GA, Danneels LA, Cambier DC. Scapular muscle recruitment patterns: trapezius muscle latency with and without impingement symptoms. *Am J Sports Med.* Jul-Aug 2003;31(4):542-549.

22. Ludewig PM, Hoff MS, Osowski EE, Meschke SA, Rundquist PJ. Relative balance of serratus anterior and upper trapezius muscle activity during push-up exercises. *Am J Sports Med.* Mar 2004;32(2):484-493.

23. Wadsworth DJ, Bullock-Saxton JE. Recruitment patterns of the scapular rotator muscles in freestyle swimmers with subacromial impingement. *Int J Sports Med.* Nov 1997;18(8):618-624.

24. Myers JB, Ju YY, Hwang JH, McMahon PJ, Rodosky MW, Lephart SM. Reflexive muscle activation alterations in shoulders with anterior glenohumeral instability. *Am J Sports Med.* Jun 2004;32(4):1013-1021.

25. Garrett WE SK, Kirkendall DT. Principles and Practice of Orthopaedic Sports Medicine. 2000:329-510.

26. Medicine NAoS. Performance Enhancement Specialist. 2007.

27. Myers JB, Lephart SM. The Role of the Sensorimotor System in the Athletic Shoulder. *J Athl Train.* Jul 2000;35(3):351-363.

28. Wu. ISB Recommendation on Definitions of Joint Coordinate Systems of Various Joints for the Reporting of Human Joint Motion—Part II: Shoulder, Elbow, Wrist and Hand. *Journal of Biomechanics.* 2005;38:981-992

29. Ebaugh DD, McClure PW, Karduna AR. Scapulothoracic and glenohumeral kinematics following an external rotation fatigue protocol. *J Orthop Sports Phys Ther.* Aug 2006;36(8):557-571.

30. Bach HG, Goldberg BA. Posterior capsular contracture of the shoulder. *J Am Acad Orthop Surg.* May 2006;14(5):265-277.

31. Myers JB, Guskiewicz KM, Schneider RA, Prentice WE. Proprioception and Neuromuscular Control of the Shoulder After Muscle Fatigue. *J Athl Train.* Oct 1999;34(4):362-367.

32. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Glenohumeral range of motion deficits and posterior shoulder tightness in throwers with pathologic internal impingement. *Am J Sports Med.* Mar 2006;34(3):385-391.

33. Tyler TF NS, Roy T, Gleim GW. Quantification of posterior capsule tightness and motion loss in patients with shoulder impingement. *AM J Sports Med.* 2000;29(4):668-673.

34. Borstad JD, Ludewig PM. The effect of long versus short pectoralis minor resting length on scapular kinematics in healthy individuals. *J Orthop Sports Phys Ther.* Apr 2005;35(4):227-238.

35. Ludewig PM, Cook TM, Nawoczenski DA. Three-dimensional scapular orientation and muscle activity at selected positions of humeral elevation. *J Orthop Sports Phys Ther.* Aug 1996;24(2):57-65.

36. Smith J, Dahm DL, Kaufman KR, et al. Electromyographic activity in the immobilized shoulder girdle musculature during scapulothoracic exercises. *Arch Phys Med Rehabil.* Jul 2006;87(7):923-927.

37. Ebaugh DD, McClure PW, Karduna AR. Three-dimensional scapulothoracic motion during active and passive arm elevation. *Clin Biomech (Bristol, Avon).* Aug 2005;20(7):700-709.

38. Myers JB, Riemann BL, Ju YY, Hwang JH, McMahon PJ, Lephart SM. Shoulder muscle reflex latencies under various levels of muscle contraction. *Clin Orthop Relat Res.* Feb 2003(407):92-101.

39. Safran MR BP, Lephart SM, Fu FH, Warner JJP Shoulder Proprioception in Baseball Pitchers. *J Shoulder Elbow Surg.* 2001;10:438-444.

40. Bandholm T, Rasmussen L, Aagaard P, Jensen BR, Diederichsen L. Force steadiness, muscle activity, and maximal muscle strength in subjects with subacromial impingement syndrome. *Muscle Nerve.* Nov 2006;34(5):631-639.

41. Hughes RE, An KN. Force analysis of rotator cuff muscles. *Clin Orthop Relat Res.* Sep 1996(330):75-83.

42. Konrad. ABC of EMG – A Practical Introduction to Kinesiological Electromyography. 2005.

43. Michener LA, Boardman ND, Pidcoe PE, Frith AM. Scapular muscle tests in subjects with shoulder pain and functional loss: reliability and construct validity. *Phys Ther.* Nov 2005;85(11):1128-1138.

44. Myers. On-the-Field Resistance-Tubing Exercises for Throwers: An Electromyographic Analysis. *J Athl Train.* 2005;40(1):15-22.

45. Reddy AS, Mohr KJ, Pink MM, Jobe FW. Electromyographic analysis of the deltoid and rotator cuff muscles in persons with subacromial impingement. *J Shoulder Elbow Surg.* Nov-Dec 2000;9(6):519-523.

46. Conner. The Relationship Between Humeral Rotation and Scapular Tipping. *In Submission.* 2006.

47. McMahon. Comparative electromyographic analysis of shoulder muscles during planar motions: anterior glenohumeral instability versus normal. *J Shoulder Elbow Surg.* 1996;5(2):118-123.

48. Cools. Scapular Muscle Recruitment Pattern: Electromyographic Response of the Trapezius Muscle to Sudden Shoulder Movement Before and After a Fatiguing Exercise. *J Orthop Sports Phys Ther.* 2002;32(5):221-229.

49. Cools. Trapezius Activity and Intramuscular Balance During Isokinetic Exercise in Overhead Athletes with Impingement Symptoms. *Scand J Med Sci Sports.* 2007;17:25-33.

50. McMaster WC RA, Stoddard T. A Correlation Between Shoulder Laxity and Interfering Pain in Competitive Swimmers. *The American Journal of Sports Medicine.* 1998;26(1):83-86.

51. Cools. Rehabilitation of Scapular Muscle Balance: Which Exercises to Prescribe? *Am J Sports Med.* 2007;X(X):1-8.

52. Thigpen CA PDea. Effects of Forward Head and Rounded Shoulder Posture on Scapular Kinematics, Muscle Activity, and Shoulder Coordination. *In Press.* 2006.

53. Bull. Accuracy of an electromagnetic tracking device. *J Biomech.* 1997;30(8):857-859.

54. Bull. Accuracy of an electromagnetic measurement device and application to the measurement and description of knee joint motion. *Proc Inst Mech Eng.* 1998;212(5):347-355.

55. Eckhouse. A comparison of kinematic recording instruments. *J Med Syst.* 1996;20(6):429-456.

56. Finley. Reliability of biomechanical variables during wheelchair ergometry testing. *J Rehabil Res Dev.* 2002;39(1):73-81.

57. Milne. Accuracy of an electromagnetic tracking device: a study of the optimal range and metal interference. *J Biomech.* 1996;29(6):791-793.

58. Kendall. Muscles: Testing and Function with Posture and Pain. 2005.

59. Ludewig. Comparison of Surface Sensor and Bone-Fixed Measurement of Humeral Motion. *J Appl Biomech.* 2002;18(2).

60. Borstad JD, Ludewig PM. Comparison of scapular kinematics between elevation and lowering of the arm in the scapular plane. *Clin Biomech (Bristol, Avon).* Nov-Dec 2002;17(9-10):650-659.

61. Thigpen CA, Padua DA, Morgan N, Kreps C, Karas SG. Scapular kinematics during supraspinatus rehabilitation exercise: a comparison of full-can versus empty-can techniques. *Am J Sports Med.* Apr 2006;34(4):644-652.

62. Veeger HE, van der Helm FC, Chadwick EK, Magermans D. Toward standardized procedures for recording and describing 3-D shoulder movements. *Behav Res Methods Instrum Comput.* Aug 2003;35(3):440-446.

63. Portney LG WM. *Foundations of Clinical Research Applications to Practice*. 2nd ed. Upper Saddle River, NJ: Prentice Hall Health; 2000.

64. Johnson MP. New method to assess scapular upward rotation in subjects with shoulder pathology. *J Orthop Sports Phys Ther.* 2001 Feb;31(2):81-9.

65. Myers JB. Reliability, Precision, Accuracy, and Validity of Posterior Shoulder Tightness Assessment in Overhead Athletes. In Press. 2007.