BIOMECHANICAL EVALUATION OF GLENOHUMERAL JOINT STABILIZING MUSCLES DURING PROVOCATIVE TESTS DESIGNED TO DIAGNOSE SUPERIOR LABRUM ANTERIOR-POSTERIOR LESIONS

by

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A thesis

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of the requirements for the degree of

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The following individuals read and discussed the thesis submitted by student Vanessa J.C. Wood, and they evaluated her presentation and response to questions during the final oral examination. They found that the student passed the final oral examination.

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ABSTRACT

Biomechanical Evaluation of Glenohumeral Joint Stabilizing Muscles during Provocative Tests Designed to Diagnose SLAP Lesions

Vanessa Wood

Despite considerable advances in the understanding of glenohumeral (GH) biomechanics and glenoid labral pathologies, arthroscopy remains the only definitive means of Superior Labrum Anterior-Posterior (SLAP) lesion diagnosis. Unfortunately, natural GH anatomic variants limit the reliability of radiography. Accurate clinical diagnostic techniques would be advantageous due to the invasiveness, patient risk, and financial cost associated with arthroscopy. Twenty provocative tests designed to elicit labral symptoms as a diagnostic sign have shown promising accuracy by their respective original authors, but later studies generally fail to reproduce those findings. The purpose of this study was to compare the behavior of GH joint stabilizing muscles in promising tests. Electromyography (EMG) was used to characterize the activation of GH joint stabilizing muscles, with particular interest in the Long Head Biceps Brachii (LHBB) behavior, as activation of the LHBB and subsequent tension in the biceps tendon should illicit labral symptoms in SLAP lesion patients.

Volunteers (n=21) with no history of shoulder pathology were recruited for this study. The tests analyzed were Active Compression, Speed's, Pronated Load, Biceps Load I (Bicep I), Biceps Load II (Bicep II), Resisted Supination External Rotation (RSER), and Yergason's. Test modifications that allowed the use of the Biodex System improved reproducibility. EMG was used to record activity for GH muscles: the LHBB, short head of the biceps brachii, anterior deltoid, pectoralis major, latissimus dorsi, infraspinatus, and supraspinatus. An indwelling electrode was used to monitor supraspinatus activity, and the remaining muscles utilized surface electrodes. EMG data were recorded at 1250 Hz and filtered with custom MATLAB software. Muscle activity for each test was characterized by activation and selectivity. Muscle activation was defined as the muscle's peak normalized EMG amplitude. Muscle selectivity was defined as the ratio of muscle activation for the muscle of interest over the sum of all seven muscles' peak activations.

Results indicated that Bicep I and II had the greatest potential for the clinical detection of SLAP lesions because both tests 1) elicited large LHBB activation, suggesting that during these tests more tension was applied to the biceps tendon, and also 2) remained highly selective for the LHBB, which should reduce the potential sources for confounding results. Also, tests that elicited promising LHBB behavior for either a single suite or for both activation and selectivity, shared design patterns relating to location of the applied load, forearm orientation, joint position, and line of pull. These characteristics should be further examined to determine their potential role in optimizing SLAP test design and improving clinical diagnostic techniques.

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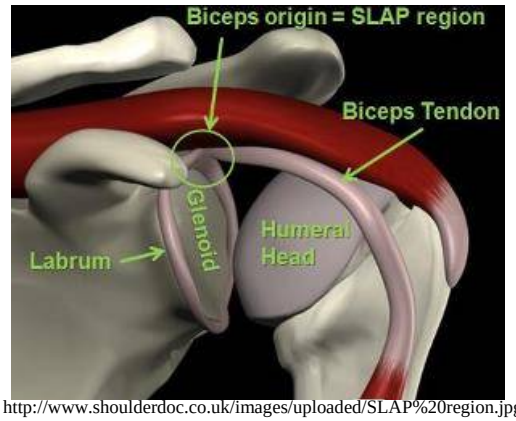
INTRODUCTION

The shoulder complex is an inherently complicated system with regards to both structure and function; hence improving prevention, detection, and treatment of glenohumeral pathologies has been difficult. Fortunately, shoulder biomechanics is an expanding and developing field that has and will continue to play an instrumental role in furthering the understanding of the anatomy and behavior of the shoulder complex and in enhancing the ability of medicine to detect and repair various pathologies. Advancements in the biomechanical analysis of the glenohumeral joint and in the ability of medicine to manage shoulder injuries are closely related, and both biomechanics and medicine have reaped considerable benefits from technological and scientific developments in medical imaging and surgical techniques that have enabled a new perspective of the glenohumeral joint with regards to both form and function.



http://www.shoulderdoc.co.uk/images/uploaded/SLAP%20region.jpg

Figure 1: SLAP Lesion Injury Region

The advent of shoulder arthroscopy as a medical tool led to the initial identification and description of glenoid labral tears in 1985, a glenohumeral

musculoskeletal pattern and abnormality that was previously undetectable [1]. The specific glenoid labral pathology, a superior labrum anterior-posterior tear was coined 'SLAP lesion' in 1990 (*Figure 1*) [32]. Although it has been more than two decades since SLAP lesions were defined and despite considerable advances in the understanding of glenohumeral biomechanics and glenoid labral pathologies, SLAP lesion detection remains difficult. Radiography and physical examination have proven useful for assessing a wide variety of orthopedic injury, but have shown limited potential with regards to SLAP lesion detection. Arthroscopy remains the 'gold standard' and the only definitive means of SLAP lesion diagnosis [18, 22, 33 4, 8]. An alternative to shoulder arthroscopy would be advantageous due to the invasiveness, financial cost, and patient risk associated with arthroscopy.

Recent developments in advanced imaging methods have drastically improved the diagnostic reliability of radiography, particularly in the detection of musculoskeletal patterns and injuries that previously, due to the low contrast of x-ray and computed tomography (CT), were impossible to identify with radiography. Although, magnetic resonance (MR) arthrography, specifically in high contrast, has shown some promise as a supplementary tool in SLAP lesion diagnosis, natural anatomic variants inherent in shoulder anatomy limit the reliability of radiographic diagnoses [3, 4, 7, 21, 26, 35]. Furthermore, problems with radiography follow those of arthroscopy; MR arthrography, for example, can be invasive, expensive, and dangerous, causing life threatening allergic reactions in some patients [5]. Therefore radiography is an imperfect means of SLAP lesion detection.

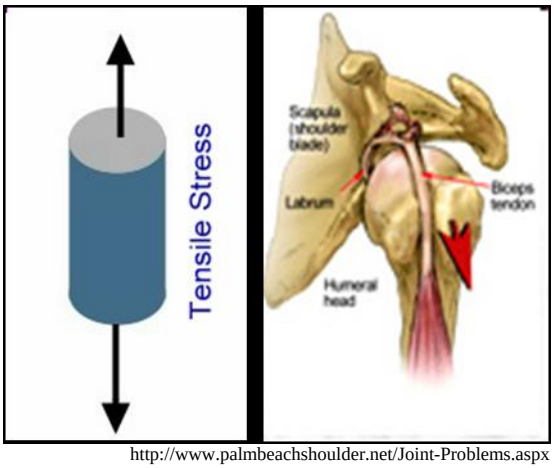
More than 20 provocative tests for the clinical evaluation of SLAP lesions are proposed in the literature. In most cases, the evaluation of these physical exams claim to have promising accuracy for the detection of SLAP lesions by their original authors [2, 19, 24, 27, 36, 37]. However, secondary studies fail to reproduce the initial findings, typically reporting much lower values for the sensitivity and specificity of the physical examination tests [8, 11, 14, 22, 28, 29]. The discrepancies between the findings of these studies most likely reflect two primary difficulties;

1) The clinical detection of SLAP lesions is hindered by the fact that SLAP lesions are rarely isolated; meaning they are frequently accompanied by other various glenohumeral pathologies which are potential sources for labral symptoms [3].
2) Differences in study protocols and problems associated with the methods used to verify accuracy of the design of these tests make comparisons between studies and verification of SLAP lesion tests difficult [8].

The bulk of the literature determines SLAP lesion test diagnostic accuracy utilizing a single verification method. Typically a patient with a suspected SLAP lesion performs the provocative tests of interest in a clinical setting before shoulder arthroscopy. The outcome of the SLAP lesion tests from the clinical evaluation is then verified with conclusive arthroscopic findings [7-9, 11, 14, 22, 23, 29]. The results of these comparative studies have significant quantitative discrepancies, but a fundamental qualitative conclusion recurs; no single SLAP lesion test has the sensitivity or specificity to independently determine the presence or absence of a SLAP lesion[7-9, 28]. Although previous studies attempt to assess the diagnostic accuracy of SLAP lesion tests, the analyses do little to

explain the reasons behind their apparent failure and rarely suggest or point to any means of improving the performance of the tests.

Although, clinically based evaluations of SLAP lesion tests account for the majority of studies to date, studies have also assessed test accuracy by attempting to validate the fundamental design behind various tests. Provocative SLAP lesion tests, by definition, function to provoke labral symptoms (primarily in the form of shoulder pain) as a positive diagnostic sign, by reenacting one of two injury mechanisms.



http://www.palmbeachshoulder.net/Joint-Problems.aspx

Figure 2: SLAP Lesion Injury Mechanism I

The first mechanism (*Figure 2*) elicits active tension in the biceps tendon and is typically associated with an acute traction trauma to the arm or elicited from repetitive overhead throwing injuries. The tensile load produced in the biceps tendon can pull and damage the superior labrum, the functional link between the insertion of the biceps tendon and the glenoid rim. The second injury mechanism produces passive compression of the humeral head and is often associated with a fall to outstretched arms (*Figure 3*).

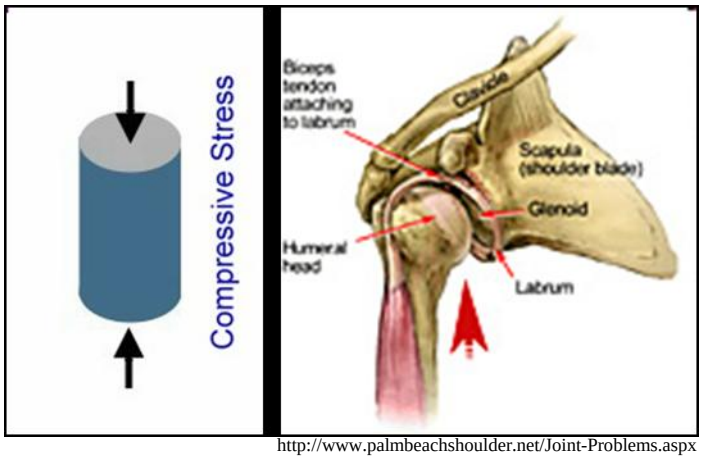


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Figure 3: SLAP Lesion Injury Mechanism II

The compressive load causes superior humeral head translation within the glenohumeral joint and can result in a collision between the humerus and labrum, potentially damaging the soft tissue of the labrum[1] The ability of proposed tests to reenact the injury mechanisms that they were designed to replicate has been examined from a few different perspectives including anatomic[10, 17, 30] and electromyographic[10, 34] methods. The results of these studies illuminate the importance of design validation during the development of clinical testing procedures.

The results of these studies do not clearly define the most accurate test for SLAP lesion diagnosis. Therefore the purpose of this study was to use electromyography (EMG) to biomechanically assess the ability of seven provocative tests to create active tension in the biceps tendon by activating the long head of the biceps brachii (LHBB). The SLAP lesion tests in this study were expected to successfully elicit LHBB activity because they were designed by their original author to reproduce a SLAP lesion injury mechanism in that manner; hence this study was a means of verifying the design of SLAP lesion tests that are meant to reproduce the first SLAP lesion injury mechanism. Furthermore, this study also examined six other glenohumeral joint stabilizing muscles,

the short head of the biceps brachii (SHBB), anterior deltoid (DELT), pectoralis major (PECT), latissimus dorsi (LAT), infraspinatus (INFRA), and supraspinatus (SUPRA), with EMG to determine how effectively each test isolated the activation of the LHBB and to characterize the behavior of each of the glenohumeral joint stabilizing muscles. Selectively activating the LHBB should reduce confounding implications of labral symptoms elicited from a source other than a SLAP lesion. Also, slight modifications were made to each of the original authors' depiction of the tests to allow the use of the Biodex System II Dynamometer to improve the uniformity between each subject's anatomical orientation and performance for each test. Additionally, the Biodex System aided the attempt to control and limit differences that may have resulted from inconsistencies in the magnitude of load applied for each test and the impact of the variability in subject's strength and their respective ability to resist the applied load.

The primary objective of this study was to evaluate seven SLAP lesion tests, chosen based on the findings of a pilot study, in subjects with no history of shoulder pathology, using EMG according to two variables, muscle activation and muscle selectivity, to characterize particular aspects of LHBB behavior. Additionally, differences in the LHBB behavior between male and female gender groups were examined. Furthermore, a brief supplementary analysis characterized the behavior of the six other joint stabilizing muscles in the same manor as for the LHBB.

The hypotheses of this study was that there would be no difference between the seven SLAP lesion tests in the LHBB behavior for either variable, no crucial behavior differences were anticipated between tests for the other six glenohumeral joint stabilizing

muscles, and furthermore differences between gender groups were also not anticipated. The statistical hypotheses will be as follows:

$H_O : \mu_1 = \mu_2 = ... = \mu_7$ (All SLAP lesion tests elicit the same value in)

- LHBB activation
- LHBB selectivity
- Gender groups

$H_A : \mu_1 = \mu_2 = ... = \mu_7$ (All SLAP lesion tests elicit a different value in)

- LHBB activation
- LHBB selectivity
- Gender groups

Operational Definitions

This study involves two dependent variables that characterize a specific behavior of the individual muscles. The principal interest for SLAP lesion detection was the LHBB behavior, but regardless of the muscle analyzed, for each respective muscle the variables were quantified by the same method for each of the seven respective muscles.

- *LHBB activation*: the maximum LHBB activity elicited
    - as indicated by the peak LHBB amplitude of the normalized EMG signal recorded during each respective provocative tests, units of percent maximum contraction, range ideally between zero and 100%
- *LHBB Selectivity*: the ratio depicting the ability of a respective provocative test to selectively activate the LHBB

- as indicated by the ratio of the LHBB activation defined above, over the sum of all seven glenohumeral muscles respective activations, defined by the same method above but with respect to each muscle, the normalized maximum peak EMG signal amplitudes elicited during a respective provocative test, unitless due to the ratio of units of percent maximum contraction over units of percent maximum contraction, range between zero and one

Limitations

The results of this study are limited to individuals who are represented by the sample population: healthy males and females who have no history of shoulder pathology. The findings in this study are only representative of individuals falling within the subject parameters noted above. It should be noted that patients with a suspected SLAP lesion may have considerable differences in muscle behavior than those who have had no history of shoulder pathology, like the cohort in this study.

Delimitations

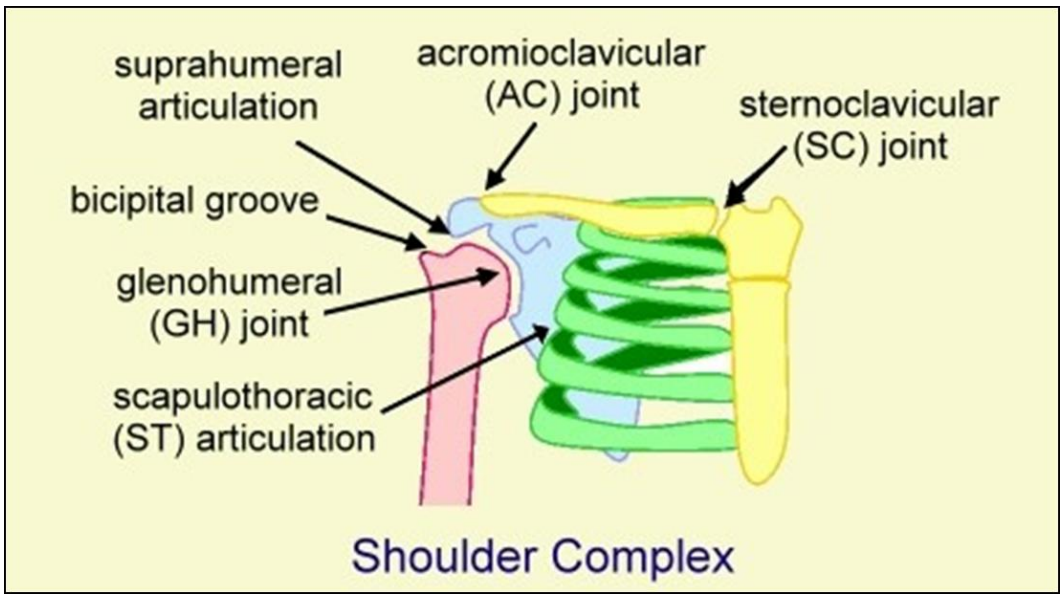
The results of this study are applicable to all physicians and clinicians using any of the seven tests because the focus is simply to verify the tests' design by assessing the ability of each test to reproduce a specific SLAP lesion injury mechanism. In elaboration, this study allows the verification of the test design, which is limited in other studies with subjects who have a shoulder injury as a successful test. The findings of such studies may not accurately represent the fundamental ability of the test to reproduce the injury mechanism.

LITERATURE REVIEW

This chapter summarizes the findings of relevant journal articles that have been published regarding SLAP lesions to date. Topics include shoulder and labral anatomy, the history of SLAP lesions, and the literature containing evaluations of the seven provocative SLAP lesion tests of interest for the present study described in this thesis

Shoulder Anatomy

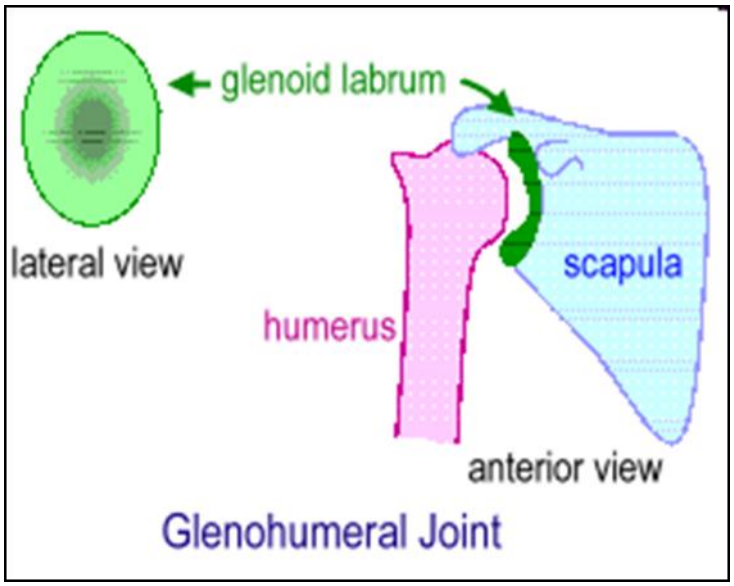
The shoulder complex (*Figure 4*) is an intricate system containing four bones (clavicle, humerus, thorax, and scapula), three anatomical articulations (acromioclavicular, sternoclavicular, and glenohumeral), and one functional articulation (scapulothoracic), which is supported by ligamentous structures, soft tissues, and the musculature surrounding of the shoulder girdle.



http://www.pt.ntu.edu.tw/hmchai/Kinesiology/KINupper/Shoulder.files/ShoulderStructure.htm

Figure 4: Shoulder Complex - Bony Anatomy

The interaction of these components produces the most dynamic and mobile joint complex in the body. The stability of this uniquely mobile joint, and specifically the glenohumeral ball and socket joint (*Figure 5*), is maintained by a number of static and dynamic stabilizing structures. The articular surfaces of the proximal humerus and glenoid are mismatched with regards to size and orientation, which grants the joint extreme mobility, and essentially eliminates bony stability[21].



http://www.pt.ntu.edu.tw/hmchai/Kinesiology/KINupper/Shoulder.files/ShoulderStructure.htm

Figure 5: Glenohumeral Joint Anatomy

Shoulder stability is maintained by a complex web of contributors including the static soft tissue structures of the joint itself and the musculature surrounding the shoulder girdle. A recent publication by Veeger and colleagues on shoulder biomechanics articulately noted that shoulder function is the 'perfect compromise between stability and mobility'[1]. Clearly, the stability of the shoulder can be easily compromised due to the number of components and the complexity of their interactions. Glenoid labral pathologies can hinder the careful balance required by this unique biomechanical system. Glenoid labral musculature, surrounding connective tissues, and negative intra-articular

pressure are all proposed constituents involved in maintaining the stable position of the humeral head within the glenoid. The labrum itself plays a valuable role in joint stability. The glenoid labrum creates a suction effect on the humeral head and it increases the depth of the glenoid cavity by fifty percent. Hence, the presence of glenoid labral pathologies inherently affects stability of the glenohumeral joint.

SLAP Lesions

In the last century, the development of medical imaging techniques has radically expanded the understanding of human anatomy. Radiography began with the discovery of the x-ray in the late nineteenth century, and its diagnostic value was quickly realized. Today the term radiography encompasses the range of imaging modalities not limited to computed tomography (CT) and magnetic resonance imaging (MRI). Both CT and MRI create three dimensional reconstructed images, which improve on the flat, two dimensional nature of the x-ray. MRI has far greater contrast than CT, which enables various soft tissues such as muscles and ligaments to be distinguished, greatly aiding the understanding of musculoskeletal anatomy. Magnetic resonance (MR) arthrography, where a contrast medium is injected into a joint or region of interest prior to MRI to further improve tissue differentiation, has been particularly useful for examining fine musculoskeletal pathologies including lesions of the shoulder[4]. Clearly, radiography has allowed human anatomy and physiology to be viewed in a new perspective, and specifically, advanced imaging methods have helped to illuminate the difficult form, function, and carefully balanced means of maintaining the stability of the shoulder complex.

The introduction of shoulder arthroscopy enabled the characterization of musculoskeletal pathologies that were previously unidentifiable with open surgical techniques, medical imaging modalities, or through the use of any other medical tools. In this manner, the complex musculoskeletal structure of the shoulder greatly benefited from the advent of shoulder arthroscopy. Specifically, in 1985 glenoid labral lesions were first described in throwing athletes after Andrews et al diagnosed 73 patients with the pathology after arthroscopic surgery. Andrews et al made several hypotheses based on observations during this early study, and they remain relevant today; 1) during throwing the biceps tendon undergoes large forces, 2) the most frequent location of glenoid labral tears is near the biceps tendon insertion at the anterior-posterior area of the glenoid (occurring in 83% of the glenoid labral lesion patients in the study by Andrews et al), and 3) the biceps tendon is likely the cause of glenoid labral lesion[1].

In 1990, Snyder et al coined the term superior labrum anterior-posterior (SLAP) lesion to simply identify the labral lesion first described by Andrews et al[32]. Interestingly, although almost two decades has passed since this publication, the difficulty associated with SLAP lesions detection without arthroscopy, which was noted in the publication, has not changed considerably. Snyder et al examined more than 700 shoulders with arthroscopy and found 27 SLAP lesions. SLAP lesions were further categorized into four grades of severity ranging from Type I (where fraying and a general degenerative appearance of the superior aspect of the labrum is present) to Type IV (where 'bucket handle' tears are present and often are displaced into the joint with the lesion extending into the biceps tendon). Furthermore, importance of this study is indicated by the frequency with which it is referenced in the literature. Snyder et al was

the first to acknowledge the frequent occurrence of other shoulder pathologies with SLAP lesions, and the findings supported and improved upon concepts relating to SLAP lesion injury mechanisms. The injury mechanisms were related to either a tensile load on the biceps tendon or a compressive load received on the labrum itself. The injury occurred from either traction to the arm from a sudden traumatic increase in load, from a repetitive tensile load seen in overhead throwing athletes, or from a compressive load caused from a fall to outstretched arms.

An EMG study during baseball pitching noted that peak biceps brachii muscle activity occurred following ball release, during the deceleration phase of the arm while throwing[15]. These findings supported Andrews et al's biomechanical evaluation of throwing in the 1985 manuscript, examining the elbow and shoulder moments using three dimensional high speed cinematography and computer assisted analysis. Andrews determined that during the peak acceleration phase of throwing, the elbow extends from 80° to 30° in a 25-ms period, producing a peak moment of 600 inch-pounds prior to deceleration. The hypothesis is that the burst of biceps activity at the beginning of the deceleration phase indicates that the biceps play a role in decelerating the joint and that the large mechanical moment noted may be dampened and controlled by the LHBB, supporting the possibility that the deceleration phase of throwing may be a likely cause of SLAP lesions[1].

The present findings and consensus in the literature continues to support the proposed SLAP lesion injury mechanisms suggested over twenty years ago in the first two SLAP lesion publications[1, 32], but contrarily there have been some unique case reports whose findings seem to question these mechanisms. Specifically, the role of

tension in the biceps tendon in SLAP lesions has been called into question. A case study by Keefe et al discussed the potential need to reevaluate the pathomechanics behind SLAP lesion mechanisms due a patient with a SLAP lesion (arthroscopic verification) that did not have a biceps tendon. The patient could not recall a traumatic traction event, a fall to outstretched arms, and clearly the injury could not have resulted due to tension in the biceps tendon during overhead throwing, though the subject was an active throwing athlete. Although a lack of the biceps tendon may not be common for the general population, this case study may imply that the role of the tendon in the deceleration phase of throwing may need to be reevaluated for the general population and for the tendon's part in SLAP lesions[16].

Provocative Tests

Active Compression Test

In 1988 O'brien et al first proposed the Active Compression test. It was originally intended to assess acromioclavicular (AC) joint pathologies, but after anatomic validation using cadaver studies and following testing on subjects with suspected SLAP lesions, the authors claimed that the Active Compression test was useful for the clinical detection of SLAP lesions and various AC joint pathologies. The test was originally designed based on the description of a patient with a degenerative AC joint, who described the primary movements that reproduced his symptoms of pain. The author conducted a study of 318 patients with shoulder pain and reported promising findings. The results of the clinical tests were confirmed by either arthroscopic verification or radiography, and the findings alleged that the Active Compression test had 100% sensitivity, 99% specificity, a positive predictive value of 94.6%, and a negative

predictive value of 100% [27]. These results have not been reproduced in secondary studies, and one large potential source of error for the study is that medical imaging was used to verify the presence of SLAP lesion, and radiography is known to exhibit poor accuracy for SLAP lesion detection[8]. The Active Compression Test is one of the most evaluated SLAP lesion tests in the literature. Most studies indicate it performs with some promise, but in general high accuracy is not reported.

In response to questions associated with the initial study's findings and reliability of the accuracy for the Active Compression test, McFarland et al conducted a study with 426 patients all of which underwent arthroscopic confirmation and found 47% sensitivity, 55% specificity, a positive predictive value of 10%, and a negative predictive value of 91% [22]. This study clearly does not support previous findings, and again the discrepancy could be linked to the error associated with radiographic diagnoses and their use in the study.

Similar studies report findings which parallel the results of McFarland et al, including the 2001 study by Kim et al and 2006 study of Parentis et al. Both of the studies further categorized SLAP lesions into various subtypes, to assess potential improvements in clinical test performance when severity and type of SLAP lesion where taken into account. Unfortunately in both studies the accuracy of the Active Compression test was below 65 % regardless of the type of SLAP lesion[19, 29].

Though much less in number, several other studies have attempted to evaluate the accuracy of the Active Compression SLAP lesion test utilizing different test design verification methods, and these analyses are of particularly interest to the study in this thesis. A study in 2004 assessed the anatomical basis for the test, using MRI. The

findings suggested there was an anatomical basis for the test. The study suggests that the internal rotation of the arm, required for the palm-up position of the test, causes consistent physical contact between the superior labrum and lesser tuberosity, which likely would be a source of pain for subjects with a damaged labrum. Furthermore, this contact between articular surfaces is eliminated with internal rotation of the forearm as the palm-down position requires. These findings support the anatomic validation of the Active Compression Test because the test is considered positive for a SLAP lesion only when the patient has labral symptoms during the palm-up portion of the test that are not present during the palm-down portion of the test[30].

In 2006 another study proposed that Type II SLAP lesions may be best detected with the Active Compression tests and EMG was used to determine which clinical tests in the study elicited the most promising muscle behavior. The study found that strong LHBB activity peak was elicited during the Active Compression test, indicating that the test may be a better diagnostic tool than other tests in that study[34].

In 2008 a study also attempted to anatomically validate the Active Compression test using two methods. First, the objective was to quantify the active tension in the biceps tendon using EMG and twelve healthy subjects. Second, the objective was to quantify the passive tension in the tendon in five cadaver shoulders, using a custom designed load cell to determine strain on the biceps tendon. In contrast to Parentis et al, this study found that the anatomic basis of the Active Compression test was not valid[10].

Although the Active Compression Test is examined frequently in the literature, the findings of these studies are limited, and this is a pattern that is repeated for the remaining six tests examined in this study. Majority of the studies assess this test, and all

SLAP lesion tests, by a single comparative method. The clinical findings of the test are compared with the concrete arthroscopic diagnoses, and such a comparative analysis is has considerable limitations. These comparative studies provide no indication as to why the test performed successfully or otherwise and furthermore, the tests provide no information or means of improving upon test performance. Studies that examine tests using other methods such as with EMG and cadaver specimens, improve upon the comparative analysis, in that they provide information that potentially explains reasons for their performance. In the case of the Active Compression test, biomechanical analyses were seen in two studies, using EMG and cadaver, but more studies in this manner would benefit SLAP lesion tests.

Speed's Test

In 1998, Speed's Test was introduced to assess a variety of shoulder pathologies, and this study is frequently used today in the clinical setting. Bennett et al assessed 46 shoulders in 45 patients with arthroscopic confirmation, and determined that Speed's had a promising sensitivity of 90%, but found the test performed poorly for other accuracy measures with 14% specificity, a 23% positive predictive value, and a 83% negative predictive value[2]. Another study countered these findings, eliciting low sensitivity results for Speed's tests at 32%, 75% specificity, a 50% positive predictive value, and a 58% negative predictive value. This study concluded that Speed's was moderately specific, but the test was unlikely to influence the pretest diagnosis held by the clinician. The authors reiterated the fallibility of clinical assessments, because depending on the setting and population, they argue that predictive values vary inherently[14].

In 2007, another study examined the accuracy of Speed's to detect partial tears in the biceps tendon in 847 consecutive patients who underwent arthroscopy, and 40 of those had confirmed SLAP lesions. In this study, Speed's tests had a sensitivity of 50%, specificity of 67%, a positive predictive value of 8%, and negative predictive value of 96%. The frequent occurrence of other shoulder pathologies was attributed to the reason behind the poor behavior of Speed's and the anticipated unreliability of any clinical exam to detect partial tears of the biceps tendon. Of the 847 patients, 40 had partial bicep tendon tears, 34 had partial rotator cuff tears, three had anterior instability, two had impingement without rotator cuff tear, and 1 had degenerative arthritis. The study concluded that no single physical examination test can accurately predict the presence of a partial tear in the biceps tendon. The study also suggested that tests designed to produce tension in biceps tendon are not helpful in detecting partial tears of the bicep tendon[9]. Again the lack of valuable information that can be derived from these types of comparative studies must be reiterated, and Speed's has not been studied biomechanically or anatomically to date.

Pronated Load Test

The performance of the Pronated Load test has not been evaluated beyond the mention of promising sensitivity by Wilk et al in 2005. The test was designed to simulate the injury mechanism and peel back behavior seen during stimulation. The Pronated Load test is meant to have the promising behavior of the Pain Provocation Test which causes passive external rotation of the forearm, coupled with a position that enables large activity from the LHBB during contraction[36]. No biomechanical studies are presently available on the Pronated Load test.

Bicep Load I Test

In 1999, a new SLAP lesion test was proposed; the Bicep Load I Test. This provocative test was designed to detect SLAP lesions in subjects who have recurrent anterior shoulder dislocation, typically found in SLAP lesion Type II subjects. The original and only study evaluating this test was a cohort study, and evaluated 75 patients who all underwent arthroscopic surgery. The Bicep Load I test indicated that 12 subjects had SLAP lesions, and 10 of these were arthroscopically confirmed as Type II SLAP lesions. The resulting test sensitivity was 90.9%, specificity was 96.9%, positive predictive value was 83.0% and negative predictive value was 98.0%[20]. Although, the original findings are promising, the test is designed for SLAP lesion detection only in shoulders with recurrent dislocation and may not be as reliable for those patients without the additional shoulder pathology. Furthermore, no biomechanical or anatomic studies have evaluated this test.

Bicep Load II Test

In 2001, another SLAP lesion test was proposed, Bicep Load II test, as a complement to Bicep I. The Bicep Load II test was designed with the intent to detect isolated SLAP lesions. 127 subjects were evaluated in the study, 38 were positive for a SLAP lesion according to the Bicep Load II test, and 35 were confirmed to have SLAP lesions following arthroscopy. Again, promising accuracy was reported with 89.7% sensitivity, 96.6% specificity, 92.1% positive predictive value, and 95.5% negative predictive value[19]. These findings may be limited to isolated SLAP lesions, which is inherently uncommon.

In 2008, first study evaluating the ability of a combination of more than one provocative tests to detect Type II SLAP lesions was published. Several tests, including the Bicep Load II test, were included in this study, and interestingly the Bicep Load II test was categorized as a high performing test with respect to specificity. This study found that the combined findings from two relatively sensitive and one relatively specific test improved SLAP lesion detection accuracy dramatically, such that sensitivity was a minimum of 70% when one of the three tests were positive and specificity was a minimum of 90% when all three SLAP lesion tests were positive. Furthermore, in this study, the author explicitly stated that no single SLAP lesion test would have the capability, with regards to simultaneous strength in sensitivity and specificity, to be individually able to detect a Type II SLAP lesion[28]. Bicep Load II is another SLAP lesion test that has not been evaluated by means other than comparative assessment.

Resisted Supination External Rotation Test

In 2005, the Resisted Supination External Rotation was developed to mimic the peel-back mechanism associated with SLAP lesions. The study examined 40 athletes, of which 29 had SLAP lesions verified by arthroscopy. The results from the Resisted Supination External Rotation test were compared to those of the Crank test and the Active Compression test. Meyers et al claimed the Resisted Supination External Rotation test has better performance than both of the others, with a sensitivity of 82.8%, a specificity of 81.8%, a positive predictive value of 92.3%, and a negative predictive value of 64.3%[24]. Further evaluation of this test is needed, as the only study evaluating the test is this original study, and no biomechanical studies have been published to date.

Supination Sign Test

The Supination Sign test another provocative test designed to elicit labral symptoms as a means of SLAP lesion detection that is anatomically based. The Supination Sign Test was shown to have specificity and low sensitivity across four studies including Nakagawa et al, Guanche et al, Holtby et al, and Parentis et al resulting in comparable finding for sensitivity (14%, 12%, 43%, and 13% respectively for each study) and similarly for specificity (98%, 96%, 79%, and 93%). Although in general the Supination Sign test has a high specificity, high specificity is likely not a good method for stand alone evaluation of the presence of a SLAP lesion[11 14 25 29]. Once again, no biomechanical assessment has been done.

In summary, no study has biomechanically assessed the accuracy and performance of these tests. Although many studies have attempted to determine test accuracy by comparative analysis and some studies have examined a single test anatomically or with EMG, further biomechanical assessment is necessary to properly evaluate the ability of these tests to aid in the detection of SLAP lesions in the clinical setting. A biomechanical evaluation of these tests, will not only help to verify the design of these test and provide an alternative method to quantify test accuracy, but biomechanical assessment could also provide valuable information as to how these tests may be improved.

METHODS

This chapter addresses the methodology and procedures that were used to acquire the data necessary to fulfill the purpose of this study. Topics that will be addressed in this chapter include the experimental protocol for this study, the original descriptions of each provocative SLAP lesion test and the modifications used in this study, the methods used to filter and analyze the EMG data, including the definitions and mathematical equations for muscle activation and muscle selectivity, and the statistical methods used to determine the significance of the data.

Experimental Protocol

Subject and IRB approval

A cohort of 21 healthy volunteers comprised of 11 females (24.7 ± 6.7 years, 168.4 ± 5.3cm, 66.9 ± 9.1kg) and 10 males (29.4 ± 10.6 years, 178.1 ± 6.6 cm, 80.0 ± 6.4kg) with right arm dominance and no history of shoulder pathology were recruited for subjects in this study. Subjects recruited were either college students at Boise State University or medical health professionals from the local area. All procedures were approved by the Institutional Review Board at Boise State University, and all participants read and signed a statement of informed consent prior to the start of testing.

Electromyography Apparatus and Subject Preparation

EMG was used to record muscle activity for seven muscles surrounding the dominant arm's glenohumeral joint including the long head of the biceps brachii (LHBB), short head of biceps brachii (SHBB), anterior deltoid (DELT), pectoralis major (PECT),

latissimus dorsi (LAT), infraspinatus (INFRA), and suprasinatus (SUPRA). Each subject was instrumented with a single 44-gauge fine-wire indwelling electrode and six surface bipolar silver-silver chloride EMG electrodes (Noraxon, USA Inc, Scottsdale, AZ). The surface electrodes were positioned over the muscle belly and parallel with the orientation of the muscle fibers, as seen below where a) LHBB and SHBB, b) DELT, c) PECT, d) LAT, e) INFRA, and f) SUPRA (*Figure 6*)[6].

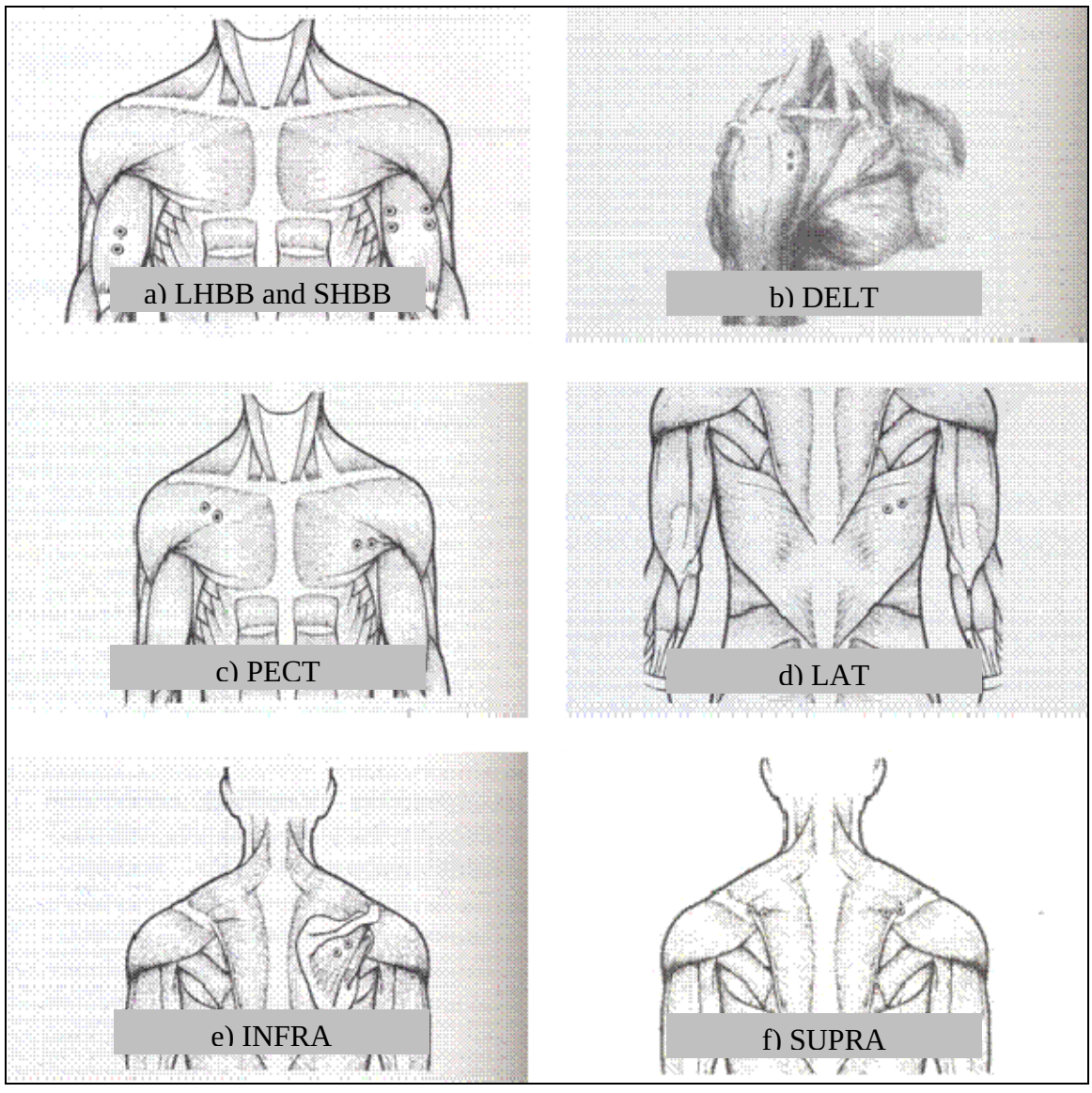


Figure 6: Electrode Placement for EMG (a – f)

An additional surface electrode was placed on the acromion process of the non-dominant shoulder to serve as a reference. Due to the location of the SUPRA deep to the trapezius, the indwelling electrode was necessary to acquire SUPRA activity. Using sterile techniques a certified medical technician placed the fine-wire indwelling electrode using a 27-guage sterile needle. EMG data were recorded using the Vicon Nexus Software (Vicon, Los Angelos, CA) at 1250 Hz using a Noraxon Telemyo 900 EMG system (Noraxon USA, Inc, Scottsdale, AZ).

Subject Protocol

Using established EMG protocols, each subject was asked to perform Maximum Voluntary Isometric contractions (MVICs) for each of the seven muscles in random order on a Biodex System II Dynamometer (Biodex Medical Systems, Shirly, NY). Modifications were made to the MVIC recommendations of Cram, Hintermeister, and Rowlands[6, 12, 31] to accommodate for the use of the Biodex System (*Table 1*).

Muscle	Joint Position	Resisted Maneuver
DELT	Arm at side	Shoulder flexion
LHBB / SHBB	Elbow flexed 90º, shoulder flexed 90º	Elbow flexion
INFRA	Arm abducted 45º, elbow flexed 90º	External Rotation
LAT	Shoulder flexed 90º, arm internally rotated	Shoulder extension
PECT	Arm abducted 90º, forearm supinated	Horizontal adduction
SUPRA	Arm abducted 90º, forward flexed 30º, and internally rotated	Maintain against resistance

Table 1. MVIC Joint Positions and Resisted Maneuvers

For each trial six seconds of data were recorded to ensure that the entire burst of muscle activity was captured during each MVIC. The subjects performed three trials for each MVIC and were asked to maximally contract for a count of three seconds. Subjects rested for thirty seconds between MVIC trials to avoid fatigue effects. For each MVIC, the peak amplitude of the EMG signal among the three trials was used to normalize the provocative test data to a percentage of effort.

Similarly seven provocative tests were performed in random order based on the descriptions of the original authors but with modifications to accommodate for use of the Biodex System. These tests were chosen based on the findings of a preliminary pilot study that evaluated clinical tests from relevant literature that were designed to reenact SLAP lesion injury mechanisms. Again, each subject performed three trials for a three second count for each test, six seconds of data were recorded for each trial, and the subjects rested for thirty seconds between trials to avoid fatigue affects.

Once all MVIC and SLAP lesion test trials had been completed the surface electrodes were removed from the subject, and a trained medical technician, using sterile techniques, removed the indwelling electrode from the SUPRA by applying gentle and steady traction to the leads. A sterile bandage and pressure were applied to the location where the indwelling electrode was removed. Each subject was advised to seek medical attention if an infection appeared to develop at the site, although infection was not anticipated.

Test Descriptions and Modifications

Each of the seven SLAP lesion tests performed in this study were provocative tests that were designed by their respective original authors to reproduce one SLAP

lesion injury mechanism, by activating the LHBB to induce tension in the biceps tendon. For the purposes of this study, modifications were possible and made for six of the seven SLAP lesion tests utilizing the Biodex System II Dynamometer to improve standardization between subjects and hopefully to reduce the potential for differences in muscle behavior or test performance due to variable subject and clinician strengths that could alter the intended test position or function. Tests requiring static resistance against an applied load maintained the static subject position through the stationary preset up of the Biodex System in isometric mode. Tests requiring dynamic resistance to an applied load were controlled by the Biodex System allowing a motion with a constant velocity regardless of the force applied by the subject through the isokinetic mode of the Biodex System. The original description of each test and the modifications employed in this study are noted below.

Active Compression Test (ACPU and ACPD)

The Active Compression Test has two positions, palm-down (ACPD) and palm-up (ACPU), which vary only by internal or external rotation of the arm. The patient is standing with the elbow in full extension, the shoulder is flexed to 90°, and adducted $10-15^{\circ}$ medial to the sagittal plane. For ACPD (*Figure 7*) the arm is maximally internally rotated such that the thumb points down. The patient is asked to resist a uniform downward load applied to their arm by the clinician. For ACPU (*Figure 8*) the initial patient positioning is unchanged except the arm is externally rotated such that the palm faces up. Again, the patient is asked to resist a uniform downward load applied by the clinician[27].

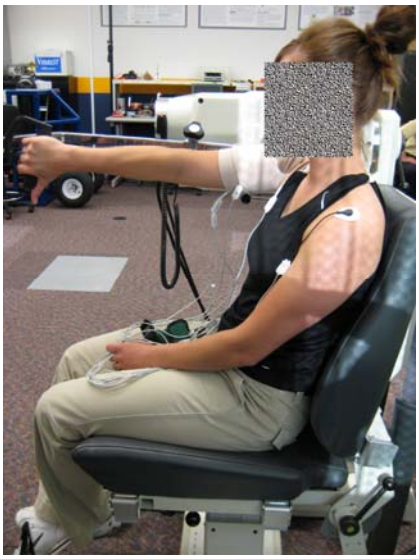


Figure 7: ACPU

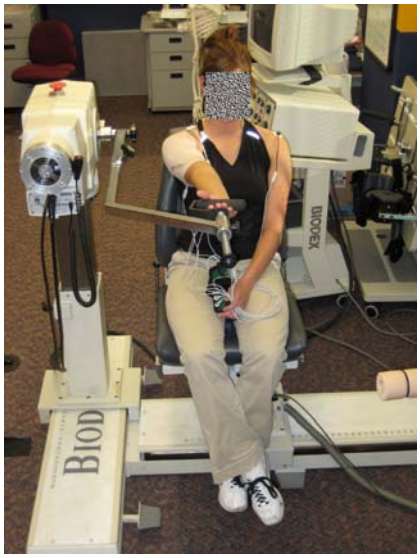


Figure 8: ACPD

For the purpose of analysis and due to the nature of the subject population, a control group having no history of shoulder pathology, this study treated ACPU and ACPD as two independent tests. Both tests were modified such that the subject was seated in the Biodex System. The orientation of the subject's arm remained true to original description of the two test positions by O'Brien et al, but the subject was

asked to resist the stationary set-up of the arm of the Biodex System by attempting to lift his/her arm superiorly for both ACPD and ACPU.

Speed's Test

According to the original description of Speed's test (Speed), the patient is standing and resists a downward force applied to the upper extremity with the elbow extended, forearm supinated, and arm elevated to 90° [2]. In this study the orientation of the subject's arm remained similar to the original definition, but the test was modified into a dynamic movement controlled by the Biodex System. The subject's arm started hanging beside and parallel to the body with the palm facing up (*figure 9*), and then the subject was asked to raise the arm (flex the shoulder) with as much force as possible to 90°. Regardless of the force exerted by the subject, motion was restricted to a constant velocity by the Biodex System in the isokinetic setting of 60° per second.

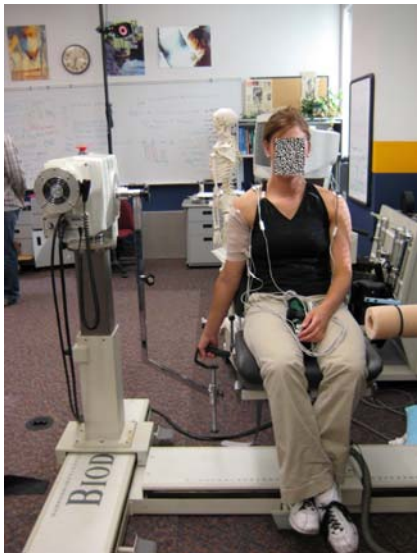


Figure 9: Speeds Starting Position

Pronated Load Test

In the original description, the patient is in the seated position with elbow flexed to 90°, the arm abducted to 90°, maximally externally rotated, and the forearm is fully pronated. The patient is then asked to perform an isometric contraction of the biceps (a 'curl' with the forearm pronated)[36]. The Pronated Load test (ProLoad) was negligibly modified for this study. The subject sat in the original orientation in the Biodex System, the subject's arm was supported just proximal to the elbow. The subject was asked to perform an isometric bicep contraction (pronated curl) which was resisted by the static set up of the Biodex System.

Bicep Load I Test

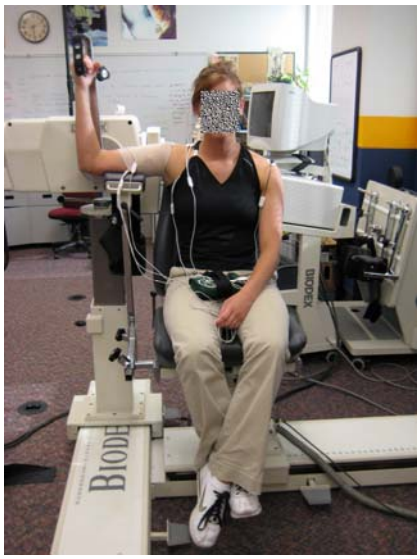


Figure 10: Bicep Load I

According to the original author's definition of the Bicep Load I test (Bicep I), the patient is in the supine position when an anterior apprehension test is performed starting with the arm abducted 90° with the forearm fully supinated[20]. Bicep I was modified such that the patient was seated in the Biodex System, in the same position

as ProLoad, except the forearm was fully supinated (*Figure 10*). The subject was asked to perform a bicep contraction (a traditional 'curl'), which was resisted by the static set up by the Biodex System.

Bicep Load II Test

The patient is supine with the arm abducted to 120° degrees, the elbow flexed to 90° degrees, and the forearm fully supinated.

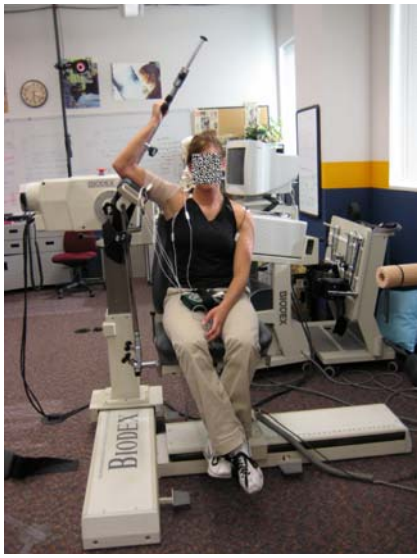


Figure 11: Bicep Load II

The patient is then asked to flex the elbow against the resistance of the clinician[19]. For this study the modification for Bicep Load II test (Bicep II) paralleled those made to Bicep I except the arm was abducted to 120° degrees instead of 90° (*Figure 11*).

Resisted Supination External Rotation Test

The original authors describe putting the patient in the supine position with scapula near the edge of an evaluation table; the patient's arm is supported by the physician at the wrist, with the arm abducted to 90° and the elbow flexed between 65° and 70° degrees. The clinician then externally rotates the arm while the patient is

asked to supinate the forearm[24]. The Resisted Supination External Rotation test test was essentially unchanged for this study (*Figure 12*), and the movement was not controlled by the Biodex System because the position and motion could not be recreated with the Biodex System for all subjects. One certified athletic trainer performed RSER with each subject in the study in the supine position on the Biodex System.

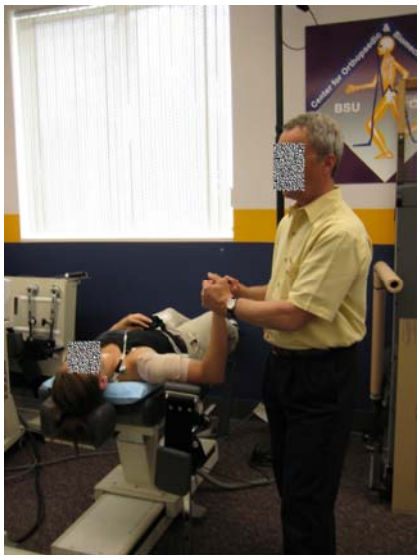


Figure 12: RSER Starting Position

Supination Sign Test

As originally defined, the Supination Sign test (Yergason), in the seated position with the elbow flexed to 90° and forearm fully pronated, the patient is asked to attempt to supinate the forearm while the physician resists the attempted motion while holding the wrist[37]. Yergason was scarcely modified in this study; as the patient maintained the defined orientation but the forearm was fastened to the static arm of the Biodex System (*Figure 13*). The subject was asked to attempt to supinate the forearm against the static setup of the Biodex System.

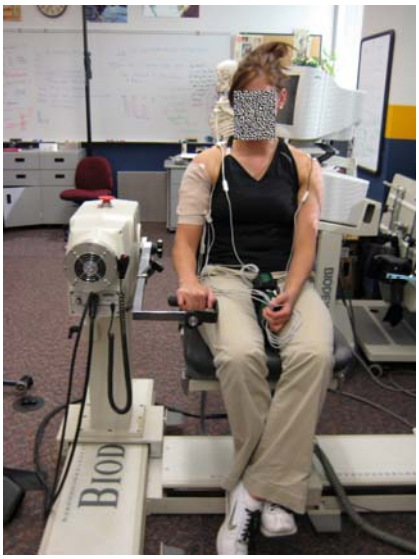


Figure 13: Yergason's

Electromyography Analysis

The raw EMG signals were filtered, normalized, and then analyzed to characterize muscle behavior using custom MATLAB software. The raw EMG signals were processed a traditional EMG filtering technique that is frequently employed with EMG and noted in the literature. After processing the EMG signals, numerical values were calculated for the LHBB activation and LHBB selectivity as a means of characterizing LHBB behavior. Supplementary calculations were made for the muscle activations and muscle selectivities of the remaining six glenohumeral muscles in this study.

Initial EMG Processing

During all testing, EMG data from each muscle were acquired at 1250 Hz, and the raw data was band-pass filtered from 16 to 500 Hz by the data collection unit internally prior to transmission of the data to the wireless receiver for further processing. Next, custom MATLAB software was used to further process, normalize, and analyze the EMG signals.

EMG Filtering Technique

The majority of EMG signals that were collected, the LHBB, SHBB, DELT, PECT, and SUPRA, were filtered by a traditional smoothing technique and band-pass filtered from 20 to 500 Hz, and then the signals were rectified and smoothed using a root mean square algorithm in combination with a 20-ms forward moving window average.

Normalizing Provocative Test Data with MVIC Maximums

The provocative test EMG signals were normalized to a percentage of effort based on the peak EMG amplitude elicited during each muscle's respective Maximum Voluntary Isometric Contraction (MVIC). Ideally the normalized and filtered EMG signals for each provocative test would have a muscle activation range of zero to 100% MVIC. Muscle activation and muscle selectivity were calculated for each muscle during each test.

Muscle Activation

Muscle activation was used to determine how effective each SLAP lesion test was at causing the individual muscles to activate and was defined as the peak muscle activities elicited during the three normalized trials.

Muscle Selectivity

The ability of each provocative test to isolate the LHBB is important for diagnosing SLAP lesions due to the common association of SLAP lesions with other shoulder pathologies. Therefore in this study, a ratio depicting the ability of each test to selectively activate each muscle was calculated. The muscle selectivity for each test was defined as the ratio of the peak activation of the muscle of interest over the sum of peak activations for all seven muscles examined in the study. For example, a selectivity ratio

of 1.0 for any muscle, N, would indicate that muscle N was the only active muscle contributing to the EMG signal while the other six muscles remained inactive. For each test the selectivity for muscle N was calculated using the following equation.

$$N\_SelectivityRatio = \frac{A_N}{\sum (A_{LHBB} + A_{SHBB} + A_{DELT} + A_{PECT} + A_{LAT} + A_{INFRA} + A_{SUPRA})}$$

- where N is the muscle of interest (LHBB, SHBB, DELT, PECT, LAT, INFRA, or SUPRA)
- where $N\_SelectivityRatio$ is the selectivity ratio of muscle N
- where A_N is the peak muscle activation for muscle N

Statistical Analysis

Statistical analysis was conducted using SPSS Statistics Software 17.0 for Windows to determine significance of the data, specifically, differences in maximum muscle activations and muscle selectivities for each test, between tests, and between male and female groups. A repeated measures analysis of variance (ANOVA) test was performed to identify significant differences between provocative tests for each individual muscle. A pair-wise T-test post-hoc analysis was performed to compare results between each test using a p-value sliding scale Bonferroni adjustment[13]. Likewise, a paired-sample T-test was used to examine potential differences in muscle activation ($p = 0.05$) between male and female groups.

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APPENDIX

Manuscript for Submission to the Journal of Shoulder and Elbow Surgery

Abstracts for this study have been submitted and accepted for presentation at two annual conferences. First, the study will be presented as a podium at the 2009 Northwest Biomechanics Symposium (NWBS) at Washington State University, June 5th and 6th in Pullman, WA. Also, the study will be presented at the 2009 Annual Meeting of the American Society of Biomechanics (ASB) at Pennsylvania State University, August 26th through 29th in State College, PA. This manuscript has not been submitted for journal publication; furthermore no portion of the data, methods, results, or findings from this work have been previously submitted, published, or printed with the exception of the abstracts for the conference submissions noted above.

This manuscript has been read and approved by all authors, and each author believes the manuscript to be honest work. Dr. Michelle B. Sabick should be noted as the corresponding author for future inquiry with regards to this manuscript; her contact information is as follows:

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The study was approved by the Institutional Review Board at Boise State University, approval number 130.05.018, and each subject signed a form of informed consent prior to their participation in the study.

Title Page

GLENOHUMERAL MUSCLE ACTIVATION DURING PROVOCATIVE TESTS DESIGNED TO DIAGNOSE SUPERIOR LABRUM ANTERIOR-POSTERIOR LESIONS

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Abstract

BACKGROUND

Despite considerable medical advances, arthroscopy remains the only definitive means of Superior Labrum Anterior-Posterior (SLAP) lesion diagnosis. Natural shoulder anatomic variants limit the reliability of radiographic findings and clinical evaluations are not consistent. Accurate clinical diagnostic techniques would be advantageous due to the invasiveness, patient risk, and financial cost associated with arthroscopy. The purpose of this study was to examine the behavior of the joint stabilizing muscles in promising provocative tests for SLAP lesions. Electromyography was used to characterize the muscle behavior, with particular interest in the long head biceps brachii, as activation of the long head and subsequent tension in the biceps tendon should elicit labral symptoms in SLAP lesion patients.

METHODS

Volunteers (N=21) without a history of shoulder pathology was recruited for this study. The tests analyzed were Active Compression, Speed's, Pronated Load, Biceps I, Biceps II, Resisted Supination External Rotation, and Supination Sign. Tests were performed on a dynamometer to improve reproducibility. Muscle activity was recorded for the long and short heads of the biceps brachii, anterior deltoid, pectoralis major, latissimus dorsi, infraspinatus, and supraspinatus. Muscle behavior for each test was characterized by peak activation and selectivity.

RESULTS

Speed's, Active Compression Palm-Up, Bicep I and Bicep II, produced higher long head activations. Resisted Supination External Rotation, Bicep I, Bicep II, and Yergason's, produced higher long head selectivities.

CONCLUSION

Bicep I, and Bicep II elicited promising long head behavior (high activation and selectivity). Speed's and ACPU elicited large long head activity, and Resisted Supination and Yergason's elicited selective long head activity. These top performing tests utilize a unique range of test variables that may prove valuable for optimal SLAP test design and performance.

LEVEL OF EVIDENCE: Diagnostic Study Level I

KEY WORDS: SLAP, Superior Labrum Anterior Posterior Lesion, provocative test, long head biceps brachii, diagnoses

Introduction.

The advent of shoulder arthroscopy as a medical tool led to the description of glenoid labral tears in 1985 [1], and superior labrum anterior-posterior tears were coined 'SLAP lesions' in 1990 [30]. Although it has been almost two decades since SLAP lesions were defined, diagnosis remains difficult [17, 21, 31] despite considerable advances in the understanding of glenohumeral biomechanics and glenoid labral pathologies. In spite of these advances, arthroscopy remains the only definitive means of SLAP lesion detection [4, 8]. Accurate clinical diagnostic techniques, as an alternative to shoulder arthroscopy, would be advantageous due to the invasiveness, financial cost, and patient risk associated with arthroscopy.

Radiography and physical examination have proven useful for assessing a wide variety of orthopedic injury, but have shown limited potential with regards to SLAP lesion detection. Though radiography, particularly MR arthrography in high contrast, has shown some promise as a supplementary tool in SLAP lesion diagnosis, natural anatomic variants limit the reliability of all radiographic diagnoses. Furthermore, parallel to arthroscopy, radiography can be invasive, expensive, and dangerous, causing life threatening allergic reactions [5] in some patients, rendering radiography an imperfect means of SLAP lesion detection [3, 4, 7].

More than 20 provocative tests for the clinical evaluation of SLAP lesions are proposed in the literature. In most cases, the evaluation of the physical exams by the original authors reveals promising accuracy for the detection of SLAP lesions [2, 18, 19, 23, 24, 33, 34]. However, secondary studies often fail to reproduce the initial findings, typically

reporting much lower values for the sensitivity and specificity of the physical examination tests [8, 12, 15, 21, 25, 26]. The discrepancies between the findings most likely reflect two primary difficulties: 1) the clinical detection of SLAP lesions is hindered by the fact that SLAP lesions are rarely isolated; meaning they are frequently accompanied by other various glenohumeral pathologies which are potential sources for labral symptoms [3], and 2) differences in study protocols and problems associated with the methods used to verify accuracy of the physical examinations make comparisons between studies difficult [8].

The bulk of the literature assesses SLAP lesion tests by determining diagnostic accuracy through a single verification method. Typically a patient with a suspected SLAP lesion performs the provocative tests of interest in a clinical setting before shoulder arthroscopy. The outcome of the SLAP lesion test is then verified with conclusive arthroscopic findings [7-9, 12, 15, 21, 22, 26]. The results of these comparative studies have significant quantitative discrepancies, but a fundamental qualitative conclusion recurs; no single SLAP lesion test has the sensitivity or specificity to independently determine the presence or absence of a SLAP lesion [7-9, 25]. Although previous studies assess the diagnostic accuracy of specific SLAP lesion tests, they do little to explain the reasons behind their apparent failure and rarely suggest or point to any means of improving the performance of the tests.

Clinically based evaluations of SLAP lesion tests account for the majority of studies to date; however, studies have also assessed test accuracy by attempting to validate the fundamental design behind various SLAP lesion tests [11, 16, 27, 32]. Provocative SLAP lesion tests, by definition, function to provoke labral symptoms (primarily pain) as

a positive diagnostic sign, by reenacting one of two injury mechanisms. The first mechanism elicits active tension in the biceps tendon and is typically associated with an acute traction trauma to the arm or from the accumulation of microtrauma events over time from repetitive movements such as overhead throwing. The tensile load produced in the biceps tendon can pull and damage the superior labrum, the functional link between the insertion of the biceps tendon and the glenoid rim. The second injury mechanism produces passive compression of the humeral head and is often associated with a fall to outstretched arms. The compressive load causes superior humeral head translation within the glenohumeral joint and can result in a collision between the humerus and labrum, potentially damaging the soft tissue of the labrum [1]. The ability of proposed SLAP lesion tests to reenact the injury mechanisms that they were designed to replicate has been examined from several perspectives including anatomic [11, 16, 27], kinematic [20], and electromyographic [11, 32] methods and results illuminate the importance of design validation during the development of clinical testing procedures.

The purpose of this study was to assess the ability of seven provocative tests to create active tension in the biceps tendon, by characterizing the behaviors of glenohumeral joint stabilizing muscles, with particular interest in the long head of the biceps brachii (LHBB) muscle activation and LHBB muscle selectivity. Tests that elicit larger activation of the LHBB should serve as better diagnostic indicators for SLAP lesions. Also the other joint stabilizing muscles were examined to determine individual muscle contributions during the tests, outlining the ability of each test to selectively activate the LHBB. Selectively activating the LHBB should reduce diagnostic complications related to the frequent presence of other confounding pathologies with

191 SLAP lesions.

Materials and Methods.

Subjects and IRB approval

A cohort of 21 healthy volunteers comprised of 11 females (24.7 ± 6.7 years, 168.4 ± 5.3cm, 66.9 ± 9.1kg) and 10 males (29.4 ± 10.6 years, 178.1 ± 6.6 cm, 80.0 ± 6.4kg) with right arm dominance and no history of shoulder pathology were recruited as subjects in this study. All procedures were approved by the Institutional Review Board at Boise State University, and all participants read and signed a statement of informed consent prior to the start of testing.

EMG Apparatus and Subject Preparation and Electrode Placement

Electromyography (EMG) was used to record muscle activity for seven muscles surrounding the dominant glenohumeral joint including the long and short heads of biceps brachii (LHBB and SHBB), anterior deltoid (DELT), pectoralis major (PECT), latissimus dorsi (LAT), infraspinatus (INFRA), and suprasinatus (SUPRA). Each subject was instrumented with one 44-gage fine-wire indwelling electrode and six surface bipolar silver-silver chloride EMG electrodes (Noraxon, USA Inc, Scottsdale, AZ). The surface electrodes were positioned over the muscle belly and parallel with the orientation of the muscle fibers as suggested by Cram [6]. An additional surface electrode was placed on the acromion process of the non-dominant shoulder to serve as a reference. Due to the location of the SUPRA deep to the trapezius, the indwelling electrode was necessary to acquire SUPRA activity. Using sterile techniques, an emergency medical technician who was trained specifically for this task by a medical doctor placed the fine-wire indwelling

electrode. EMG data were recorded using the Vicon Nexus Software (Vicon, Los Angelos, CA) at 1250 Hz using a Noraxon Telemyo 900 EMG system (Noraxon USA, Inc, Scottsdale, AZ).

EMG Analysis

The EMG signals were analyzed using custom MATLAB software (The MathWorks, Inc, Natick, MA). A traditional filtering method was used for the EMG signals for the LHBB, SHBB, DELT, PECT, and SUPRA. Each signal was smoothed by implementing a root mean square algorithm in combination with a 20ms forward moving window average. The signals were normalized to a percentage of effort based on their respective Maximum Voluntary Isometric Contraction (MVIC) peak EMG signal amplitudes, ideally resulting in a muscle activation range of zero to 100%. Maximum muscle activation and muscle selectivity were determined for each muscle during each test. The raw data for the DELT and LAT were processed by the same method, but the MVIC peak signals were further examined to ensure that the peak amplitude of the signal did not overlap with a peak from the heartbeat artifacts.

Muscle activation and muscle selectivity were calculated to characterize muscle behavior during the provocative tests, with particular interest in the LHBB behavior. Muscle activation was used to determine how effective each SLAP lesion test was at causing individual muscles to activate and was defined as the mean of the peak muscle activities elicited during the three normalized trials. The ability of each provocative test to isolate the LHBB is important for diagnosing SLAP lesions due to its common association with other shoulder pathologies. Therefore in this study, a ratio indicating the

ability of each test to selectively activate each muscle was calculated. The muscle selectivity for each test was defined by the ratio of the peak activation of the muscle of interest over the sum of peak activations for all seven muscles examined, such that a selectivity ratio of 1.0 for any muscle, N, would indicate that muscle N was the only active muscle contributing to the EMG signal while the other six muscles remained inactive. For each test the general selectivity calculation for muscle N was defined as:

$$N\_SelectivityRatio = \frac{A_N}{A_{LHBB} + A_{SHBB} + A_{DELT} + A_{PECT} + A_{LAT} + A_{INFRA} + A_{SUPRA}}$$

N is the muscle of interest (LHBB, SHBB, DELT, PECT, LAT, INFRA, or SUPRA)

$N\_SelectivityRatio$ is the selectivity ratio of muscle N

A_N is the peak muscle activation of muscle N

Subject Protocol – MVICs and Provocative Tests

Using established EMG protocols, each subject was asked to perform MVICs for each muscle of interest on a Biodex System II Dynamometer (Biodex Medical Systems, Shirly, NY). Modifications were made to the MVIC recommendations of Cram, Hintermeister, and Rowlands [6, 13, 29] to accommodate for the use of the Biodex system [Table I]. For each trial, six seconds of data were recorded to ensure that the entire burst of muscle activity was captured during each MVIC. The subjects performed three trials for each MVIC and were asked to maximally contract for a count of three seconds. Subjects rested for thirty seconds between MVIC trials to avoid fatigue effects. For each MVIC, the peak amplitude of the EMG signal among the three trials was used to normalize the provocative test data.

Similarly seven provocative tests were performed based on the descriptions of the original authors but with modifications to accommodate for use of the Biodex System. These tests were chosen based on the findings of a preliminary pilot study that evaluated clinical tests from relevant literature that were designed to reenact either SLAP lesion injury mechanism [20]. Again the subject performed three trials for a three second count for each test, six seconds of data were recorded for each trial, and the subjects rested for thirty seconds between trials.

Provocative Test Descriptions and Study Modifications

The modifications for each MVIC and six of the seven SLAP lesion tests utilized the Biodex System for the purpose of reducing the influence of variances in muscle behavior and test performance. Tests requiring static resistance against an applied load maintained the static subject position through the stationary preset up of the Biodex System. Tests requiring dynamic resistance to an applied load were controlled by the Biodex System allowing a constant velocity regardless of the force applied by the subject.

Active Compression Test (ACPD and ACPU)

Active Compression has two positions, palm down (ACPD) and palm up (ACPU), which vary only by rotation of the arm. The patient is standing with the elbow in full extension, the shoulder is flexed to 90°, and adducted $10-15^{\circ}$ medial to the sagittal plane. For ACPD, the forearm is fully pronated and the glenohumeral joint is maximally internally rotated such that the thumb points down. The patient is asked to resist a uniform downward load applied to their arm by the clinician. For ACPU the

initial patient positioning is unchanged except the arm is externally rotated such that the palm faces up. Again, the patient is asked to resist a uniform downward load applied by the clinician [24].

For the purpose of analysis and due to the nature of the subject population as a control group having no history of shoulder pathology and therefore asymptomatic, this study treated ACPU and ACPD as two independent tests. Both tests were modified such that the subject was seated in the Biodex. The orientation of the subject's arm remained true to O'Brien's original description of the test, but the subject was asked to resist the stationary position of the Biodex arm by attempting to lift his/her arm superiorly for both ACPD and ACPU.

Speed's Test (Speed's)

According to the original description, the patient is standing and resists a downward force applied to the upper extremity with the elbow extended, forearm supinated, and arm elevated to 90° [2]. In this study the orientation of the arm remained similar to the original definition, but Speed's test was modified into a dynamic movement controlled by the Biodex System. The subject's arm started hanging beside and parallel to the body, and then the subject was asked to raise the arm (flex the shoulder) with as much force as possible to 90°. Regardless of the force applied by the subject, motion was restricted to a constant velocity by the Biodex System of 60° per second.

Pronated Load Test (ProLoad)

In the original description, the patient is in the seated position with elbow flexed to 90°, the arm is abducted to 90°, maximally externally rotated, and the forearm is fully pronated. The patient is then asked to perform an isometric contraction of the Biceps [33]. The Pronated Load Test was negligibly modified for this study. The subject sat in the original orientation in the Biodex System, which was set up such that the arm was supported just proximal to the elbow. The subject was asked to perform a bicep contraction (pronated curl) which was resisted by the static set up of the Biodex System.

Biceps Load I Test (Bicep I)

The patient is in the supine position when an anterior apprehension test is performed starting with the arm abducted 90° and the forearm fully supinated according to its original definition[19]. Bicep I was modified such that the patient was seated in the same position as ProLoad, except the forearm was fully supinated. The subject was asked to perform a bicep contraction (curl), which was resisted by the static set up by the Biodex System.

Biceps Load II Test (Bicep II)

The patient is supine with the arm abducted to 120° degrees, the elbow flexed to 90° degrees, and the forearm fully supinated. The patient is then asked to flex the elbow against the resistance of the clinician [18]. For this study the modification for Bicep II paralleled those made to Bicep I except the arm was abducted to 120° degrees instead of 90°.

Resisted Supination External Rotation Test (RSER)

The authors describe putting the patient in the supine position with scapula near the edge of the table, the patients arm is supported by the physician at the wrist, with the arm is abducted to 90° and the elbow is flexed between 65° and 70° degrees. The clinician then externally rotates the arm while the patient is asked to supinate the forearm [23]. The RSER test was essentially unchanged for this study, and the movement was not controlled by the Biodex System. One board certified athletic trainer performed RSER with each subject in the study in the supine position on the Biodex.

Supination Sign Test (Yergason's)

In the seated position with the elbow flexed to 90° and forearm fully pronated, the patient is asked to attempt supination of the forearm while the physician resists the motion while holding the wrist [34]. Yergason's was scarcely modified; as the patient maintained the defined orientation but with the forearm fastened to the static Biodex arm. The patient was asked to attempt to supinate the forearm against the static setup of the Biodex.

Statistical Analysis

Statistical analysis was conducted using SPSS Statistics Software (SPSS, Inc, Chicago, IL) to determine significant differences in maximum muscle activations and muscle selectivities for each test, between tests, and between male and female groups. A

repeated measures analysis of variance (ANOVA) test was performed to identify significant differences between provocative tests for each individual muscle. A pair-wise T-test post-hoc analysis was performed to compare results between each test using a p-value sliding scale Bonferroni adjustment [14]. Likewise, a paired-sample T-test was used to examine potential differences in muscle activation ($p = 0.05$) between male and female groups.

Results.

A post-hoc pair-wise comparison between males and females showed no differences between male and female groups for any muscle or for any provocative test with all p-values exceeding 0.05. Therefore male and female data were pooled for all subsequent statistical analyses. For each individual muscle, the repeated measures ANOVA analysis found significant differences in both muscle activation and muscle selectivity among the eight provocative tests ($p < .05$).

To determine which provocative tests resulted in the greatest activations for the individual muscles, 28 pair-wise comparisons between the eight tests were made for each muscle. Each muscle analyzed showed a significant difference in peak muscle activity between one or more of the pairs of provocative tests with the exception of the LAT. Specifically, the LHBB demonstrated a significant difference ($p=.000$) in activity between tests. The eight statistically significant pair-wise comparisons enabled the tests to be characterized into one of two performance groups based on their respective LHBB activation; high performing and low performing. Speed's, ACPU, Bicep I, and Bicep II, tests were 'high performing', eliciting the largest mean peak EMG amplitudes without statistical differences among the four tests, while RSER, Yergason's, ACPD, and ProLoad were classified as 'low performing' (Figure 2). The mean normalized peak activations (% MVIC) for each muscle elicited during all eight tests are noted in Table II.

The statistical analysis with regards to muscle selectivity for each test proved similar to those for muscle activation. There were significant differences in muscle selectivity across the provocative tests ($p=.000$). A post-hoc pair-wise comparison showed that one or more pairs of tests had significant differences in muscle selectivity for

each muscle with the exception of the LAT and INFRA. The eleven statistically significant pairs allowed the tests to be categorized into high and low performance groups based on LHBB selectivity. RSER, Bicep I, Bicep II, and Yergason's tests were 'high performing', recruiting the LHBB more selectively than ProLoad, Speed's, ACPU, and ACPD, which were categorized as 'low performing' (Figure 3). Again there was no statistical difference among tests within each group. The mean selectivities of each muscle for all eight tests are noted in Table III.

Discussion.

The aim of this study was to characterize the muscle behavior of seven glenohumeral joint stabilizing muscles, focusing on the LHBB, during eight modified provocative tests that were designed to detect SLAP lesions by loading the biceps tendon in tension through LHBB activation. The active tension in the biceps tendon is thought to reproduce the injury mechanism of a SLAP lesion, which should provoke a response from suspected SLAP lesion patients yielding a positive diagnostic sign [1, 33]. In this study, Bicep I and Bicep II were the most promising SLAP lesion tests according to their favorable LHBB behavior, eliciting high LHBB activity while remaining highly selective for the LHBB, indicating these two tests should function effectively as assessment tools for the clinical evaluation of SLAP lesions.

The magnitude of LHBB activation during each of the clinical evaluations is a measure of the sensitivity of the maneuver to incite active tension in the LHBB tendon which should increase the likelihood of detecting a SLAP tear. Although EMG signal amplitude cannot be directly related to muscle force in most cases, the tests that most strongly activate LHBB should provide relatively higher traction forces to the superior labrum. Speed's, ACPU, Bicep I, and Bicep II tests produced the largest LHBB activities, reaching above 90 % MVIC, suggesting that a greater respective load was applied to the biceps tendon during these tests. Although none of the tests apply loads sufficient to produce a SLAP lesion, Speed's ACPU, Bicep I, and Bicep II tests created the largest LHBB activation and therefore reproduced the injury mechanism more effectively than the other four low-performing tests (RSER, Yergason's, ACPD, and ProLoad). Although

SLAP lesion test assessment is prevalent in the literature, comparison between studies is difficult due to the lack of overlap of tests between similar studies. However, two studies support the findings of this study in that ACPU and Bicep II have both been reported to elicit large LHBB EMG amplitudes[11 32].

LHBB selectivity served as an equally important variable to consider for characterizing LHBB behavior and for assessing SLAP lesion tests, as it is an indicator of test specificity. The diagnostic accuracy of SLAP lesion tests are often hindered by the frequent occurrence of other glenohumeral pathologies, such as rotator cuff tears, that make determining the origin of shoulder symptom challenging at best [9, 17, 21, 31]. Consequently provocative tests that are able to isolate the LHBB would be beneficial because high LHBB selectivity denotes a lesser contribution from other joint stabilizing muscles that can produce a false SLAP lesion diagnosis. RSER, Bicep I, Bicep II, and Yergason's tests were 'high performing' with regards to selectively recruiting the LHBB. Each high performing test resulted in LHBB selectivity between 0.23 and 0.25, compared to the range of 0.12 and 0.16 selectivity for the 'low performing' tests (Proload, Speeds, ACPU, and ACPD). Unfortunately LHBB selectivity is not reported elsewhere in the relevant literature, but these results concur with the findings of the preliminary pilot study [20].

The two overall top performing SLAP lesion tests, Bicep I and Bicep II, elicited large LHBB activation while demonstrating high LHBB selectivity. The clinical implications derived from the remaining tests that were 'high performing' in only a single area of LHBB behavior, either highly specific (activation – ACPU and Speed's) or highly sensitive (selective – RSER and Yergason's), may be limited if used on their own. Top

performing SLAP lesion tests, that elicited large LHBB activation and were highly selective for the LHBB, should be closely examined in hopes of defining the characteristics that may be responsible for their promising LHBB behavior.

Bicep I and Bicep II are very similar tests, varying only by the flexion of the shoulder joint. Bicep I, Bicep II, Speed's, and ACPU, all of have desirable behavior in one or both suites and may be useful for future work, by examining the clinical implications of these tests in combination. These four tests, Bicep I, Bicep II, Speed's, and ACPU, share similar test and design characteristics relating to location of the applied load, forearm orientation, joint position, and line of pull during either a static or dynamic provocative test designed to activate the LHBB. Each of these tests was performed with a supinated forearm and required active resistance to an external load applied perpendicular to the palm of the subject's hand. Each high performing test was performed in one of two joint positions which placed the LHBB and biceps tendon in a direct line of pull with the superior labrum. The first joint position (Speed's and ACPU) flexed the shoulder to a maximum of 90° with the elbow fully extended. The second joint position (Bicep I and Bicep II) had the shoulder abducted at or above 90° with the elbow flexed at 90°. The major difference between these four tests is the way the tests are performed; Speed's is a dynamic test while Bicep I, Bicep II, and ACPU are static tests, where the patient resists the load without the ability to move.

In this study ACPU and the Speed's were extremely similar and although both were 'high performing' for LHBB activation, their differences may prove important means of understanding the role and importance of SLAP lesions test characteristics. The tests have slight differences in patient orientation and type of movement; ACPU places

the arm medial to the sagittal plane and the test is static, while Speed's is parallel to the medial plane and involves a dynamic movement. These small differences may have important consequences, and a close examination of these kinds of test characteristics and their relation to test performance may help illuminate a means of improving test design and accuracy.

Although the focus of this study was the behavior of the LHBB, six other joint stabilizing muscles were recorded to enable LHBB selectivity calculations and in hopes of characterizing any other muscle behaviors or patterns. Peak muscle activities and muscle selectivity were examined for all remaining muscles (SHBB, DELT, PECT, LAT, INFRA, and SUPRA), and statistical analysis revealed that it may be unnecessary to monitor the LAT and INFRA during these tests, because none of the tests had a significant difference in terms of activation of the LAT or in selectively isolating either the LAT or INFRA muscles.

The primary inherent limitation of this study is that the subjects had no history of shoulder pathology; therefore labral symptoms were not used as a means to assess SLAP test performance. Also the healthy subject pool may misrepresent SLAP lesion patients due to the potential for differences in muscle behavior between healthy subjects and those with labral pathology. Furthermore, the EMG signals were all normalized based on peak activities elicited during MVIC, and results exceeded 100% in some cases and may make comparison between subjects difficult. Specifically, the dynamic Speed's test, which had the largest mean activation (140.9% MVIC) among the tests, was not a surprising finding, as the dynamic movement was normalized to a static MVIC. Muscle activation is known to vary with both muscle length and shortening or lengthening velocity. Therefore,

comparing activation during a dynamic test to data collected in a static configuration may not be optimal. For the static tests, LHBB activations were generally below or much less than that of Speed's, suggesting that the normalization procedure was more appropriate for those tests. However, in some tests subjects were able to achieve more than 100% MVIC in some muscles, which means either that the tests were more effective in isolating those muscles than the MVIC configurations, or that slight differences in positioning or in subject effort in the clinical tests and the MVIC tests affected the muscle activation values recorded.

Future studies would improve on the scope of this study by recruiting subjects who have a suspected SLAP lesion and are scheduled for arthroscopic assessment. Employing the methods and results of this study, improvements would utilize the promising LHBB behavior of the top performing modified tests (Bicep I and Bicep II) in conjunction with analyses of associated joint torques. Although joint torque data was not collected in this study due to the inability to acquire torque information for all of the eight modified tests, the top performing SLAP lesions tests are oriented such that the Biodex System could easily provide such information. An analysis of joint torques and associated loads during these tests may further quantify the ability of these tests to create tension in the biceps tendon.

Recent studies utilizing arthroscopic verification for clinical evaluations have documented a drastic increase in SLAP lesion detection by using the indications of two or more SLAP tests, specifically when at least one test is highly sensitive and another is highly specific [8, 25]. Consequently, assessing the array of 'high performing' test

combinations, utilizing various combinations of single suite high performance tests with various test characteristics may have surprising results and prove worthwhile.

Lastly, although difficult to determine and requiring a large pool of control and experimental data, comparisons between the muscle behaviors of a healthy population and those who have a suspected SLAP lesion may illuminate some general pattern differences that could be indicative of SLAP lesions and be useful for furthering clinical diagnostic techniques and accuracy.

Conclusions.

In summary, modified versions of Bicep I and Bicep II resulted in the greatest LHBB activation and LHBB selectivity of the SLAP lesion tests in this study. ACPU, and Speed's resulted in the large LHBB activation, but were not selective for the LHBB. Bicep I, Bicep II, ACPU, and Speed's each elicit some promising LHBB behavior, and maybe useful in combination to aid the clinical detection of SLAP lesions. These four tests utilize a unique range of test variables that may prove valuable for optimal SLAP test design and function. Future studies should evaluate the importance of these variables, incorporate joint torque analyses, and expand the scope of the study to include patients who have a suspected SLAP lesion to optimize, validate, and improve the diagnostic accuracy of provocative SLAP lesion test.

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Tables

Table I: MVIC joint positions and resisted maneuvers for the seven muscles of interest.

Table II: Resulting mean normalized peak muscle activations (%MVIC) and standard deviations monitored during each SLAP lesion test.

Table III: Resulting mean muscle selectivity values and standard deviations monitored during each SLAP lesion test.

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Muscle	Joint Position	Resisted Maneuver
DELT	Arm at side	Shoulder flexion
LHBB / SHBB	Elbow flexed 90º, shoulder flexed 90º	Elbow flexion
INFRA	Arm abducted 45º, elbow flexed 90º	External Rotation
LAT	Shoulder flexed 90º, arm internally rotated	Shoulder extension
PECT	Arm abducted 90º, forearm supinated	Horizontal adduction
SUPRA	Arm abducted 90º, forward flexed 30º, and internally rotated	Maintain against resistance

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Muscle Peak Mean Activation (% MVIC) and Standard Deviation During SLAP Lesion Tests														
	LHBB		SHBB		DELT		PECT		LAT		INFRA		SUPRA	
ACPD	116.6	(75.2)	86.7	(60.4)	192.1	(97.6)	89.2	(42.8)	116.2	(161.6)	182.3	(174.8)	84.8	(38.6)
ACPU	74.9	(66.4)	15.7	(12.8)	116.7	(65.7)	28.4	(17.5)	93.8	(146.3)	110.1	(82.9)	105.4	(62.7)
Speeds	140.9	(100.9)	104.2	(90.6)	158.4	(72.0)	88.0	(35.4)	124.1	(158.4)	143.3	(96.4)	107.7	(58.5)
Bicep I	97.6	(37.2)	88.9	(36.5)	43.9	(49.4)	36.0	(20.0)	72.6	(65.4)	56.8	(58.9)	26.7	(22.5)
Bicep II	94.0	(48.0)	88.7	(42.8)	49.8	(56.9)	44.8	(21.8)	56.2	(46.4)	41.9	(37.9)	30.7	(43.1)
ProLoad	58.1	(32.8)	39.8	(19.0)	58.9	(49.5)	28.5	(15.9)	70.1	(60.4)	69.2	(53.7)	39.4	(31.1)
RSER	89.2	(65.7)	85.6	(65.0)	15.4	(17.4)	23.5	(19.5)	49.5	(32.6)	59.2	(61.1)	25.9	(22.5)
Yergasons	81.1	(46.3)	81.6	(52.0)	22.3	(19.1)	31.8	(19.9)	98.4	(152.8)	56.3	(50.4)	24.1	(19.7)

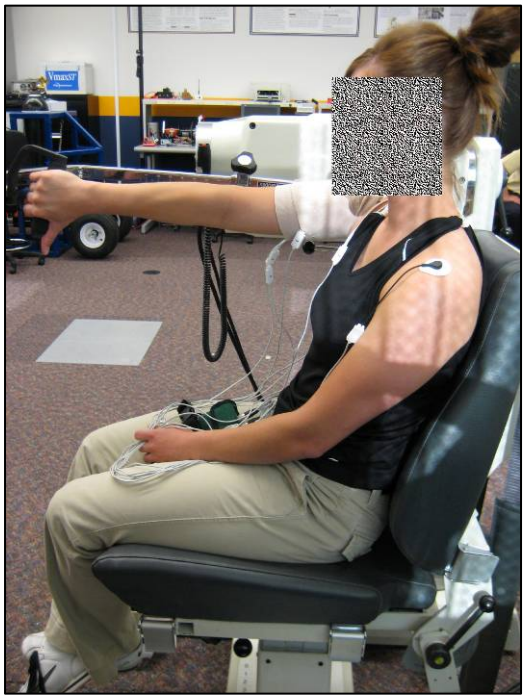
Muscle Mean Selectivity and Standard Deviation During SLAP Lesion Tests														
	LHBB		SHBB		DELT		PECT		LAT		INFRA		SUPRA	
ACPD	0.132	(0.056)	0.118	(0.059)	0.300	(0.104)	0.225	(0.139)	0.268	(0.203)	0.633	(0.145)	0.105	(0.049)
ACPU	0.122	(0.091)	0.034	(0.032)	0.259	(0.118)	0.102	(0.074)	0.264	(0.178)	0.494	(0.200)	0.213	(0.122)
Speeds	0.152	(0.065)	0.138	(0.066)	0.263	(0.076)	0.209	(0.076)	0.283	(0.214)	0.566	(0.182)	0.131	(0.064)
Bicep I	0.244	(0.079)	0.308	(0.124)	0.188	(0.134)	0.229	(0.110)	0.479	(0.206)	0.587	(0.230)	0.064	(0.055)
Bicep II	0.231	(0.070)	0.303	(0.113)	0.217	(0.135)	0.293	(0.146)	0.452	(0.188)	0.558	(0.281)	0.072	(0.082)
ProLoad	0.160	(0.069)	0.144	(0.078)	0.204	(0.124)	0.150	(0.078)	0.401	(0.252)	0.602	(0.220)	0.113	(0.087)
RSER	0.255	(0.086)	0.336	(0.136)	0.092	(0.080)	0.164	(0.106)	0.415	(0.229)	0.636	(0.203)	0.075	(0.047)
Yergasons	0.225	(0.086)	0.311	(0.158)	0.104	(0.063)	0.186	(0.086)	0.427	(0.226)	0.653	(0.189)	0.067	(0.044)

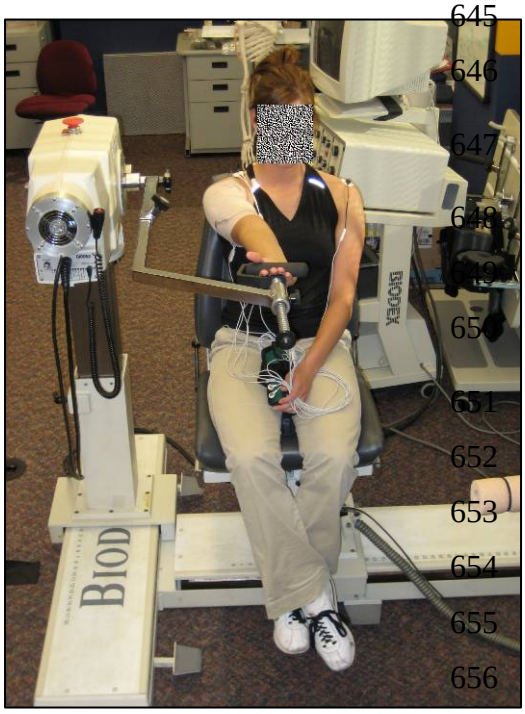
Illustrations and Legends

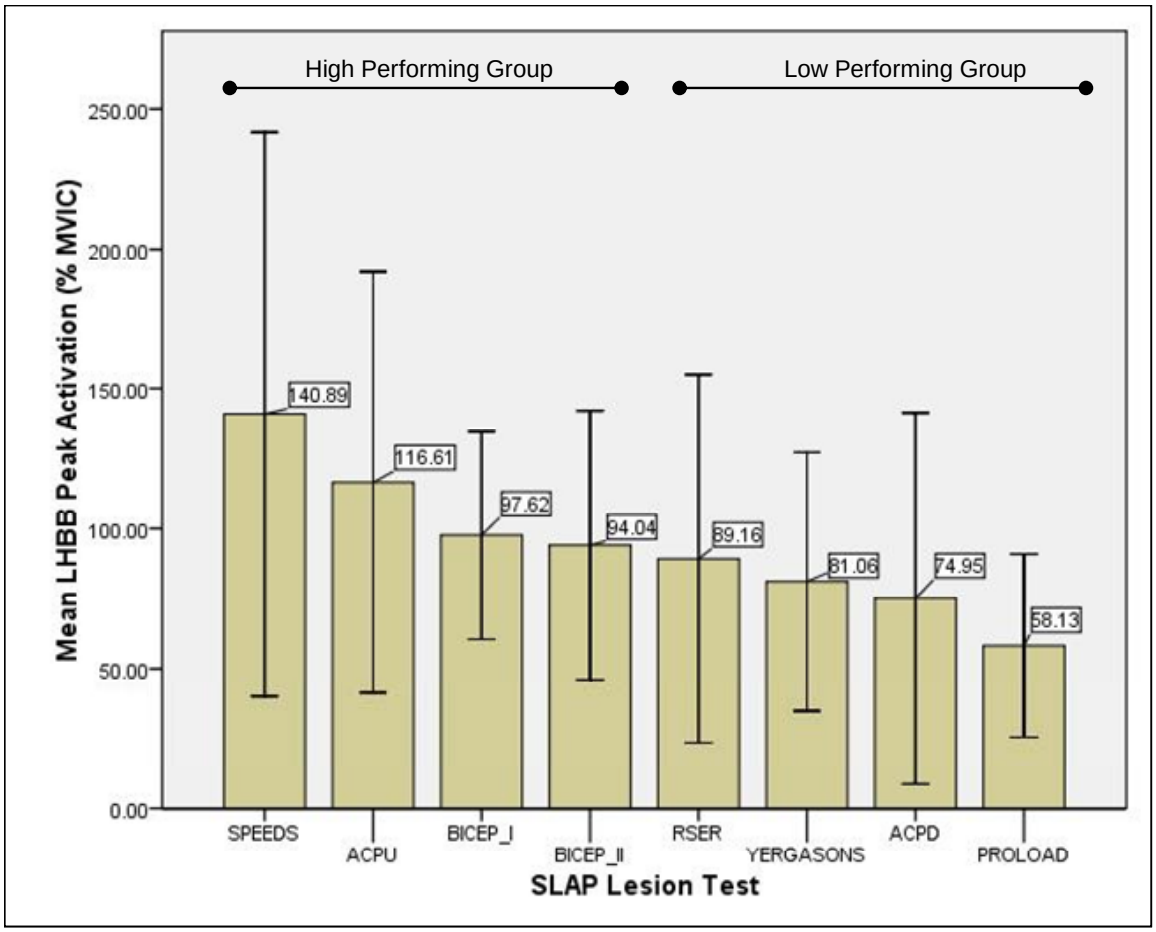
Figure 1: Example of Biodex System modifications, ACPD and ACPU.

Figure 2: LHBB mean muscle activation (%MVIC) for each SLAP test.

Figure 3: LHBB mean muscle selectivity for each SLAP test.




BIODEX


High Performing Group
Low Performing Group
Mean LHBB Peak Activation (% MVIC)
250.00
200.00
150.00
100.00
50.00
0.00
140.89
116.61
97.62
94.04
89.16
81.06
74.95
58.13
SPEEDS
ACPU
BICEP_I
BICEP_II
RSER
YERGASONS
ACPD
PROLOAD
SLAP Lesion Test

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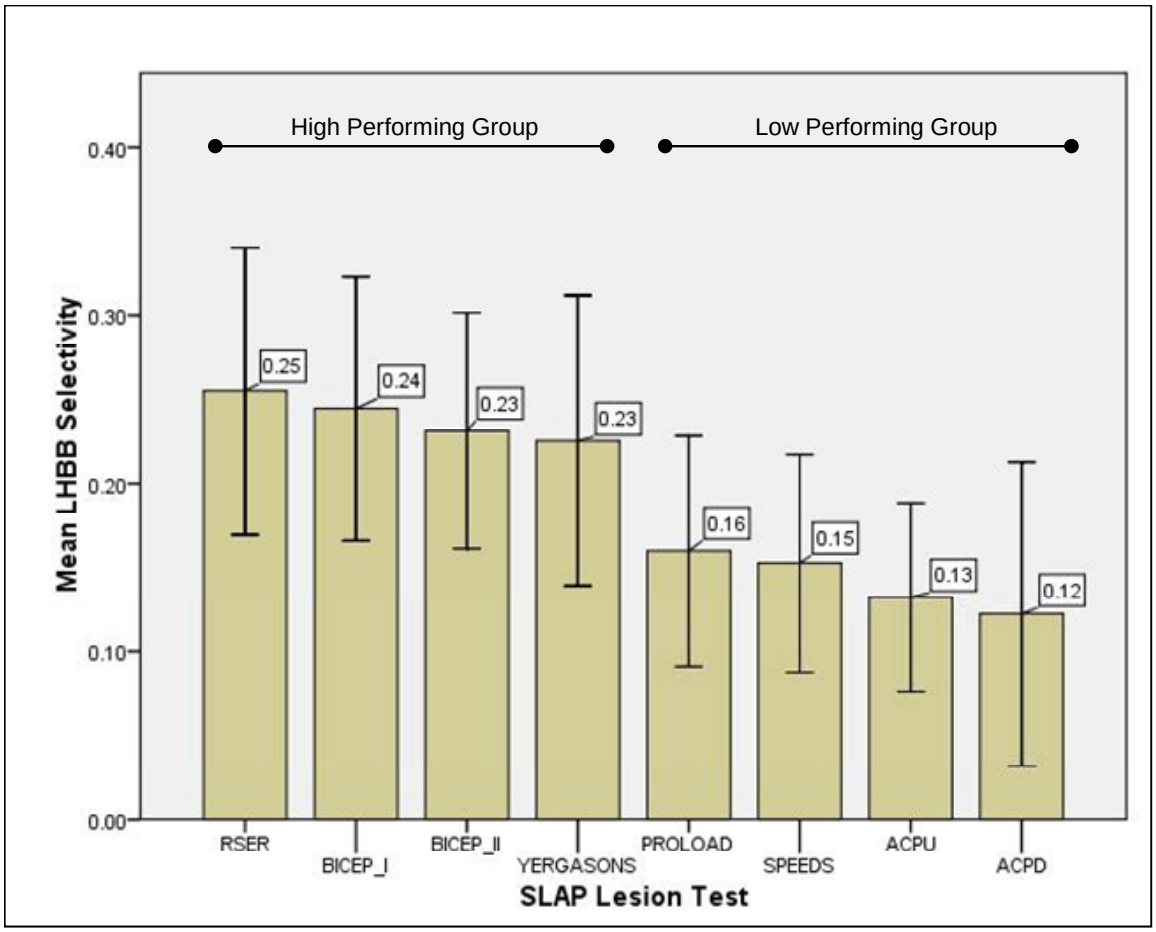
High Performing Group
Low Performing Group
Mean LHBB Selectivity
0.40
0.30
0.20
0.10
0.00
0.25
0.24
0.23
0.23
0.16
0.15
0.13
0.12
RSER
BICEP_I
BICEP_II
YERGASONS
PROLOAD
SPEEDS
ACPU
ACPD
SLAP Lesion Test

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