Reflexive Muscle Activation Alterations in Shoulders With Anterior Glenohumeral Instability

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Background: Patients with glenohumeral instability have proprioceptive deficits that are suggested to contribute to muscle activation alterations.

Hypothesis: Muscle activation alterations will be present in shoulders with anterior glenohumeral instability.

Study Design: Posttest-only control group design.

Methods: Eleven patients diagnosed with anterior glenohumeral instability were matched with 11 control subjects. Each subject received an external humeral rotation apprehension perturbation while reflexive muscle activation characteristics were measured with indwelling electromyography and surface electromyography.

Results: Patients with instability demonstrated suppressed pectoralis major and biceps brachii mean activation; increased peak activation of the subscapularis, supraspinatus, and infraspinatus; and a significantly slower biceps brachii reflex latency. Supraspinatus-subscapularis coactivation was significantly suppressed in the patients with instability as well.

Conclusions and Clinical Relevance: In addition to the capsuloligamentous deficiency and proprioceptive deficits present in anterior glenohumeral instability, muscle activation alterations are also present. The suppressed rotator cuff coactivation, slower biceps brachii activation, and decreased pectoralis major and biceps brachii mean activation may contribute to the recurrent instability episodes seen in this patient group. Clinicians can implement therapeutic exercises that address the suppressed muscles in patients opting for conservative management or rehabilitation before and after capsulorraphy procedures.

Keywords: instability; reflexes; electromyography; shoulder

Stability is defined as the state of remaining unchanged even in the presence of forces that would normally change such a state.[62] Adapting this definition for specific application to the shoulder joint, stability is defined as proper alignment of the humerus within the glenoid fossa through an equalization of forces.[39] Shoulder joint stability requires both passive and dynamic components to maintain proper alignment in this inherently unstable ball-and-socket joint.[47] Specifically, the passive components of shoulder stability include the osseous geometry, negative intra-articular pressure, glenoid labrum, and capsuloligamentous restraints.[50] Dynamically, stability is maintained by the rotator cuff musculature and the biceps brachii, as well as other shoulder musculature (ie, pectoralis major, deltoid, latissimus dorsi, etc).[50] As separate entities, the passive or dynamic components cannot act alone to provide stability. Bigliani et al[10] suggested that the capsuloligamentous structures, mediated by the sensorimotor system, must work in synergism with well-balanced muscle activation to provide stability at the shoulder joint.[47]

The sensorimotor system encompasses all of the sensory, motor, and central integration and processing components of the central nervous system involved with maintaining functional joint stability.[39] Sensory information (proprioception) travels through afferent pathways to the central nervous system where it is integrated with input from other levels of the nervous system, which elicit efferent motor responses (neuromuscular control) vital to coordinated movement patterns and functional stability.[39] Specifically, the shoulder relies on proprioceptive input from the glenohumeral ligaments and capsule, musculo-

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No author or related institution has received financial benefit from research in this study.

The American Journal of Sports Medicine, Vol. 32, No. 4
DOI: 10.1177/0363546503262190

tendinous structures, and cutaneous structures for mediation of activation of the shoulder musculature, centralization of the humeral head within the glenoid fossa through muscle coactivation, dynamic tension of the capsule, and reflexive stabilization.[13,16,25,60,66,68]

Anterior instability, resulting from dislocation/subluxation of the shoulder, is described as excessive anterior translation of the humeral head on the glenoid fossa and results in significant tissue compromise at the shoulder complex. The compromised structures include consequential disruption in capsular integrity,[22,50] excessive capsular volume,[30] rotator interval deficiencies,[49] Bankart lesions,[5,22,52,53] lengthening of the subscapularis,[61] and tendinous rupture.[69] Disruption of the stabilizing structures (capsuloligamentous and musculotendinous) results in mechanical instability of the shoulder joint.[31,61]

In addition to studying the mechanical instability that exists after a dislocation/subluxation, our work as well as the work of others have demonstrated sensorimotor alterations manifesting as decreased proprioception.[21,40,59,71] Yet the neuromuscular control alterations that contribute to glenohumeral instability have not been demonstrated. To date, neuromuscular control alterations, including reflexive muscle activation and muscle coactivation, have not been reported. The purpose of this study was to assess reflexive muscle activation characteristics as a measure of neuromuscular control in the shoulders of patients with anterior glenohumeral instability. Given the proprioceptive deficits seen in shoulders with glenohumeral instability, it was hypothesized that neuromuscular control alterations are also present.

MATERIALS AND METHODS

Subjects

The glenohumeral instability group in this study consisted of 11 patients diagnosed by an orthopaedic surgeon with recurrent traumatic unidirectional anterior instability. Recurrent instability was operationally defined as 3 to 25 episodes of instability and based on previous data that suggest that more than 1 episode of traumatic anterior dislocation may be needed to have recurrent instability through subfailure of the capsuloligamentous structures. Thus, to have recurrent instability manifest, a subject needed 2 prior episodes.[15,70] Instability was defined as either a glenohumeral dislocation or subluxation. All episodes of instability occurred acutely and traumatically during athletic events, progressive resistance training, or an altercation with another individual. All anterior instability patients were nonoperative at the time of the study but failed conservative treatment. All experimental subjects were matched with control subjects according to age, height, weight, and limb dominance and involvement. The control group in this study consisted of 11 subjects with no history of upper extremity injury. Overhead throwing athletes from both the instability and control groups were excluded from participation, given the neuromuscular adaptations that are present in this population.[1,14,57]

TABLE 1
Subject Demographics

	Glenohumeral Instability Group		Control Group		
	Mean	±SD	Mean	±SD	*P* Value[a]
Age, years	21.10	5.82	20.80	3.49	.89
Height, cm	177.04	11.18	175.51	9.22	.74
Weight, kg	81.27	13.45	80.45	12.68	.89
Episodes, n	12.60	9.66	0	0	

[a]An independent sample *t* test was performed to ensure that no group differences existed between the 2 groups of participants ($P < .05$).

Subject demographics appear in Table 1. An independent sample *t* test was performed to ensure no group differences existed between the glenohumeral instability and control group subjects for age, height, and weight (see Table 1).

Instrumentation

Electromyography data were collected with the Noraxon Telemyo (Noraxon, Scottsdale, Ariz) electromyography system. The Telemyo system is a frequency modulated (FM) telemetry system. Electromyographic signals collected from the electrodes were passed through a single-ended amplifier (gain 500) to an 8-channel FM transmitter. A receiver unit collected the telemetry signals from the transmitter. The receiver amplified (gain 500) and filtered (15-500 Hz Band Pass Butterworth filter, common mode rejection ratio of 130 db) the signals. Signals from the receiver were converted from analog to digital data via a PCM16S/12 (16-channel, 12-bit) analog/digital board (ComputerBoards, Middleboro, Mass) at a rate of 1000 Hz. The digital data were collected and stored with MyoResearch 2.02 (Noraxon) on a personal computer for data reduction.

The shoulder apprehension perturbation device consisted of a Biodex System III isokinetic dynamometer (Biodex Medical, Shirley, NY) and modified lever arm (Figure 1). The lever arm consisted of securing a Model 41 (Sensotec Inc, Columbus, Ohio) 500-lb compression load cell in series with the modified Biodex isokinetic lever arm. A molded plastic, half-sphere, high-density foam contact pad was secured to the load cell and acted as the contact point between the lever arm and the subject's limb. The voltage from the load cell was collected and synchronized with electromyographic data in the MyoResearch software. The dynamometer chair was fitted with a pad that supported the upper limb just distal to the axilla, which assisted with subject positioning.

Testing Procedures

Before testing, the subjects were prepared for electromyographic analysis. Dual fine electrodes constructed with 0.05-mm nickel chromium alloy wire insulated with nylon

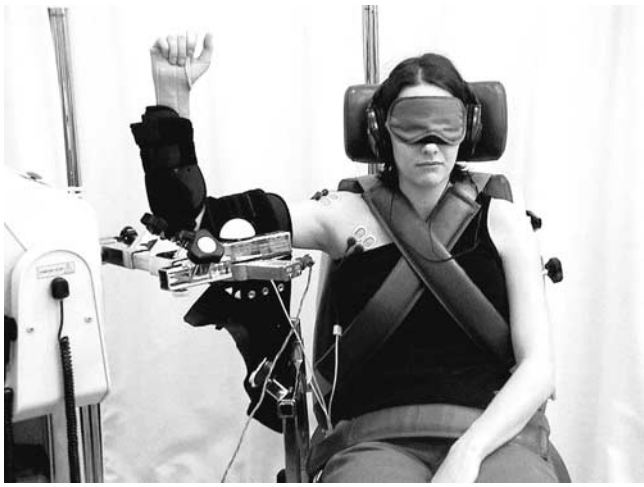

Figure 1. A subject positioned in the shoulder apprehension perturbation device.

(California Fine Wire Co, Grover Beach, Calif) were prepared.[7,8] Indwelling electrodes were inserted intramuscularly via a 1.5-in 25-gauge needle into the supraspinatus, infraspinatus, and subscapularis.[23,32] Insertion sites were sanitized using 70% isopropyl alcohol and iodine solution. A medical physician performed all indwelling electrode insertions.

Silver-silver chloride surface electrodes (Medicotest Inc, Rolling Meadows, Ill) were used for measurement of superficial muscle activity. To lower impedance, we prepared the skin by shaving any hair present, applying mild abrasion with a low-abrasive emery board, and wiping the area with 70% isopropyl alcohol. Two adjacent surface electrodes were placed side by side with 1 cm separating the centers of the electrode.[19] The electrodes were placed perpendicular to the orientation of the muscle fibers.[19] Surface electrodes were placed on the sternal portion of the pectoralis major, anterior deltoid, latissimus dorsi, and biceps brachii.[6] A ground electrode was placed on the olecranon process of the elbow. Correct positioning of all electrodes was confirmed through isolated manual muscle tests of each muscle.[33]

During testing, the subjects were in a seated position on the isokinetic dynamometer chair, inclined 5° from vertical. The subject's involved limb was positioned and supported at 90° of abduction and 30° of horizontal adduction in the scapular plane. A fixed hinge brace (ROM Elbow Deluxe, DonJoy Orthopedics, Vista, Calif) maintained the elbow at 90° of flexion. A rigid wrist brace maintained neutral position of the wrist. Each subject was asked to maximally externally rotate without compensatory motion. An electronic range-of-motion stop from the Biodex System III was set for that position. The range-of-motion stop protected the subject from achieving range of motion outside of an active range. Before each trial, the shoulder was placed in a position of 35° before end range with the assistance of the internal electrogoniometer present within the Biodex System III. This was the position that the perturbation was applied. Subjects were asked to maintain this position while the investigator visually inspected movement out of this position before application of perturbation. Subjects were repositioned if the position was not maintained.

The perturbation consisted of the lever arm striking the limb at 180 deg/sec. Subjects were instructed not to intervene with the perturbation.[17] Visual, auditory, and tactile cues were eliminated with a blindfold, headphones, and low-intensity vibration provided by an air compressor. Six trials were completed by each subject and analyzed.

Data Reduction

Electromyography data were band pass filtered (20-500 Hz) using a 4th-order Butterworth zero-phase filter similar to recommendations set forth by the *Journal of Electromyography and Kinesiology*. The analog data from the compression load cell were filtered using a 20-Hz, 4th-order Butterworth zero-phase filter. Perturbation onset was determined by finding the maximum voltage value from the compression load cell data. The mean and SD of a linear window from 550 to 350 milliseconds before the maximum value were calculated. Perturbation onset threshold (POT) was calculated as the mean plus 2 SDs.[14,20,28] The onset of perturbation was evaluated by comparing compression load cell data points in a point-by-point fashion until the POT criterion was met. Once the POT criterion was met, perturbation application was considered at that data point (Figure 2A). Muscle activity onset was determined by calculating the mean and SD amplitude of rectified electromyographic data 150 milliseconds before onset of perturbation. To determine onset, the muscle voltage threshold (MVT) must be calculated; MVT was calculated as the mean of the linear envelope 150 milliseconds before perturbation plus 3 times the SD.[14,20,28] Onset of muscle activity was determined in a point-by-point fashion until the MVT was met[14,20,28] (Figure 2B). Muscle reflex latency was calculated for all 7 muscles tested. Reflex latency was calculated as the time interval between perturbation onset and muscle activity onset (Figure 2C). Mean activation of the reflexive response was calculated as the average of 150 milliseconds of activation after muscle activation onset (Figure 2D). Peak activation was calculated as the maximum activation occurring within 150 milliseconds after muscle activation onset (Figure 2E). Muscle activation (mean and peak) was reported as percentage of the total activation occurring during a mean ensemble occurring during the first 200 milliseconds of activation.

In addition, coactivation was calculated for the rotator cuff muscles. We operationally defined coactivation, according to Rudolph et al,[56] as the simultaneous activation of 2 muscles using the following equation: EMGS/EMGL × (EMGS + EMGL), where EMGS is the mean activation in the less active muscle and EMGL the mean activation in the more active muscle. This ratio was multiplied by the sum of the activity found in the 2 muscles. This method provided a sample-by-sample estimate of the relative activation of the pair of muscles as well as the magnitude of coactivation. High coactivation values represented a high

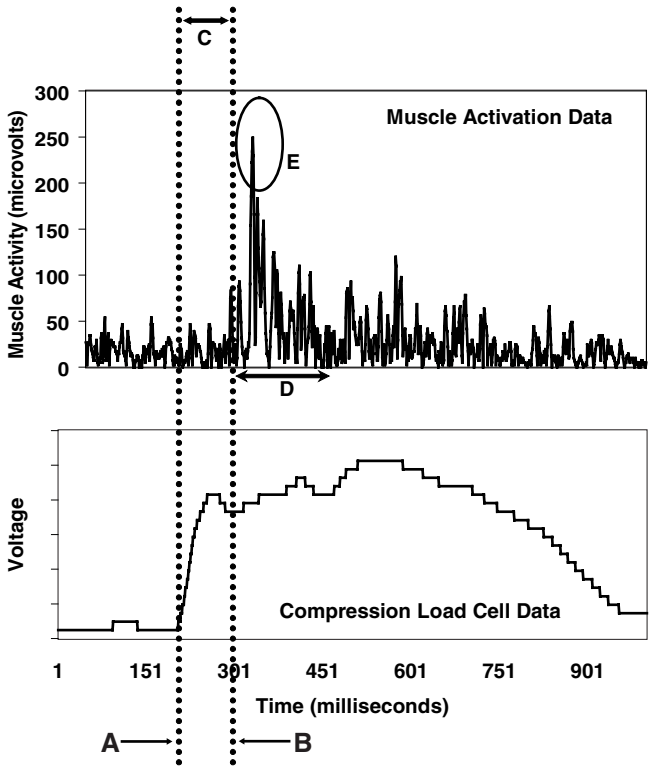

Figure 2. Muscle activity and compression load cell data used for calculation of perturbation onset (A), muscle activation onset (B), muscle reflex latency (C), mean activation (D), and peak activation (E).

level of activation of both muscles, whereas low coactivation values indicated either low-level activation of both muscles or high-level activation of one muscle along with low-level activation of the other muscle in the pair. Low coactivation values represented more selective activation of muscles, whereas large coactivation values represented more generalized muscle activation. Coactivation was calculated for the supraspinatus-infraspinatus, supraspinatus-subscapularis, and subscapularis-infraspinatus muscle pairs.

Data Analysis

To assess group differences (glenohumeral instability group vs control group), all variables were analyzed using separate independent sample 2-tailed t tests with SPSS version 11 (SPSS Inc, Chicago, Ill) statistical software. Independent sample t tests were chosen given that all data met the assumption of normality according to Kolmogorov-Smirnov tests for normality. An alpha level of .05 was set a priori to determine significance for all statistical analyses.

RESULTS

Patients with instability demonstrated suppressed pectoralis major (P = .04) and biceps brachii (P = .04) mean activation (Figure 3) as well as increased peak activation of the subscapularis (P = .05), supraspinatus (P = .05), and infraspinatus (P = .02) compared to the control subjects (Figure 4). Descriptive statistics for mean activation and peak activation appear in Tables 2 and 3, respectively. The instability patients also exhibited a significantly slower biceps brachii reflex latency (P = .01) than their control counterparts (Figure 5). Descriptive statistics for the

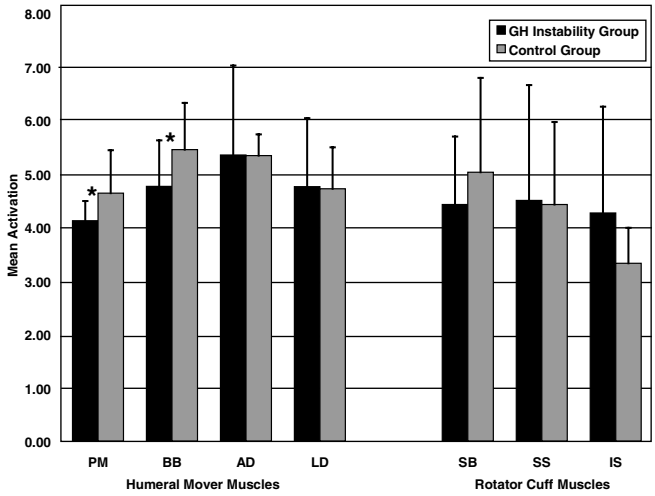

Figure 3. Mean muscle activation for both the humeral mover muscles and rotator cuff muscles. *Significantly lower mean activation in the glenohumeral (GH) instability group than in the control group. PM, pectoralis major; BB, biceps brachii; AD, anterior deltoid; LD, latissimus dorsi; SB, subscapularis; SS, supraspinatus; IS, infraspinatus.

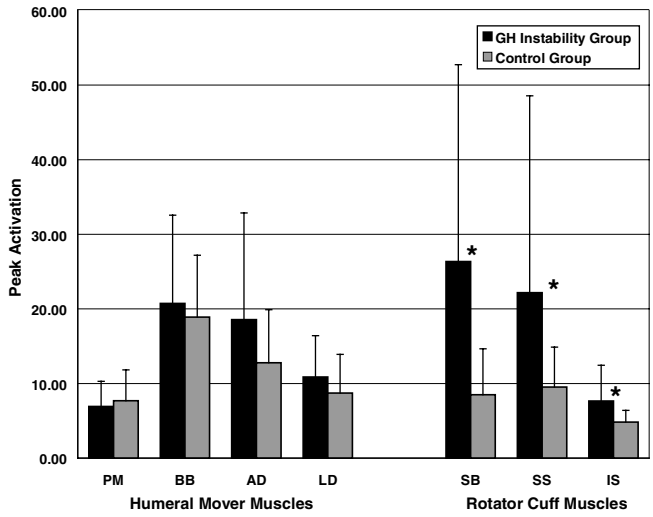

Figure 4. Peak activation for both the humeral mover muscles and rotator cuff muscles. *Significantly higher peak activation in the glenohumeral (GH) instability group than in the control group. PM, pectoralis major; BB, biceps brachii; AD, anterior deltoid; LD, latissimus dorsi; SB, subscapularis; SS, supraspinatus; IS, infraspinatus.

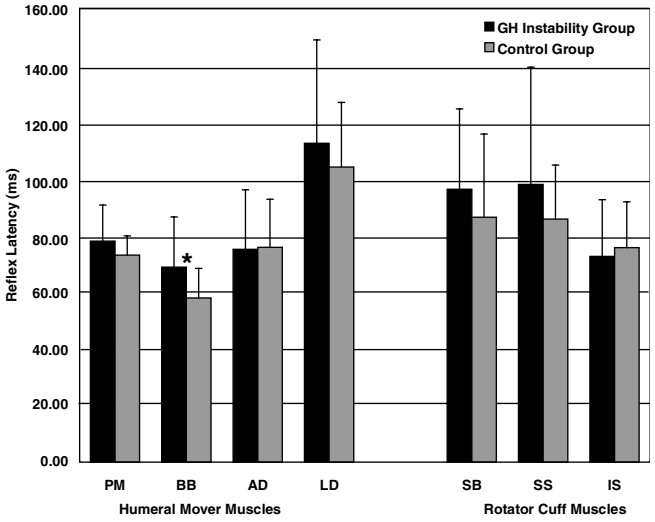

Figure 5. Muscle reflex latency for both the humeral mover muscles and rotator cuff muscles. *Significantly longer time to activation in the glenohumeral (GH) instability group than in the control group. PM, pectoralis major; BB, biceps brachii; AD, anterior deltoid; LD, latissimus dorsi; SB, subscapularis; SS, supraspinatus; IS, infraspinatus.

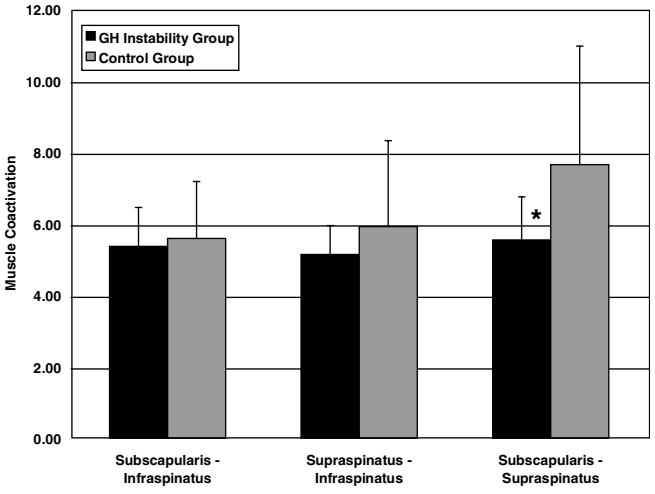

Figure 6. Coactivation of the rotator cuff muscles. *Significantly lower coactivation in the glenohumeral (GH) instability group than in the control group.

TABLE 2
Mean Muscle Activation (%)

	Glenohumeral Instability Group		Control Group	
	Mean	±SD	Mean	±SD
Pectoralis major	4.11[a]	0.37	4.63	0.80
Biceps brachii	4.76[a]	0.86	5.44	0.87
Anterior deltoid	5.35	1.67	5.33	0.40
Latissimus dorsi	4.75	1.28	4.71	0.78
Subscapularis	4.42	1.27	5.02	1.76
Supraspinatus	4.50	2.16	4.42	1.54
Infraspinatus	4.26	1.99	3.32	0.66

[a]Significantly different from the control group.

TABLE 3
Peak Muscle Activation (%)

	Glenohumeral Instability Group		Control Group	
	Mean	±SD	Mean	±SD
Pectoralis major	6.91	3.37	7.69	4.14
Biceps brachii	20.74	11.82	18.90	8.30
Anterior deltoid	18.56	14.30	12.79	7.10
Latissimus dorsi	10.87	5.54	8.72	5.17
Subscapularis	26.35[a]	26.40	8.49	6.17
Supraspinatus	22.18[a]	26.39	9.52	5.34
Infraspinatus	7.65[a]	4.79	4.83	1.57

[a]Significantly different from the control group.

TABLE 4
Muscle Reflex Latency (milliseconds)

	Glenohumeral Instability Group		Control Group	
	Mean	±SD	Mean	±SD
Pectoralis major	77.99	12.74	73.05	6.78
Biceps brachii	68.82[a]	17.69	57.96	10.40
Anterior deltoid	75.11	21.01	75.82	16.94
Latissimus dorsi	112.55	36.45	104.13	22.80
Subscapularis	96.32	28.36	86.44	29.34
Supraspinatus	98.00	41.39	85.80	19.05
Infraspinatus	72.59	20.03	75.71	16.17

[a]Significantly different from the control group.

muscle reflex latencies appear in Table 4. Supraspinatus-subscapularis coactivation was significantly suppressed (P = .04) in the instability group (Figure 6). Descriptive statistics for coactivation appear in Table 5.

DISCUSSION

Results from this study indicated that patients diagnosed with recurrent traumatic anterior instability displayed altered neuromuscular control of the shoulder dynamic stabilizers. Shoulder muscle dysfunction like that seen in the current study can have dramatic effects on the resulting glenohumeral joint forces, thereby affecting joint stability and possibly contributing to the recurrence commonly seen in patients with glenohumeral instability.[26,29,42,55] Specifically, the instability group demonstrated suppressed pectoralis major and biceps brachii mean activation, increased peak activation of the rotator cuff muscles, a slower biceps brachii reflex latency, and supraspinatus-subscapularis suppression. Other studies have reported

TABLE 5
Muscle Coactivation (%)

	Glenohumeral Instability Group		Control Group	
	Mean	±SD	Mean	±SD
Subscapularis-infraspinatus	5.36	1.09	5.58	1.59
Supraspinatus-infraspinatus	5.13	0.81	5.92	2.40
Subscapularis-supraspinatus	5.54[a]	1.26	7.65	3.31

[a]Significantly different from the control group.

altered muscle firing patterns in patients with instability. Glousman et al[24] measured muscle activity during pitching and demonstrated increased compensatory supraspinatus and biceps brachii activity in unstable individuals likely to accommodate for a lack of glenohumeral stability. In addition, Glousman et al[24] reported decreased subscapularis, pectoralis major, latissimus dorsi, and serratus anterior activity during the late cocking phase of pitching in throwers with shoulder instability. Kronberg et al[34] demonstrated decreased anterior and middle deltoid activity with shoulder flexion and shoulder abduction movements in subjects with instability. McMahon et al[44] performed a similar study and showed that individuals with anterior instability demonstrated decreased supraspinatus muscle activity during abduction and scaption and decreased serratus anterior muscle activity during abduction, scaption, and forward flexion. This disrupted muscle activity may alter the force couple mechanism that exists between the deltoid and rotator cuff muscle as well as scapular stabilization mechanisms vital to functional stability and coordinated movement patterns.

Although previous studies have demonstrated muscle activation alterations present during movements such as throwing and humeral elevation, the current study attempted to assess muscle activations during conditions that mimicked the way instability episodes occur. Muscle activation alterations were measured in a position of apprehension (abduction and external rotation), in which instability episodes are most likely to manifest.[26,45] Others have measured muscle activation in an apprehension position in patients diagnosed with instability. Wallace et al[65] measured muscle reflex latency of the pectoralis major and infraspinatus muscles. Like the current study, no differences were reported in muscle reflex latency of those particular muscles.

Although no differences in pectoralis major reflex latencies were observed, patients with recurrent instability did demonstrate suppressed mean activation of the pectoralis major in response to an apprehension perturbation. This suppressed pectoralis major activation is consistent with other reported findings in the literature.[24] Bassett et al[9] used a biomechanical model to determine which shoulder muscles are most likely to provide anterior stability at the shoulder joint. The results indicated that flexors of the shoulder joint (pectoralis major, short head of the biceps, coracobrachialis, anterior deltoid, and the subscapularis) were the most effective in resisting an anterior dislocation.[9] As such, suppression of the pectoralis major may compromise anterior shoulder instability. Interestingly, McMahon and Lee[45] hypothesized that in a position of apprehension, the pectoralis major may contribute to joint instability rather than its commonly believed stabilization role. Others have also suggested that the pectoralis major is capable of acting as a destabilizer.[2,58] As such, the role of the suppressed pectoralis major contributing to either instability or an adaptation to curb anterior instability is an area of further investigation.

In addition to the suppressed pectoralis major activation, the biceps also exhibited decreased mean activation as well as an increased reflex latency. Previous work has demonstrated the importance of the biceps brachii in providing stability through its ability to depress the humeral head, increase the shoulder's resistance to torsional forces in the vulnerable abducted and externally rotated position, and diminish the stress placed on the inferior glenohumeral ligament.[35,51,54,67] Yet in patients with glenohumeral instability, this important stabilizer is suppressed and slower to respond to a perturbing episode, possibly contributing to the recurrent instability episodes experienced by this patient group.

Coactivation of the supraspinatus-subscapularis was suppressed in the patients with glenohumeral instability. Simultaneous contraction of the entire rotator cuff complex, in combination with the passive restraints, is responsible for centralization of the humeral head within the glenoid.[12,41] Yet the coactivation of the muscles that contribute to the anterior-posterior component of centralization is functioning less efficiently in the instability patients, possibly contributing to the recurrent instability episodes. The importance of normal function of the rotator cuff muscles to compress the humeral head has been suggested at end ranges of motion, including the apprehension position used in the current study.[38,43]

Unlike the suppressed muscle activation of the pectoralis major, biceps, and subscapularis-supraspinatus seen in the patients with instability, the rotator cuff muscles (supraspinatus, infraspinatus, and subscapularis) exhibited increased peak activation in response to the apprehension perturbation. It must be clarified that the coactivation of the rotator cuff is calculated from the mean activation (rather than the peak activation) and is an indicator of the activation relationship between 2 muscles. Thus, increased peak activation would have very little bearing on the coactivation that exists between 2 muscles. This increased peak activation may be a compensatory mechanism of the rotator cuff to provide the stability that has been compromised by the capsuloligamentous disruption seen in patients with instability resulting from acute, traumatic injury mechanisms.

Although the findings in this study, in combination with the current literature,[24,34,44] clearly demonstrate that mus-

cle activation abnormalities are present in patients with glenohumeral instability, questions remain about the source of these abnormalities. It is not known if these muscle activation abnormalities were present before the patients' suffering the instability episodes, thus contributing to the increased risk of instability, or if they manifested as a result of the instability episodes. To answer such questions, a true prospective study (before experiencing an instability episode for the first time) to identify abnormal muscle activation as a risk factor for injury would have to be initiated. To date, the literature is void of any such investigation. Although a study of this type has not been performed, many have hypothesized that neuromuscular control alterations manifest from capsuloligamentous joint injury. The consensus of the 45 scholars who participated in the 1997 workshop titled "The Role of Proprioception and Neuromuscular Control in the Management and Rehabilitation of Joint Pathology," sponsored by the AOSSMs Foundation for Sports Medicine Education and Research, was that with capsuloligamentous injury (ie, glenohumeral dislocation/subluxation), joint deafferentation (ie, decreased proprioception) develops that contributes to muscle neuromuscular control abnormalities (muscle activation alterations). Lephart and Henry[37] provided an overview of this joint deafferentation model. Addressing questions related to whether muscle activation alterations result from or contribute to joint instability would be a fertile area for future research.

The authors of the current study recognized several limitations with the apprehension model used. The position of application of the apprehension perturbation was abduction and external rotation in the scapular plane, not the true apprehension position. During pilot testing, patients with instability were uncomfortable when tested in the true apprehension position of abduction, external rotation, and horizontal abduction. Pilot work by the investigators comparing muscle activation characteristics in normal patients in both the position used for testing and a position that better mimics the apprehension position (abduction and external rotation in the frontal plane)[48] demonstrated no differences in muscle firing characteristics between the 2 positions. A second limitation recognized by the authors was that the perturbation applied only consisted of an external rotation force rather than the combination of external rotation–horizontal abduction commonly seen during instability episodes.[43] The uniaxial nature of our perturbation device (modified isokinetic dynamometer) as well as replication of previous literature[36,48,65] dictated that we use a model that limits perturbation to external humeral rotation.

Clinically, the results of this study may be used by physicians and therapists to understand the deficits that may exist in the unstable shoulder by implementing therapeutic exercise that can address such deficits in patients opting for conservative management, as well as in postsurgical rehabilitation after capsulorraphy procedures. For example, reflexive mean activation of the pectoralis major was suppressed in patients with glenohumeral instability. As a means of possibly restoring normal muscle reflexive firing, therapists can implement interventions such as rhythmic stabilization exercises that incorporate clinician-applied perturbation in varying directions to the upper extremity while the patient maintains limb position.[47] Arwert et al[3] demonstrated with electromyographic analysis that pectoralis muscle activation is increased when the upper extremity is placed in a position of 90° of elevation in the scapular plane (similar to the position of perturbation in the current study) and a medial, downward force is produced by the upper extremity. By using this biomechanical information by Arwert et al, a clinician could apply perturbation in an equal and opposite direction (up and outward force) to facilitate pectoralis major reflexive muscle activation.[47] Similarly, coactivation of the rotator cuff was suppressed in the current study. Fortunately, the clinical literature has demonstrated that coactivation is facilitated through closed kinetic chain exercises for the upper extremity.[27] Uhl et al have shown the effectiveness of several rehabilitations in eliciting simultaneous activation of the rotator cuff muscles, beneficial to restoring dynamic joint stability in glenohumeral instability patients. Although Uhl et al as well as others[4,11,18,46,63] have demonstrated the efficacy of some of the therapeutic exercises used by clinicians to elicit muscle activation, many of the exercises used on a daily basis are still deemed appropriate because of anecdotal evidence. Additional investigation to validate therapeutic exercises for reestablishing neuromuscular control over joint stability is a necessary area of future research.

CONCLUSIONS

In addition to the capsuloligamentous deficiency and proprioceptive deficits present in recurrent traumatic anterior instability, muscle activation alterations also are present. Patients with instability demonstrated suppressed pectoralis major and biceps brachii mean activation as well as increased peak activation of the subscapularis, supraspinatus, and infraspinatus compared to the control subjects. The instability patients also exhibited a significantly slower biceps brachii reflex latency than their control counterparts. Supraspinatus-subscapularis coactivation was significantly suppressed in the instability group. Shoulder muscle activation dysfunction like that seen in the current study can have dramatic effects on the resulting glenohumeral joint forces, thereby affecting joint stability and possibly contributing to the recurring episodes of instability commonly seen in patients with glenohumeral instability.

ACKNOWLEDGMENT

The authors wish to thank Gamal Zayed, MD, and Ali Engin, MD, for their assistance with the fine wire electromyography assessments. Financial support was provided by the Mrs Ho-Tung Cheong Research Grant through the Orthopaedic Research Laboratory Alumni Association.

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