

Shoulder Muscle Reflex Latencies Under Various Levels of Muscle Contraction

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Previous research in relaxed muscles shows that muscle reflex latencies are too slow to protect the shoulder. However, during athletic activity when injury occurs, some level of shoulder muscle contraction typically exists. The purpose of the current study was to assess shoulder muscle reflex latencies under various levels of muscle contraction. Seventeen healthy subjects participated. A perturbation consisting of an external rotation collision force to the anterior forearm in a position of apprehension under various levels of muscle contraction (0%, 20%, and 50% of a maximum voluntary muscle contraction) was applied. Muscle reflex latencies were measured as the

time from perturbation application to onset of muscle activity. Electromyography measured activity onset of the rotator cuff muscles and the primary humeral movers. During 0%, the latissimus dorsi muscle reflex latency was significantly slower than most other muscles. No difference existed between muscles in the 20% and 50% conditions. For the rotator cuff muscles, the reflex latencies significantly quickened for 20% and 50% compared with the relaxed state (0%). Overall, introducing muscle contraction significantly quickened muscle reflex latencies. These results provide clinicians with a better understanding of the role that these reflexes play in joint stability in a position of injury vulnerability like a position of apprehension.

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The shoulder has been described as a joint with a high level of mobility necessary for placing the hand in a position of function.¹ Because of this level of mobility, inherent stability is compromised. Specifically the shoulder relies on static and dynamic restraints. Static restraints include osseous geometry, negative intraarticular pressure, the glenoid labrum, and capsuloligamentous restraint.³⁴ Dynamic restraints refer to the joint stability provided by muscles that cross the shoulder through mechanical and neuromuscular mechanisms. Although described as separate entities (static versus dynamic) the shoulder relies on the

neurologic interaction between these two mechanisms for joint stability.³¹

Research has shown that a reflexive arc exists between the capsuloligamentous structures and muscles about the shoulder. Several investigators showed that a spinal reflex exists between the joint capsule and musculature surrounding the feline glenohumeral joint using electrical stimulation of the joint capsule.^{17,26,39} Jerosch et al²¹ followed up the feline model research by arthroscopically showing a similar reflex arc existing between the shoulder capsule and the deltoid, trapezius, pectoralis major, and rotator cuff musculature in humans. Stimulation of the shoulder capsule has a direct effect on the alpha motor neurons that innervates the shoulder musculature. Using a joint perturbation to better simulate what happens during injury mechanisms, Latimer et al³⁰ measured muscle latencies of the shoulder resulting from an anterior translation force. Reflex latencies ranged from 110 to 220 ms. Overall, anterior muscles fired first followed by the posterior muscles. Being that the shortest latency was approximately 110 ms, the authors concluded that the reflexive responses are too slow to protect the joint during a traumatic instability episode. One limitation with that study was that shoulder reflex latencies were assessed while the muscles were in a relaxed state. These reflexes may be altered significantly when some level of muscle activity is present during elicitation of the reflex. It has been suggested that some level of underlying muscle contraction might increase the muscle spindles sensitivity to intramuscular length changes, quickening the reflexive response of the muscle.^{2,32,38} Therefore in addition to the capsuloligamentous tissue having an effect on the alpha motor neuron, coactivation of the gamma motor neuron innervating the muscle spindle also exists.²²⁻²⁴ During athletic activity, some level of muscle contraction exists whether from throwing a ball or making a tackle. Assuming that gamma motor neuron activation heightens muscle spindle sensitivity, one could expect reflexes seen during athletic activity to be quicker than during a relaxed state. The purpose of the cur-

rent study was to measure shoulder muscle reflex latencies during various levels of muscle contraction. These muscle reflexes were assessed in a position of apprehension (the combination of shoulder abduction and external rotation) where the shoulder is most vulnerable to injury.¹⁸

METHODS

Research Design

Seventeen healthy subjects (gender, 10 males, seven females; age, 23.76 ± 4.10 years, height, 172.41 ± 9.22 cm; and weight, 75.80 ± 15.47 kg) participated. No subject had a history of shoulder instability, upper extremity injury, or other ailments that could have altered sensory input or motor function or both such as insulin dependent diabetes, rheumatologic disorders, or peripheral nerve disorders. All subjects attended one testing session where the subject's dominant limb was assessed. Dominance was defined as the limb in which one would throw a ball. All subjects provided informed consent approved by the institutional review board before participation. Subjects then were prepared for assessment and tested with the procedures described below.

Electromyography

Electromyography data were collected with the Noraxon Telemetry (Noraxon, Scottsdale, AZ) frequency modulated (FM) telemetry electromyography system. With this system, all electromyographic signals collected from the electrodes were passed through a single-ended amplifier (gain 500) to an eight-channel FM transmitter. A receiver unit obtained the telemetry signals and then 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) A/D board (ComputerBoards, Middleboro, MA) at a rate of 1000 Hz. The digital data were collected and stored with Myoresearch 2.02 (Noraxon) software on a personal computer for later data reduction.

Shoulder Apprehension Perturbation

Device

The shoulder apprehension perturbation device consisted of a modified Biodex System III isokinetic dynamometer (Biodex Medical, Shirley, NY)



Fig 1. A subject positioned in the shoulder apprehension perturbation device shows subject setup and instrumentation.

and lever arm (Fig 1). The lever arm consisted of securing a 500 lb compression load cell (Model 41, Sensotec, Inc, Columbus, OH) in series with the modified Biodex isokinetic lever arm. The voltage from this load cell was used to determine perturbation onset. 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 electromyography data in the Myoresearch software. The dynamometer chair was fitted with a pad that supported the upper limb just distal to the axilla, thereby assisting with subject position. A TLL-500 (Transducer Techniques, Inc, Temecula, CA) tension-only load cell was used to monitor isometric internal rotation muscle contraction with the Myoresearch software and a customized program written by the investigators in LabVIEW 5.1 (National Instruments, Austin, TX) programming language. The LabVIEW program signaled through audible tones when the desired percentage of the isometric internal rotation maximum voluntary contraction was achieved for each trial.

Testing Procedures

Dual fine electrodes constructed with .05-mm nickel chromium alloy wire insulated with nylon (California Fine Wire Company, Grover Beach, CA) were prepared.^{4,5} Indwelling electrodes were inserted intramuscularly via a 1.5-inch 25-gauge

needle into the supraspinatus and infraspinatus as described by Geiringer.¹⁵ Electrodes were inserted with a 1.5-inch, 25-gauge needle into the upper and lower portions of the subscapularis as described by Kadaba et al.²⁵ Two portions of the subscapularis were measured to show differences between upper and lower portions in innervation and function depending on humeral position.²⁵ In addition differences in reflex latencies are reported between the upper and lower portions.³⁰ Insertion sites were sanitized using 70% isopropyl alcohol and an iodine solution.

Silver-silver chloride surface electrodes (Medicotest, Inc, Rolling Meadows, IL) were used for measurement of superficial muscle activity. Skin preparation to lower impedance included shaving any hair present, mild abrasion with a low abrasive emery board, and wiping the area with 70% isopropyl alcohol. Two surface electrodes were placed side by side with 1 cm separating the centers of the electrode.¹² The electrodes were placed parallel to the orientation of the muscle fibers.¹² Surface electrodes were placed on the sternal portion of the pectoralis major, anterior deltoid, latissimus dorsi, and biceps brachii as described by Basmajian and Blumenstein.³ A ground electrode was placed on the olecranon process of the elbow. Correct positions of all electrodes were confirmed through isolated manual muscle tests of each muscle.³

The subjects were in a seated position on the isokinetic dynamometer chair, reclined 5° from vertical. The subject's dominant limb was positioned and supported at 90° abduction. A fixed hinge brace (ROM Elbow Deluxe, DonJoy Orthopedics, Vista, CA) maintained the elbow at 90° flexion. A rigid wrist splint (Universal Elastic Wrist Splint, DonJoy Orthopedics, Vista, CA) maintained neutral position of the wrist. Each subject was asked to externally rotate as far as possible without compensatory motion maintaining the 90° abducted position. An electronic range of motion (ROM) stop from the Biodex System III was set for that position. The ROM stop protected the subject from achieving ROM outside an active range. A position of 35° before end range then was determined from the internal electrogoniometer present in the Biodex System III. This was the location within the ROM where the perturbation was applied. By positioning the subject at -35° from end range, a window of 20° at a constant dynamometer velocity was achieved for standardization of the speed at perturbation. This was necessary because of deceleration characteristics of

the dynamometer as its lever arm reaches the set end range stop.

Before initiation of perturbation trials, each subject did three maximum internal rotation isometric voluntary contractions at the perturbation application position. The computer program written in LabVIEW software collected the tension load cell data for each of the three trials and calculated a mean maximum voluntary contraction from those contractions. The mean isometric internal rotation maximum voluntary contraction was used to calculate the percentage maximum voluntary contraction for each trial.

Before each trial, the investigator positioned the subject 35° from active end range of external humeral rotation. Each trial began by instructing the subject to produce an isometric internal rotation contraction. This force of contraction occurred at one of three levels (0%, 20%, or 50% of the isometric internal rotation maximum voluntary contraction). The correct level of contraction was signaled to the subject through audible tones within a set of headphones by the LabVIEW program. Once the level of contraction was achieved and held constant, the investigator initiated the shoulder perturbation. Visual indication of contraction level was provided immediately to the investigator to insure correct contraction level at perturbation application. Level of contraction again was verified later in the data reduction process. The perturbation consisted of the lever arm striking the limb at 180°/second while the subject maintained the correct percentage of contraction. Subjects were instructed not to intervene with the perturbation.¹¹ Visual, auditory, and tactile cues were eliminated with a blindfold, headphones, and low intensity vibration provided by an air compressor. Each subject did six counterbalanced trials at each level of muscle activation. Approximately 30 seconds of rest were provided between trials to control for any fatigue effects. Three practice trials were provided to the subjects before initiation of the collection trials.

Data Reduction

All data including the electromyograms from the eight muscles and load cell data were exported from Myoresearch as a spreadsheet. A separate LabVIEW program written by the investigators rectified all electromyograms and filtered the electromyogram, and both load cell data. Electromyogram data were band pass filtered 20 to 500 Hz using a fourth order Butterworth zero-phase shift

filter similar to recommendations set forth by the Journal of Electromyography and Kinesiology. Analog signals from the load cells were filtered using a 20 Hz, fourth order Butterworth low-pass zero-phase shift filter.^{37,38,43}

The filtered data files were reduced additionally with custom software written in Visual Basic for Applications (Microsoft Inc, Redmond, WA) programming language. All reduction procedures are described below.

Perturbation onset was determined by first finding the maximum force value from the compression load cell data. The mean and standard deviation of a 200 ms linear envelope from 550 to 350 ms before that maximum value was calculated. The perturbation onset threshold was calculated as the mean of the linear envelope plus three times the standard deviation.^{9,13,19} This 200 ms window 350 ms before the maximum force value was used to ensure that mean and standard deviation used for calculation of perturbation onset threshold was derived from compression load cell data before perturbation onset. The onset of perturbation was determined by comparing compression load cell data points in a point-by-point fashion until the perturbation onset threshold criterion was met. Once the perturbation onset threshold criterion was met, onset was considered at that data point (Fig 2).

From the point of perturbation onset, the mean amplitude of a 150 ms linear envelope from the tension load cell was compared with the desired level of contraction (0%, 20%, or 50% of the mean of the three isometric internal rotation maximum voluntary contractions taken before trial collection). If the level of contraction at 150 ms before perturbation onset met the desired level ($\pm 5\%$) the trial was included in the database. Otherwise, the trial was discarded because an appropriate level of contraction was not present, possibly altering the results. A minimum of four trials was used for statistical analysis.

The muscle reflex latencies were calculated for each of the eight muscles tested. Each muscle reflex latency was calculated as the time between perturbation onset and muscle activity onset (Fig 2). Muscle activity onset was determined by calculating the amplitude mean and standard deviation of the rectified, filtered electromyographic data in the interval between 150 ms before perturbation onset. To determine onset the muscle voltage threshold was calculated. Muscle voltage threshold was calculated as the mean of the linear envelope 150 ms

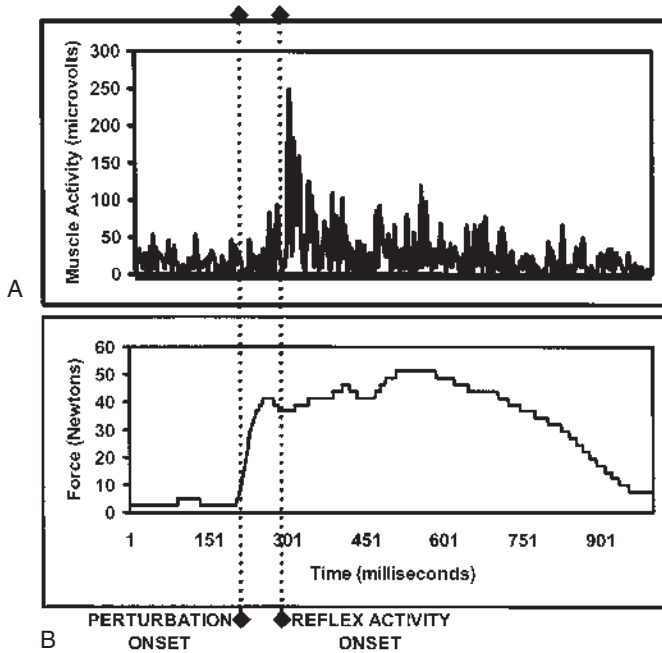


Fig 2A–B. (A) Electromyography and (B) compression load cell data used for calculation of muscle reflex latencies are shown. Muscle reflex latency is defined as the period from perturbation onset to muscle activity onset.

before perturbation plus three times the standard deviation.^{9,13,19} Onset of muscle activity was determined in a point-by-point fashion until the muscle voltage threshold was met.^{9,13,19} Because the data were sampled at 1000 Hz, latency was calculated by counting the data points between perturbation onset and reflex muscle activity onset with each point representing 1 ms.

Data Analysis

All data were analyzed using fixed effects repeated measures analysis of variance (ANOVA) models. An a priori alpha level of .05 was set before data collection and was considered the level of significance. A two within factor repeated measures ANOVA was done on SPSS 9.0 (SPSS, Chicago, IL) statistical software. The within factors were level of contraction (0%, 20%, 50%) and muscle (eight muscles tested). When applicable, Tukey's honestly significant difference post hoc analyses were used for pairwise comparisons.

RESULTS

The descriptive statistics for all shoulder muscle reflex latencies under the three levels of

contraction appear in Table 1. Statistical analysis of the muscle reflex latency data revealed a significant muscle by condition interaction ($F(14,224) = 4.983$; $p < .0001$). Comparison among muscles in each level of contraction revealed the latissimus dorsi reflex latency to be significantly slower than the biceps brachii, pectoralis major, anterior deltoid, lower portion of the subscapularis, and supraspinatus (Fig 3). No differences existed between any muscles in 20% and 50% conditions. Comparisons in muscles among levels of contraction yielded results showing that the latissimus dorsi was significantly faster during the 20% and 50% conditions compared with 0% (Fig 4). In addition, all rotator cuff muscles (upper subscapularis, lower subscapularis, supraspinatus, and infraspinatus) showed significantly quicker muscle reflex latencies during the 20% and 50% conditions compared with the 0% condition (Fig 5). A significant condition main effect also was present ($F(2,32) = 71.793$; $p < .0001$) indicating that in general, the muscle reflex latencies were significantly slower for 0% than the 20% and 50% conditions (Fig 6). No significant

TABLE 1. Descriptive Statistics (Mean ± Standard Deviation) for Muscle Reflex Latencies*

Muscle	Level of Contraction		
	0%	20%	50%
Pectoralis major	92.7 ± 27.6	69.1 ± 15.5	81.4 ± 16.2
Biceps brachii	79.4 ± 16.9	71.7 ± 11.8	76.8 ± 14.4
Anterior deltoid	101.9 ± 24.8	80.9 ± 22.8	88.9 ± 22.3
Latissimus dorsi	133.1 ± 33.4	63.9 ± 16.6	78.4 ± 19.9
Upper subscapularis	110.4 ± 21.5	63.9 ± 19.2	70.9 ± 20.8
Lower subscapularis	108.3 ± 29.4	68.8 ± 19.5	79.7 ± 19.3
Supraspinatus	100.9 ± 30.6	70.5 ± 16.5	70.3 ± 22.3
Infraspinatus	109.6 ± 29.3	67.5 ± 16.1	70.6 ± 21.9

*in ms

Fig 3. Shoulder muscle reflex latencies in the relaxed muscle condition (0%) are shown. * The latissimus dorsi (LD) reflex latencies were significantly slower than the biceps brachii (BB), pectoralis major (PM), anterior deltoid (AD), lower portion of the subscapularis (LS), and supraspinatus (SS).

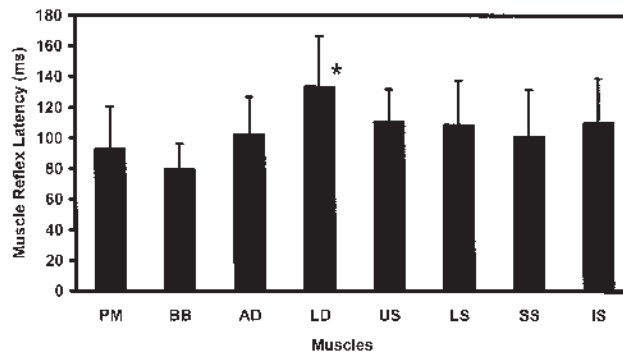
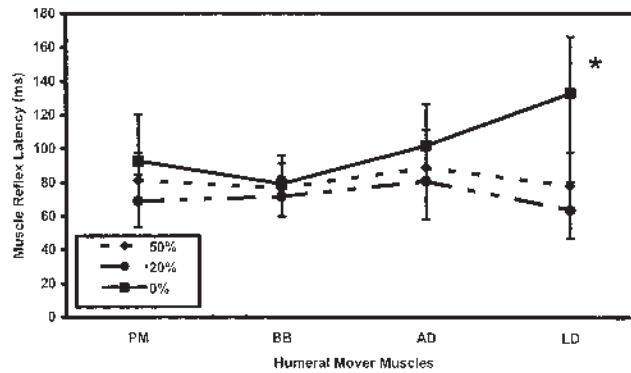


Fig 4. Muscle reflex latencies for the primary humeral movers are shown. *The muscle reflex latencies for the 0% condition were significantly slower compared with 20% and 50%. PM = pectoralis major, BB = biceps brachii, AD = anterior deltoid, LD = latissimus dorsi



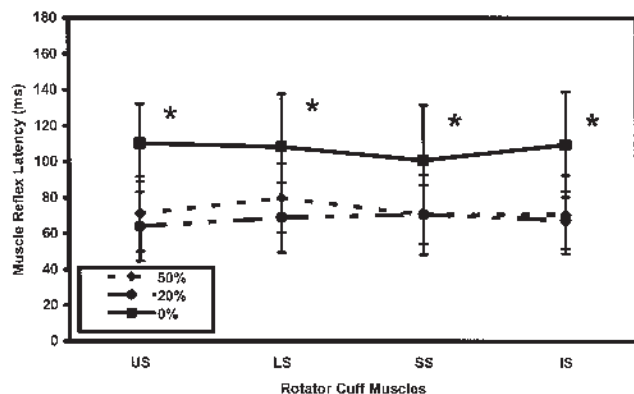


Fig 5. Muscle reflex latencies for the rotator cuff muscles are shown. *The muscle reflex latencies for the 0% condition were significantly slower compared with 20% and 50%. US = upper subscapularis, LS = lower subscapularis, SS = supraspinatus, IS = infraspinatus

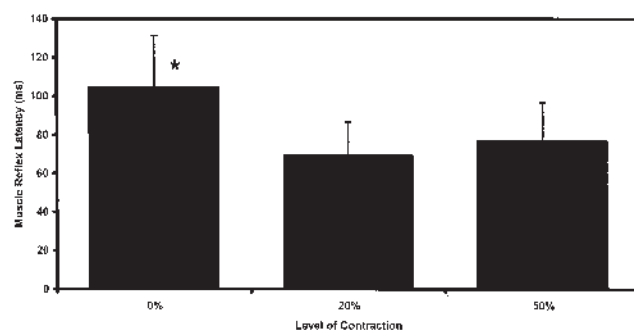


Fig 6. The condition main effect shows that muscle reflex latencies are significantly slower in a relaxed state compared with contracted states. *The muscle reflex latencies for the 0% condition were significantly slower than the 20% and 50% conditions.

differences existed between the 20% and 50% conditions.

DISCUSSION

Results from the current study indicate that the induction of muscle contraction has a significant effect on muscle reflex latency. Significant quickening of the muscle reflex latencies were seen in the latissimus dorsi, upper and lower subscapularis muscles, supraspinatus, and infraspinatus when comparing the reflex latencies obtained during no contraction (0%) and contraction levels of 20% and 50% of a maximum contraction (Figs 4, 5). In addition, the condition main effect that manifested (Fig 6) showed quickening reflexes under the contraction conditions compared with the relaxed condition. It is thought that this decrease in reflex latency results from increased gamma motor neuron coactivation. This induction of muscle contrac-

tion increases the sensitivity of the muscle spindle to intramuscular length changes.^{2,32,38} A more sensitive muscle spindle would detect the stretch caused by perturbation quicker, eliciting a faster reflexive response.

The significant results with the latissimus dorsi and subscapularis seem logical given that the subjects were asked to produce an isometric internal rotation force and the role of these muscles is to produce an internal rotation moment. The quickening of the supraspinatus and infraspinatus (abduction and external rotation muscle functions) despite the voluntary internal rotation contraction done by the subjects suggests that these muscles were contracting synergistically with the subscapularis for centralization of the humeral head. This centralization of the humeral head within the glenoid fossa is a commonly described function of the rotator cuff muscles.^{14,20,42} Only latencies were measured in the current study and amplitude of muscle

contraction was not assessed to confirm this suggestion. Additional research is needed to better understand all muscle reflex characteristics, not just reflex latency.

Unlike the ankle and knee, the scope of knowledge concerning reflex activity at the shoulder is limited, making comparisons among results of the current study and other results difficult. Several investigators showed the existence of a spinal reflex between fibrous joint capsule and musculature about the feline glenohumeral joint.^{17,26,39} Jerosch et al²¹ followed up the feline model research by arthroscopically showing a similar reflex arc between the shoulder capsule and the deltoid, trapezius, pectoralis major, and rotator cuff musculature in a human model. The latencies reported were extremely fast (3–5 ms) compared with the latencies presented in the current study (Table 1). The methods reported in these investigations elicited reflexes with electrical stimulation rather than in vivo perturbation assessment making comparison difficult. The importance of electrical stimulation investigations was the establishment of a pathway between the capsuloligamentous structures and musculature about the shoulder. Unfortunately, making clinical application with reflex latencies elicited by electric stimulation is difficult because this method does not reflect physiologic events.³⁶

The current authors are aware of only three publications that use an in vivo perturbation model to assess reflex latency at the shoulder.^{9,30,41} Wallace et al⁴¹ compared reflex latencies in unstable subjects, with similar reflex latencies with the current study. Muscle assessment was limited to the pectoralis major and infraspinatus. Brindle et al⁹ assessed reflex latency with an internal rotation perturbation in normal subjects and intercollegiate pitchers. They reported decreases in latencies in the pitchers compared with healthy subjects suggesting adaptation in elite overhead athletes. Despite using a different direction of perturbation, the latencies reported for the normal group mimic the results in the current study (ranging from 70–100 ms). Finally, Latimer et al³⁰ assessed reflex latency in response to an external

rotation apprehension perturbation. The results reported by these authors tended to be higher compared with the latencies currently reported. Methodologic differences are most likely the result of the inconsistencies. Latimer et al³⁰ reported that the perturbation application used (specifically the time between the rope release of the device and capsular tightening) might have inflated the latency results.

The results from the current study provide clinicians with a better understanding of how the muscles about the shoulder respond to injury situations (perturbation in a position of apprehension). This data provide latency values for future comparisons between healthy and pathologic shoulders. The current authors are aware of only one study that measured reflex latencies in unstable shoulders.⁴¹ No differences were reported between stable and unstable shoulders with the two muscles (pectoralis major and infraspinatus) assessed. Additional research is needed to assess reflex latencies in all muscles about the shoulder in subjects with stable and unstable shoulders. Alterations in muscle reflex latencies at the shoulder like those reported at the knee or ankle^{6,7,44,28,29} may be indicative of sensorimotor deficits contributing to shoulder instability. Assessment of muscle reflex latencies after surgical intervention may provide clinicians with an indicator of sensorimotor restoration after surgery.

Although the reflex latencies when the muscle is in a contracted state are quicker, the question remains as to whether these reflexive responses are quick enough to protect the joint. An extensive review of the literature yielded no data concerning how quickly a dislocation or subluxation episode occurs at the shoulder. Pope et al³⁵ described reflex activity at the knee as being too slow to protect the joint. They reported that knee ligament failure occurs approximately 73 ms after force application, suggesting insufficient protection by reflexes. Buchanan et al¹⁰ reported similar results, showing that the perturbation ceases before initiation of the reflexive response.

Electromyography measures the myoelectrical events associated with muscle contrac-

tion, not force production.⁸ An electromechanical delay exists. This delay is defined as the time between the onset of the electromyographic signal and the onset of biomechanical force.^{16,33,40} Electromechanical delay for involuntary muscle contractions like reflexes ranges from 21 to 66 ms in relaxed muscle.⁴⁰ As such, reflex latencies may be an underestimate of the force production response to a perturbation. This force production is the important component of joint stability.²⁷ Given the slowness of reflex latencies, other factors must be associated with shoulder stability.

The results of the current study indicate that shoulder muscle reflex latencies significantly quicken when they occur when some level of muscle contraction is present. Although these reflexes are quicker than previously reported latencies in the relaxed shoulder, questions remain whether the reflexes are fast enough to protect the joint. A better understanding is needed regarding the role these reflexes play in joint stability and how these reflexes are altered in pathologic shoulders.

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