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Suprascapular nerve block results in a compensatory increase in deltoid muscle activity

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Abstract

A balance exists between the deltoid and rotator cuff contribution to arm elevation. Both cadaver and computer models have predicted an increase in deltoid muscle force with dysfunction of the rotator cuff. The goal of the present study was to verify this phenomenon in vivo by examining the effects of paralysis of the supraspinatus and infraspinatus muscles with a suprascapular nerve block on the electrical activity of seven shoulder muscles. Electromyographic data were collected before and after the administration of the block. The block resulted in a significant increase in muscle activity for all heads of the deltoid, with a higher percentage increase noted at lower elevation angles. Although the deltoid activity was reduced as the subjects recovered from the block, even low levels of cuff dysfunction were found to result in increased deltoid activity. These results suggest that even small disruptions in the normal function of some rotator cuff muscles (e.g., due to fatigue or impingement syndrome), may result in an increase in deltoid activity. It is possible that such compensation may result in higher superior loads at the glenohumeral joint, possibly increasing the risk of tendon damage.

Keywords: Shoulder; Glenohumeral; EMG; Nerve block; Rotator cuff

1. Introduction

During normal shoulder function, there exists a delicate balance between the forces exerted by the deltoid and rotator cuff muscles. Weiner and MacNab (1970) first suggested that an impairment of this balance was responsible for the observed decrease in subacromial space width associated with rotator cuff tears, both in vivo (Golding, 1962; Weiner and Macnab, 1970) and in cadavers (Cotton and Rideout, 1964). More recently, several X-ray studies have demonstrated that patients with full-thickness rotator cuff tears exhibit greater superior translation of the humeral head when compared to healthy controls (Deutsch et al., 1996; Paletta et al., 1997; Yamaguchi et al., 2000; Bezer et al., 2005). Additionally, simulated paralysis (Sharkey and Marder, 1995; Hurschler et al., 2000) and tears (Thompson et al., 1996; Halder et al., 2001; Mura et al., 2003) of the rotator cuff muscles in a cadaver model have also consistently resulted in an increase in humeral head superior migration.

It has been established in cadaver (Sharkey et al., 1994; McMahon et al., 1995) and computer (Magermans et al., 2004) models that reducing the contribution of the rotator cuff muscles places a higher demand on the deltoid. While the elevation moment arms of the middle deltoid and supraspinatus are very similar (Liu et al., 1997), the line of action of the deltoid is directed more superiorly. Consequently, this increase in deltoid force results in a more superiorly directed joint reaction force at the glenoid in a cadaver model (Parsons et al., 2002). This unbalanced superior force is believed to be responsible for the above-mentioned superior translation. Without proper regulation, this can result in a positive feedback loop, where damage leads to increased translation that leads to even further damage. This theoretical increase in deltoid activity has not been demonstrated in vivo.

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Although the rotator cuff consists of four muscle-tendon units, the supraspinatus and infraspinatus are the most commonly injured tendons (Sher, 1999; Matsen et al., 2004). Previous work in our laboratory has focused on simulated rotator cuff dysfunction with a fatigue model (Tsai et al., 2003; Ebaugh et al., 2006). However, one of the key problems with this approach is that it is very difficult to isolate the rotator cuff musculature. Since the suprascapular nerve innervates both the supraspinatus and infraspinatus, a pharmacological block of this nerve offers an appropriate model to better understand rotator cuff dysfunction (Colachis and Strohm, 1971; Howell et al., 1986; Kuhlman et al., 1992; Werner et al., 2006b). The aim of this study was to examine the effect of a suprascapular nerve block on shoulder muscle activity. We hypothesized that this block would result in a compensatory increase in deltoid activity, similar to what has been observed in cadaver models.

2. Methods

This is a companion study to a detailed kinematic analysis recently published in Clinical Biomechanics (McCully et al., 2006). Details regarding subjects, kinematics and strength records can be found there and are summarized here.

2.1. Subjects

Fifteen subjects with no history of cervical or shoulder pain or pathology participated in this study (age range 20–33 years). There were seven females and eight males, with a mean age of 26 ± 4 years, a mean height of 174 ± 9 cm and a mean mass of 70 ± 10 kg. The dominant shoulder of each subject was tested. Approval for this study was obtained from the Institutional Review Board of the University of Oregon and informed consent was obtained from all subjects.

2.2. Kinematic measurements

A Polhemus 3Space Fastrak (Colchester, VT) was used for collecting three-dimensional kinematics of the humerus with respect to the thorax. The Fastrak transmitter was attached to a vertical stand at a distance of 50 cm behind the subject. A receiver was placed on T3 using Spirit Gum adhesive and Micropore tape. Another receiver was mounted on a custom made cuff molded from Polyform splinting material (Sammons Preston Rolyan, Bolingbrook, IL, USA) and positioned on the distal humerus. The final receiver was positioned over the scapula after mounting it on a custom made and previously validated scapular-tracking device machined from plastic (Karduna et al., 2001). The only purpose that the scapular receiver served for the present study was to help locate the center of the humeral head (see below). Although, the accuracy of the receiver orientation is 0.15° as defined by the manufacturer, skin motion artifact would be expected to result in even higher errors. However, previous bone-pin validation study has documented skin motion artifacts of less than 6° for humeral motion (Ludewig et al., 2002).

For consistency with other studies, anatomic axes were derived from the digitization of anatomical landmarks as proposed by the shoulder sub-committee of the International Society of Biomechanics committee for standardization and terminology (Wu et al., 2005). On the thorax, the seventh cervical vertebra, eighth thoracic vertebra, sternal notch, and xiphoid process were digitized, while on the humerus, the medial epicondyle, lateral epicondyle, and center of humeral head were digitized (Fig. 1). With the use of a least-squares algorithm, the center of the humeral head was determined as the point on the humerus that moved the least with respect to the scapula when the humerus was moved throughout short arcs of mid-range glenohumeral motion (Harryman et al., 1990). Humeral rotations were represented using an Euler angle sequence of plane of elevation, followed by amount of elevation, followed by internal and external rotation (An et al., 1991).

2.3. Electromyographic measurements

A Myopac Jr. (RUN Technologies, Mission Viejo, CA) unit with seven dual lead channels was used for collection and processing of electromyographic (EMG) recordings from superficial shoulder musculature (Fig. 2A). EMG activity was recorded from the upper and lower trapezius; anterior, middle, and posterior portions of the deltoid; serratus anterior; and infraspinatus. Initial identification of muscle locations was determined based on the recommendations set forth by Cram et al. (1998), with subject motion and manual palpation as the final determinate for electrode placement. Surface EMG was recorded using a bipolar lead with two pediatric electrodes (Blue Sensor, Denmark) located 3.4 cm apart (center-to-center distance), positioned parallel to the primary muscle fiber alignment. A single lead grounding electrode was placed on the dominant side clavicle for signal noise reduction. Since these were passive electrodes, there was no pre-amplification of the EMG signal. The system has a common mode rejection ratio of at least 90 dB, an amplifier input impedance of 10 MΩ and a band-pass filter (10–1000 Hz). Data were collected at a sampling rate of 1200 Hz. The data were run through a root mean square (rms) algorithm with a window of 50 ms, which served to rectify and low pass filter the data.

In order to normalize EMG activity levels during arm elevation, maximum voluntary contractions (MVC) of the muscles were obtained during 5-s contractions, with the amplitude of the contraction being determined as the average value of the rms data over the middle 2 s of the contraction. One MVC was performed for each muscle and there was a rest period of approximately 2 min between testing on different muscles. The following test positions and procedures were employed: upper trapezius and middle deltoid—90° of shoulder abduction with the elbow flexed

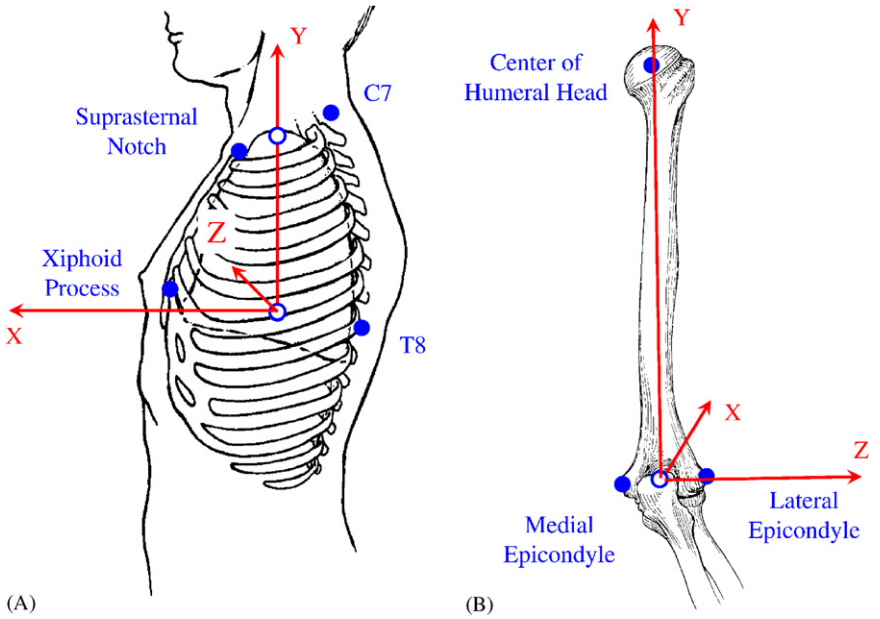

Fig. 1. Anatomical landmarks (closed circles) and intermediate points (open circles) used to determine the (A) thoracic and (B) humeral coordinate systems in accordance with the ISB recommendation (Wu et al., 2005).

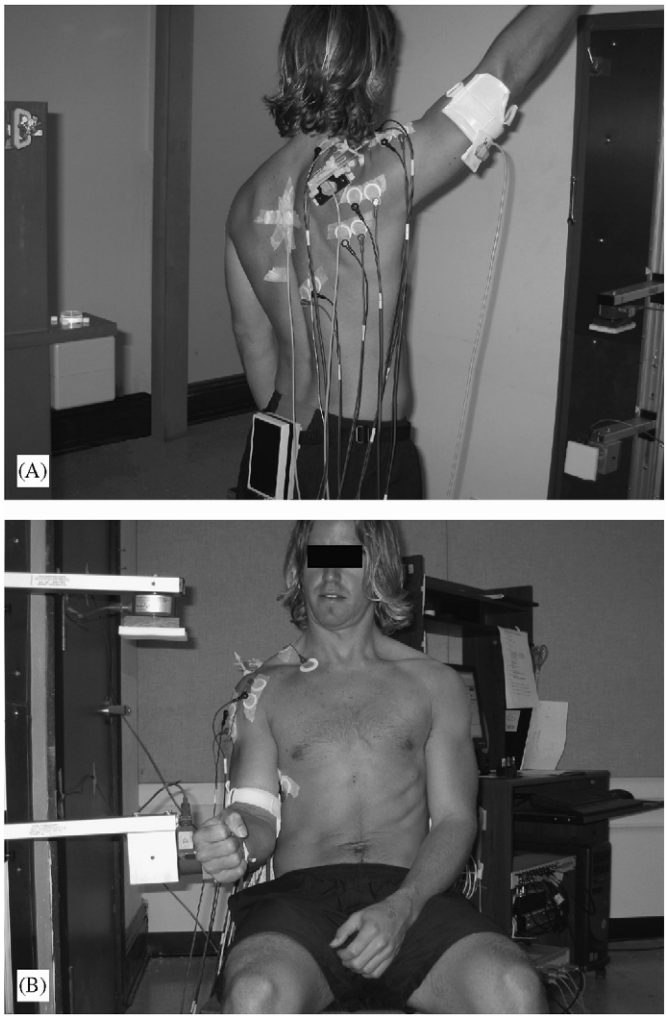

Fig. 2. (A) Placement of EMG and Polhemus sensors. (B) Positioning for isometric force assessment.

90° and the forearm parallel to the floor, resisted abduction (Alpert et al., 2000); anterior deltoid—90° of humeral flexion with the elbow flexed 90° and the forearm vertical, resisted flexion (Maffet et al., 1997); posterior deltoid—90° humeral abduction, elbow flexed 90° with forearm parallel to the floor, resisted horizontal extension (Alpert et al., 2000); lower trapezius—90° of humeral elevation in the scapular plane, elbow fixed at 90°, subject depressing and downwardly rotating the scapula (Kendall et al., 1993); serratus anterior—humerus abducted 90°, internally rotated 90°, humerus horizontal flexion movement (Decker et al., 1999); infraspinatus—90° of humeral elevation in the scapular plane, elbow fixed at 90°, with 30° of internal rotation (Kelly et al., 2000).

Both kinematic and EMG data were collected and analyzed with LabView (National Instruments, Austin, TX). The data from the serial port (kinematics) and A/D board (EMG) were collected simultaneous with Labview and were synchronized with a time stamp.

2.4. External rotation strength measurements

Shoulder external rotation force was measured with a 3390-50, 50 lb (22.7 kg) compression load cell (Lebow, Troy, MI). Subjects were seated and the height of the load cell was adjusted to that of the dorsal side of the subject's hand when the arm was at the side, in neutral shoulder rotation with the elbow in 90° of flexion (Fig. 2B). The subject exerted a maximal shoulder external rotation torque for 3 s. The average force produced during the middle 1-s period of each trial was recorded. Only subjects in whom there was a reduction of at least a 50% in external rotation force from baseline were included (Colachis and Strohm, 1971; Kuhlman et al., 1992).

2.5. Suprascapular nerve block

The nerve block was performed by a Board Certified Anesthesiologist (PK). After sterile prep of the skin, local anesthetic was infiltrated at a point 2 cm above the scapular spine and at the junction of the outer and middle one-third of the spine. A 22 gage 5 cm insulated nerve stimulator needle was advanced to the scapular notch with 0.6 mA of current at 2 Hz (Stimuplex-Dig 'Nerve stimulator for plexus anesthesia' B. Braun Medical Inc. Bethlehem). When motor stimulation was seen at current of less than 0.2 mA, 1.5 ml of 1.5% lidocaine was injected. Once repeat stimulation at 0.8 mV did not result in any muscle activity, the remaining 5.7 ml of 1.5% lidocaine (total 100 mg) were injected and the needle was removed. In one subject, the notch was not identified, but stimulation confirmed proximity to the suprascapular nerve. Ten minutes following needle withdrawal, data collection resumed.

2.6. Testing protocol

Following a standardized warm-up procedure (McCully et al., 2006), subjects were asked to stand while elevating their shoulder in the scapular plane. Plane was confirmed via on-screen visual feedback from the magnetic tracking device. Shoulder elevation trials were collected with the elbow in full extension and thumb pointing upward. While all trials began with the arm at the subject's side, the maximum elevation angle achieved was subject dependant.

Elevation and depression of the arm took a total of approximately 8 s, with three shoulder elevations and depressions constituting one complete trial. One trial was collected prior to the nerve block and nine trials were collected following the nerve block. External rotation strength measurements were collected immediately following each completed kinematic trial. Subjects were given a 5-min rest period between trials.

2.7. Data reduction and analysis

As an assessment of success of the block, the percent reduction in external rotation force due to the block was calculated. For each trial, EMG data were interpolated in 10° increments of humerothoracic elevation and averaged over the three elevations. Statistical tests were performed on the common range of motion achieved by all subjects under all conditions (humeral elevation angles from 20° to 120°). For each muscle, a two-way repeated measures analysis of variance (ANOVA) was performed with two within subject factors (elevation angle and block). If there was a significant effect of the block and a significant interaction between the two factors, follow-up paired *t*-tests were run at each humeral elevation angle. The alpha level was set at 0.05 for all analyses.

3. Results

Only 10 subjects were included for the purposes of data analysis. In four subjects the reduction in external rotation force was less than the 50% threshold and one subject could not elevate her arm without assistance after the block.

For the glenohumeral muscles, there was a significant increase in activation after the block for the anterior deltoid ($p = 0.001$), middle deltoid ($p < 0.001$) and posterior deltoid ($p = 0.002$), but no significant effect for the infraspinatus ($p = 0.452$). Follow-up *t*-tests for the deltoid muscles indicated that there was a significant increase in muscle activation at all humeral elevation angles except for 20° of elevation for the posterior deltoid ($p = 0.072$) (Fig. 3). For the scapulothoracic muscles, there was a significant effect of the block on the activation of the lower trapezius ($p = 0.032$), but no significant effect on the upper trapezius ($p = 0.061$) and serratus anterior ($p = 0.424$). Follow-up *t*-test for the lower trapezius indicated that there was a significant decrease in muscle activation at humeral elevation angles of 40° ($p = 0.001$) and 80° ($p = 0.030$) (Fig. 4).

The mean external rotation force immediately after the nerve block was 25% of the pre-block measurement. Subsequently, there was a linear force recovery, which was still significantly lower than baseline until the last trial, which took place approximately 75 min after the nerve block (Fig. 5A). As an indicator of muscle activation recovery, we averaged the deltoid data across all elevation angles (20–120°) and then again across all subjects. This gave us a representative muscle activation for each trial. These data were then normalized by the pre-block data and plotted vs. trial number. A gradual recovery in deltoid activity was noted, which was still higher than baseline at the last trial (Fig. 5B).

4. Discussion

Since the pioneering work of Inman et al. (1944) over 60 years ago, numerous investigators have attempted to further our understanding of shoulder biomechanics with the use of EMG. Surprisingly, we could only identify a few articles that measured EMG in patients with rotator cuff tears (Kido et al., 1998; Fokter et al., 2003; Hoellrich et al., 2005; Kelly et al., 2005), with no studies comparing deltoid activation to healthy controls.

In order to compare our results to other models of cuff dysfunction, we analyzed the data from two studies that simulated paralysis of cuff muscles with a cadaver model. Both models incorporated four simulated muscles: deltoid, supraspinatus, subscapularis and combined infraspinatus/teres minor. For Sharkey et al. (1994) we calculated the percent increase in deltoid force when comparing the "deltoid, infraspinatus–teres minor, and subscapularis" condition to the "deltoid and entire rotator cuff" condition from Table 1 in that paper. For McMahon et al. (1995) we calculated the percent increase in deltoid force from the

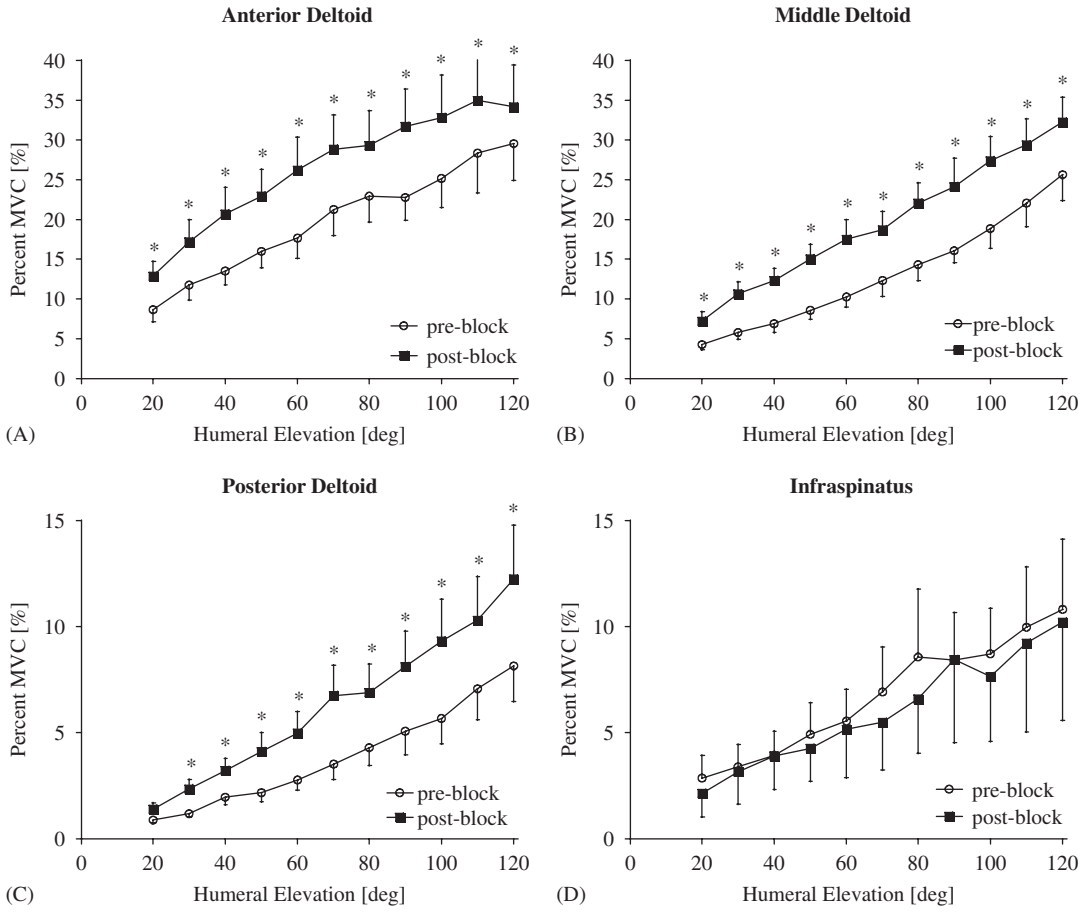

Fig. 3. Mean glenohumeral muscle EMG activity for all 10 subjects expressed as a percent of MVC activity as a function of humerothoracic elevation for the (A) anterior deltoid, (B) middle deltoid, (C) posterior deltoid and (D) infraspinatus. $*p<0.05$.

"equal force" condition to the "supra paralyzed" condition from Fig. 4 in that paper.

Additionally, the effects of supraspinatus paralysis were modeled using moment arm data reported by Liu et al. (1997) Moment arm data were taken from Fig. 4 in that paper (30° external rotation condition). Assuming static equilibrium, the following torque balance equation can be applied:

$$\sum T = F_{md}MA_{md} + F_{sup}MA_{sup} + F_{inf}MA_{inf} + F_{sub}MA_{sub} - F_{grav}MA_{grav} = 0,$$

where F is the force and MA the moment arm of gravity (grav), the middle deltoid (md), supraspinatus (sup), infraspinatus (inf) and subscapularis (sub). With the gravitational torque taken from Winter (2005) and the simplifying assumption that all muscle forces are equal, we calculated the percent increase in deltoid force when the supraspinatus was not part of the model compared to the deltoid force when the supraspinatus was included in the model. For the data in the present study, we calculated the percent increase in middle deltoid EMG in the post-block condition compared to the pre-block condition. Despite the disparity between the models (cadaver, computational and in vivo), they all show the same general trend of a large percent increase from baseline at low elevation angles that decreases as the arm is elevated (see Fig. 6). It should be noted that while Sharkey et al. (1994) included scapular motion in their model, McMahon et al. (1995) and Liu et al. (1997) did not. Consequently, for the latter two studies, we assumed a 2:1 ratio of glenohumeral to scapulothoracic motion to calculate the humerothoracic elevation numbers presented in Fig. 6.

A nerve block prevents the propagation of action potentials along an axon by interfering with sodium channel function. As the drug is removed from the area, either by diffusion or blood flow, more and more sodium channels become functional, resulting in a gradual increase in the number of function motor units, as well as a recovery of each axon's ability to propagate the signal, with an increased chance of signal passage as recovery progresses (Strichartz, 1998). In the present study, this recovery manifested in a gradual increase in force generating capacity in external rotation (Fig. 5A), presumably accomplished by an increase in the number of motor units involved and an increase in the firing rate of the motor units already recruited. For unweighted scapular plane elevation, the supraspinatus does not exceed 50% activation, but has the ability to increase its activation with an increase in external resistance (Alpert et al., 2000). Therefore, at least theoretically, when the nerve block recovery exceeded 50% of the lost strength, the

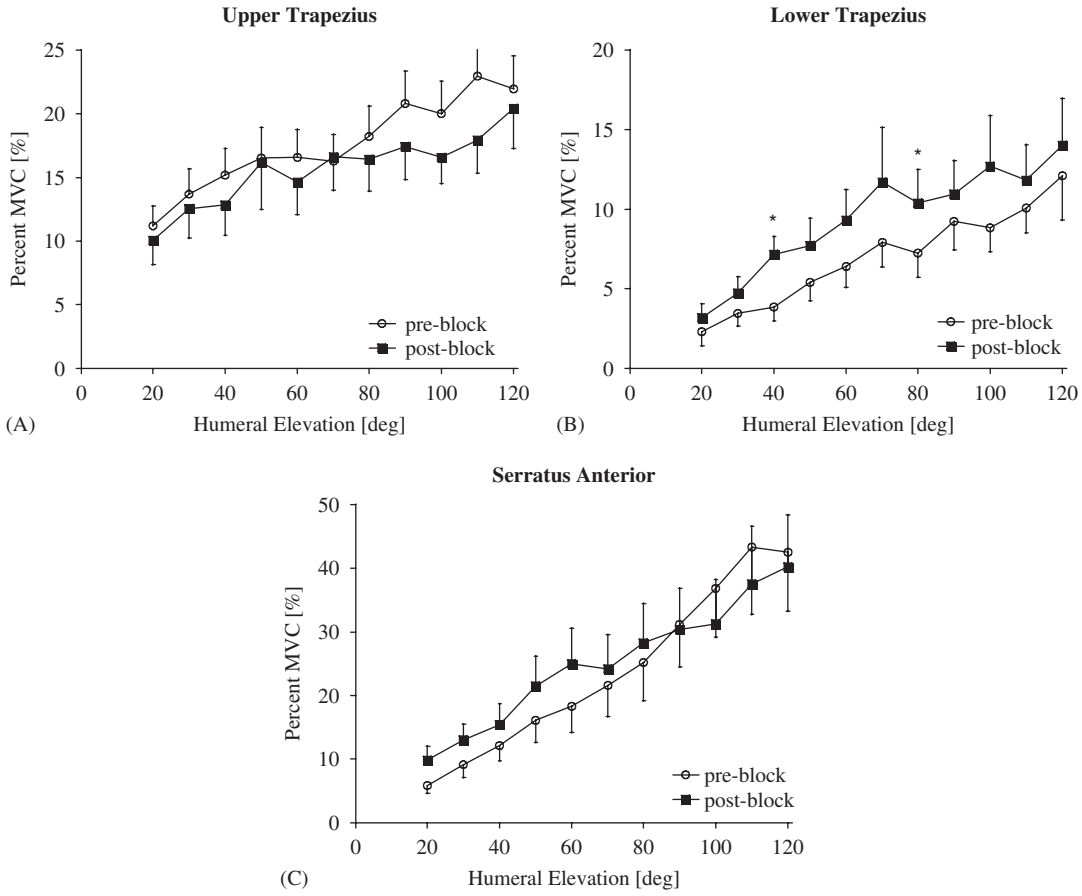

Fig. 4. Mean scapulothoracic muscle EMG activity for all 10 subject expressed as a percent of MVC activity as a function of humerothoracic elevation for the (A) upper trapezius, (B) lower trapezius and (C) serratus anterior. $*p<0.05$.

supraspinatus would have enough force generating capacity to perform its role without any increase from baseline for the deltoid. However, even at the last data collection point, when the external rotation force due to the block had recovered approximately 75%, there was still a compensatory increase in deltoid activity from baseline (Fig. 5B). In other words, even though the supraspinatus had the force generating capacity to meet its pre-block load, the central nervous system still recruited additional motor units from the deltoid. This evidence lends support to the concept that even mild rotator cuff impairment may result in a disturbance in the balance between the forces exerted by the deltoid and rotator cuff muscles, possibly leading to increased translations in situations such as impingement syndrome (Ludewig and Cook, 2002) and muscle fatigue (Chen et al., 1999; Royer et al., 2004). However, it should be noted that there could also have been compensatory changes in muscle activity in shoulder muscles for which we did not assess EMG. Interestingly, recent studies by Werner et al. demonstrated that a suprascapular nerve block does not result in either an increase in glenohumeral translation (2006b) or subacromial pressure (2006a).

Despite the fact that we have previously noted a compensatory increase in scapular upward rotation with a suprascapular nerve block (McCully et al., 2006), there were only mild changes in scapulothoracic muscle activity in the present study. This would appear to indicate that similar increases in scapular upward rotation noted in patients with cuff tears may not necessarily be due to deficits in scapulothoracic muscles (Paletta et al., 1997; Yamaguchi et al., 2000; Mell et al., 2005). The fact that we did not observe a decrease in infraspinatus activity was probably due to the difficulty in assessing EMG of this muscle with surface electrode. Under even ideal conditions, only a small portion of this muscle is accessible below the posterior deltoid. Due to motion of the scapula under the skin during shoulder elevation, there is a great deal of skin motion occurring at that region. This would have resulted in the electrodes sitting over motor units of other surrounding muscles that were not affected by the block. In the future, fine wire assessments of both the infraspinatus and supraspinatus would be more appropriate.

Another issue that needs to be addressed is fact that some subjects did not respond to the block. Incomplete blocks happen clinically in regional anesthesia. Even with nerve stimulation, it is possible to get a 'failed block'. It is possible that inadequate concentration of the drug was placed at the nerve or that the anatomy of the individual nerve was such that it had already bifurcated and only one segment was blocked. In addition, there is a variable

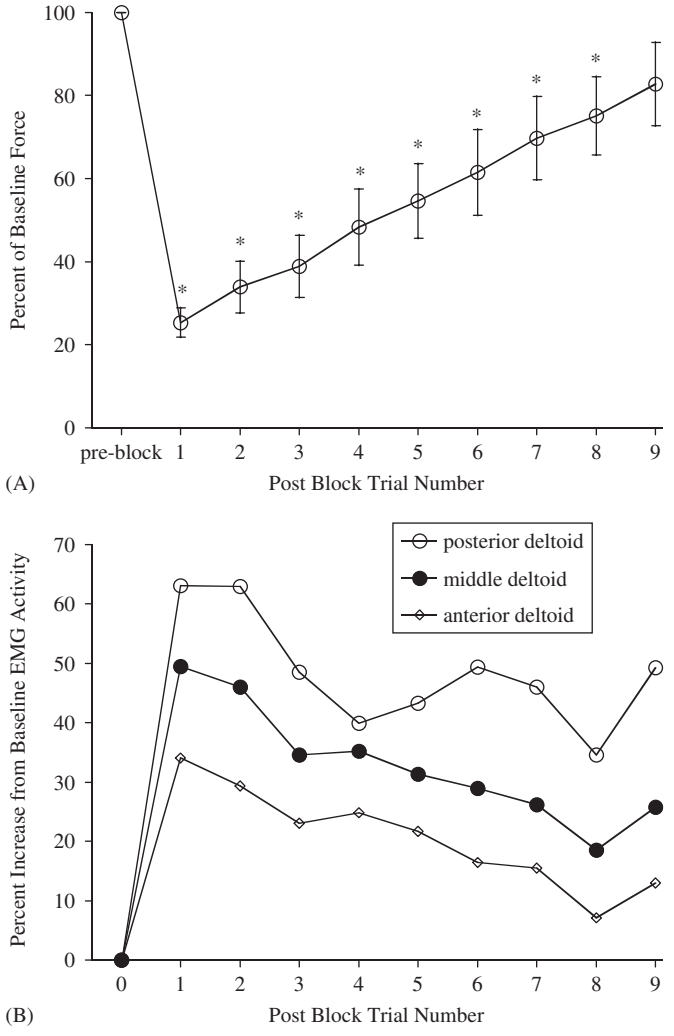

Fig. 5. Mean recovery for all 10 subjects of (A) external rotation strength and (B) deltoid EMG as a function of post block trial number. Strength data represent the percent of baseline (where 100% is the pre-block condition). EMG data represent the percent increase in activity (where 0% is the pre-block condition). A is reprinted from McCully et al. (2006) by permission of publisher.

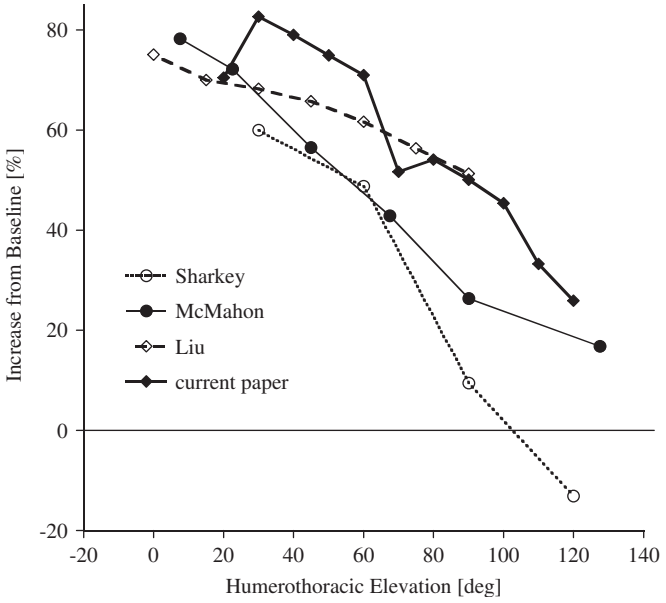

Fig. 6. Percent increase in middle deltoid activity as a function of humerothoracic elevation angle from four different data sets representing rotator cuff paralysis. Sharkey et al. (1994) and McMahon et al. (1995) are from active cadaver models, Liu et al. (1997) are from moment arm data and a simulation (see text for details) and current paper are from our middle deltoid EMG data for the first post-block trial.

response to local anesthetics and some subjects may not have had a full motor block due to this.

One of the fundamental motivations for the current study was a better understanding of the biomechanics of rotator cuff tears. However, there are many other symptoms of this pathology that were not reproduced in this study, such as pain, limited internal rotation range of motion and crepitus (Matsen et al., 2004). Additionally, both Thompson et al. (1996) and Hsu et al. (2000) have demonstrated that the biomechanical response to simulating muscle paralysis and tendon tears can be different. Future studies examining the EMG activity of the deltoid and rotator cuff muscles in patients with rotator cuff tears are warranted.

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