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Muscle fatigue does not lead to increased instability of upper extremity repetitive movements

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Abstract

Muscle fatigue alters neuromuscular responses. This may lead to increased sensitivity to perturbations and possibly to subsequent injury risk. We studied the effects of muscle fatigue on movement stability during a repetitive upper extremity task. Twenty healthy young subjects performed a repetitive work task, similar to sawing, synchronized with a metronome before and after performing each of two fatiguing tasks. The first fatigue task (“LIFT”) primarily fatigued the shoulder flexor muscles, while the second fatigue task (“SAW”) fatigued all of the muscles of the arm. Subjects performed each task in random order on two different days at least seven days apart. Instantaneous mean EMG frequencies (IMNF) decreased over both fatiguing tasks indicating that subjects did experience significant muscle fatigue. The slopes of the IMNF over time and the decreases in maximum force measurements demonstrated that the LIFT fatigue task successfully fatigued the shoulder flexors to a greater extent than any other muscle. On average, subjects exhibited more locally stable shoulder movements after the LIFT fatigue task ($p = 0.035$). They also exhibited more orbitally stable shoulder ($p = 0.021$) and elbow ($p = 0.013$) movements after the SAW fatigue task. Subjects also had decreased cocontraction at the wrist post-fatigue for both tasks ($p = 0.001$) and at the shoulder ($p < 0.001$) for the LIFT fatigue task. Therefore, increased dynamic stability of these repeated movements cannot be explained by subjects movements did not become more dynamically stable as a result of increased muscle cocontraction. Possible alternative mechanisms are discussed.

1. Introduction

Muscle fatigue is common in activities performed repeatedly over extended periods of time, such as in the workplace. Stability during these movements is crucial both to maintain performance and possibly to prevent injury. Here, we define stability as the ability to return to the same movement pattern after small perturbations. Stability is regulated during movement through feedback control (Reeves et al., 2007). Mechanisms include intrinsic properties of joints and muscles (i.e. stiffness and damping), and the central nervous system, which integrates information about joint position and movement to generate muscle (Bowman et al., 2006) and reflex responses. If any of these mechanisms are adversely affected, the resulting muscular responses may not adequately adjust for perturbations. Muscle fatigue can cause decreased proprioception (Myers et al., 1999), decreased kinesthesia (Pedersen et al., 1999), altered reflexes (Wojtys et al., 1996), increased muscle response time (Wilder et al., 1996; Wojtys et al., 1996), and increased central processing time (van Duinen et al., 2007). Therefore, muscle fatigue could decrease the ability to respond to perturbations.

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To compensate for decreased feedback and delayed responses, the body could increase muscle cocontraction to increase joint stiffness. For instance, if perturbations are introduced by imposing external forces, the body can selectively stiffen the muscles in that direction to maintain stability of reaching movements (Franklin et al., 2004). Individuals may (Stokes et al., 2000), or may not (Granata et al., 2001; Grondin and Potvin, 2009) increase cocontraction in response to expected perturbations or after muscle fatigue (Potvin and O'Brien, 1998). Increased stiffness does not always lead to more stable movements, however. Reeves et al. (2006) found that actively increasing trunk stiffness resulted in decreased postural control, possibly due to increased signal dependent noise from the increased trunk muscle recruitment. Likewise, using cocontraction to maintain stability during fatigue can initiate a vicious cycle as increased muscle activity is energetically costly and may further accelerate fatigue.

Nonlinear dynamics estimates of movement stability can quantify the control of dynamic movement tasks (Dingwell and Cusumano, 2000). Trunk flexion movements were more locally unstable after fatigue of the trunk extensors (Granata and Gottipati, 2008). Conversely, fatigue of the ankle muscles during prolonged walking caused subjects to slow down and trunk movements became more locally stable (Yoshino et al., 2004). These contrasting results may have occurred due to differences in how fatigue was induced. In the first study, fatigue was localized to a particular muscle group, the trunk extensors. In the second, fatigue was induced through prolonged walking, and was therefore not specific to a single muscle group. Specific fatigue of a single muscle group may cause greater changes in muscle activation patterns (Goerlick et al., 2003), or muscle imbalances between agonist and antagonist pairs of muscles (Alizadehkhayat et al., 2007). However, the way in which muscle fatigue and/or imbalances between opposing muscles affect the control of movement stability is not well understood.

In this study, we quantified dynamic stability for a continuous sawing task before and after subjects performed one of two fatiguing tasks. The first task was designed to primarily fatigue the shoulder flexors. The second task caused general fatigue of the arm. We tested four hypotheses. First, subjects will exhibit greater dynamic instability following both fatiguing tasks. Second, these increases in dynamic instability will be larger following targeted shoulder flexor fatigue. Third, imbalances between opposing muscles will be greater after targeted shoulder flexor fatigue than after general arm fatigue. Finally, subjects will exhibit increased cocontraction post-fatigue to help maintain stability.

2. Methods

Twenty healthy right-handed adults (9 female, 11 male; 25 ± 2.2 years, 71.2 ± 14.9 kg, and 1.71 ± 0.10 m tall) participated. All participants signed institutionally approved consent forms and were screened to ensure they had no history of medications, surgeries, injuries, or illnesses that might have affected their upper extremity movements.

Subjects completed two experiments at least one week apart. Each session followed the same general protocol (Fig. 1A). All testing was performed with the right arm only. First, each subject was seated and belted in a chair (Fig. 1B). A handle attached to a Baseline® dynamometer was fixed in the mid-position of the track so that the subject's elbow flexion angle was approximately 90° . Subjects alternately pushed and then pulled with maximal effort three times for 5 seconds each, with 60 seconds rest. The average of these six peak forces (3 push, 3 pull) defined that subject's maximum pushing/pulling strength.

To quantify imbalances between opposing muscles, maximum strength (MVC) was measured for shoulder (flexion, extension, internal rotation and external rotation) and elbow (flexion, extension) at four time points (Fig. 1A). Subjects sat, strapped into the device, with their right arms in specific positions (Table 1). They pushed against a hand-held load cell (Lafayette

Instruments, Lafayette, IN) with maximal effort two times for 5 seconds each, with no more than 30 seconds rest. The greater of the two peak forces applied defined each subject's MVC. To compare across subjects, each subject's MVCs were normalized to the maximum that they achieved over all the MVC measurements. Muscle balance ratios were computed as the ratio of strength in opposing directions (i.e. ABD/ADD, FLEX/EXT, INT/EXT). Only shoulder flexion and extension strength were measured immediately following the fatigue task to reduce muscle recovery.

Ratings of perceived exertion (RPE) were also recorded periodically (Fig. 1A) using the modified Borg scale (Borg, 1982). Participants subjectively rated their level of fatigue on a scale from 0 ("none at all") to 10 ("maximal exertion").

Subjects sat in a device that simulated a repetitive sawing-like task (Fig. 1B). The device was adjusted specifically to fit each subject (Fig 1; Gates and Dingwell, 2008). Subjects made bi-directional anterior-posterior horizontal movements with their right arm while holding a sliding handle mounted on a low friction track. Inertial resistance was supplied by weights attached to the handle, adjusted to 10% of each subject's maximum pushing/pulling strength. Subjects performed this task continuously for 5 minutes before and after each fatigue protocol (Fig. 1A).

Movement timing was imposed by a metronome. For each subject, the frequency was set to twice the average of the predicted resonant frequencies of the upper arm and forearm (approx. 1 Hz) (Gates and Dingwell, 2008). Subjects performed a 30 second warm-up (~30 cycles) to eliminate any potential learning effects. Subjects were able to learn this simple task (i.e., their mean timing errors approached zero) within just a few (< 10) movements (Gates and Dingwell, 2008).

Nineteen reflective markers defined the movements of four body segments. Trunk markers were placed at the right and left acromion processes, sternal notch, and seventh cervical vertebra. Clusters of four markers each defined the upper and lower arm segments. Markers placed at the radial and ulnar epicondyles, and third and fifth metacarpal-phalangeal joints defined the hand. Additional markers were placed on the medial and lateral humeral epicondyles for static calibration. A final marker was placed on the handle to define the beginning and end of each cycle. Marker movements were recorded continuously during all sawing trials at 120 Hz using an 8-camera Vicon-612 motion analysis system (Oxford Metrics, Oxford, UK).

Nine pairs of Delsys (Delsys Inc., Boston, MA) EMG surface electrodes were attached to the pectoralis major, upper trapezius, deltoid (anterior, middle and posterior), biceps, triceps, flexor carpi radialis, and extensor carpi radialis longus muscles. Electrodes were positioned over each muscle according to (Konrad, 2005). EMG activity was recorded continuously during all sawing trials at 1080 Hz.

Each subject completed two fatigue tasks. The first task, SAW, involved general fatigue of all arm muscles. Subjects performed the same sawing task described above, except now against ~25% of their maximum pushing/pulling strength. Subjects sawed 4 minutes, or until they felt they could no longer continue. Two subjects fatigued in less than 4 minutes. Six subjects who were not fatigued ($RPE < 6$) after 4 minutes continued for an additional 4 minutes, or until they reached $RPE \geq 8$. The remaining 12 subjects were fatigued ($RPE \geq 8$) after 4 minutes.

The second fatigue task, LIFT, was designed to primarily fatigue the shoulder flexors. Subjects lifted a weight (~10% of their maximum isometric shoulder flexion strength) in the sagittal plane with elbows extended. They performed this task at half the frequency of the sawing task (approximately 0.5 Hz). They did this for 3 minutes or until they felt they could no longer

continue. Eight subjects were unable to complete the 3 minutes. All other subjects were fatigued ($RPE \geq 8$) after 3 minutes.

Marker data were filtered at 15 Hz using a zero-lag 5th order Butterworth low-pass filter. Segment coordinate systems were defined using a least-squares algorithm (Veldpaus, 1988). Joint centers were determined from static trial marker positions (Schmidt et al., 1999). Local coordinate systems were defined using International Society of Biomechanics' (ISB) recommendations for the shoulder and elbow (Wu et al., 2005) and modified coordinate systems for the trunk (Hingtgen et al., 2006) and wrist (Rao et al., 1996). Joint angular movements were determined using Euler angles following ISB recommendations (Wu et al., 2005). Angular velocities were calculated using a 3-point difference formula.

These kinematic variables were used to define multi-dimensional state spaces for each joint (Gates and Dingwell, 2009).

$$\mathbf{S}(t) = [\theta_1(t), \theta_2(t), \theta_3(t), \dot{\theta}_1(t), \dot{\theta}_2(t), \dot{\theta}_3(t)] \in \mathbb{R}^6 \quad (1)$$

Shoulder and wrist state spaces consisted of all three rotational angles and angular velocities. Because elbow carrying angles are highly mechanically constrained, this angle and angular velocity were excluded from the elbow state spaces (i.e., $\mathbf{S} \in \mathbb{R}^4$)

Local stability of state space trajectories was quantified from maximum local divergence exponents (λ^*) (Rosenstein et al., 1993; Dingwell and Cusumano, 2000). Positive exponents indicate local instability. Larger exponents indicate greater instability. Slopes of mean log divergence curves were calculated over 0 to 1 cycles (λ_s^*) and over 4 to 10 cycles (λ_L^*) (Dingwell and Marin, 2006). These calculations are sensitive to sample size (Granata and England, 2006) and number of cycles (Bruijn et al., 2009). Each Pre/Post trial was re-sampled to 36,000 total data points, resulting in <1% change in the length of each time series. Task frequency was governed by the metronome, so the number of cycles was approximately the same (± 2 cycles) for each trial of each subject.

Orbital stability was quantified from maximum Floquet Multipliers (FM) (Kang and Dingwell, 2008). State space data for each cycle were time-normalized to 101 samples (0% to 100%), defining 101 Poincaré sections, one for each percent of the movement cycle. We assumed the average trajectory across all cycles within a trial represented the 'limit cycle', creating a single fixed point in each Poincaré section. Floquet multipliers quantify how quickly small perturbations away from these fixed points grow or decay. FM with magnitude < 1 indicate that perturbations shrink, on average, by the next cycle, reflecting a stable system. We calculated the magnitude of the maximum FM across the movement cycle (i.e., across all Poincaré sections) for each sawing pre- and post-test.

Instantaneous mean power frequencies (*IMPF*) of each EMG signal were calculated using wavelet techniques (Hostens, 2004). *IMNFs* were averaged over each cycle to give a single value per cycle. Slopes of *IMNF* vs. cycle curves were used to quantify how local fatigue states changed across consecutive cycles during each trial. Localized muscle fatigue would cause EMG frequencies to decrease (DeLuca, 1997).

Separately, raw EMG data were full wave rectified and low-pass filtered at 6 Hz with a zero-lag 2nd order Butterworth filter to obtain linear envelopes (Missenard et al., 2008b). EMG linear envelopes were normalized to the average peak muscle activation across the pre-fatigue

sawing trial. Muscle cocontraction indices (CCI) were estimated using (Missenard et al., 2008b; Fig. 2):

$$CCI = \frac{\int_0^{100} EMG_{min} dp}{\int_0^{100} (EMG_{ago} + EMG_{ant}) dp} \times 100 \quad (2)$$

where p was percent movement cycle, EMG_{min} was taken from the muscle with the lower normalized activity at each sampling point. EMG_{ago} and EMG_{ant} represented the agonist and antagonist muscles, respectively. CCIs were calculated for three muscle pairs: anterior and posterior deltoid (shoulder), biceps and triceps (elbow), and extensor and flexor carpi radialis (wrist). CCIs were calculated for each movement cycle and averaged across cycles to obtain a single value for each subject pre- and post-fatigue for each condition.

IMNF slopes during fatigue trials for each condition were compared using a single factor ANOVA to test for differences between muscles, followed by Tukey's honestly significant difference tests. MVCs were compared using 2-factor (Time point (1–4) $\times$ Saw/Lift) repeated measures ANOVAs. Cocontraction indices (CCI) and stability measures (λ_s^* , λ_L^* , and peak MaxFM) were compared using 2-factor (Pre/Post $\times$ Saw/Lift) repeated measures ANOVAs. We explored differences between Pre/Post trials for the different conditions using a least significant difference test. Significance level was set at $p < 0.05$ for all comparisons.

3. Results

All subjects exhibited significant localized muscle fatigue, as measured by decreased IMNF, during both fatigue tasks (Fig. 3). The amount of fatigue was significantly greater in the anterior deltoid than all other muscles ($p < 0.019$), except the posterior ($p = 0.101$) and lateral deltoid ($p = 0.803$) for the LIFT fatigue task (Fig. 3). Conversely, the SAW task fatigued all muscles fairly equally. The posterior deltoid was significantly less fatigued than the anterior deltoid ($p = 0.006$) and wrist extensor ($p = 0.024$). Therefore, the imbalance in fatigue rates of the anterior and posterior deltoid was more pronounced in the non-specific SAW task.

MVCs decreased post-fatigue for all strength measures ($p < 0.038$; Fig 4). Shoulder flexion MVCs were significantly different between conditions ($p = 0.035$). Subjects exhibited greater decreases in shoulder flexion strength and increased imbalances of flexion/extension and internal/external rotation strength after the LIFT task (Fig. 4). Thus, the LIFT task was successful at primarily fatiguing the shoulder flexors.

λ_s^* decreased post-fatigue at the shoulder ($p = 0.029$; Fig. 5). Post-hoc analysis showed that the pre/post difference was significant for lifting ($p = 0.035$) but not sawing ($p = 0.241$). For elbow and wrist movements, λ_s^* did not change significantly after fatigue and were not significantly different for the two fatigue protocols. No differences in λ_L^* were found for any comparison.

Orbital stability of the elbow decreased significantly post-fatigue ($p = 0.016$), but did not change for the shoulder ($p = 0.152$) or wrist ($p = 0.060$). There were no significant differences between fatigue protocols. Protocol $\times$ fatigue interactions did not reach significance (elbow: $p = 0.054$, wrist: $p = 0.086$). Least significant difference tests revealed significant decreases in MaxFM for the shoulder and elbow post-fatigue for the SAW task ($p = 0.021$ and 0.013 respectively; Fig 6). There were no changes in orbital stability after the LIFT task.

Cocontraction decreased significantly post-fatigue at the shoulder for the SAW task ($p < 0.001$; Fig. 7) and at the wrist for both fatigue tasks ($p = 0.001$). There were no significant differences in cocontraction either at the elbow or between fatigue tasks ($p > 0.05$).

4. Discussion

This study quantified how muscle fatigue affected movement stability during a repetitive sawing task. We hypothesized that muscle strength imbalances involving targeted shoulder flexor fatigue would result in movement instability. Although moderate muscle imbalances were seen post-fatigue, these did not lead to instability. Instead, subjects became slightly more locally and orbitally stable post-fatigue. This was not accomplished through increased muscle cocontraction.

Findings that muscles respond slower to perturbations post-fatigue (Wilder et al., 1996) suggest that this could lead to unstable movements. However, many muscles cross each joint and may not fatigue at the same rate (Kumar and Narayan, 1998). The LIFT task primarily fatigued the anterior deltoid muscle (Fig. 3). Possibly, smaller less fatigued accessory muscles could compensate for decreased response time of fatigued muscles. Although we attempted to fatigue only the shoulder flexors, the LIFT task also fatigued other muscles. Subjects had to grip the weight in their hands, which may have fatigued the wrist flexors and extensors. While this task did not exclusively fatigue the shoulder flexors, it did fatigue the anterior deltoid more than any other muscle (Fig. 3).

Subjects could also have cocontracted to stiffen their muscles. This would allow intrinsic tissue properties to contribute more in responding to perturbations than muscle and reflex responses (Reeves et al., 2007). Some studies have shown such increased cocontraction in response to fatigue (Granata et al., 2001; Grondin and Potvin, 2009). Muscle fatigue also leads to increased force variability, which can impair movement accuracy. Cocontraction might also help people maintain movement accuracy (Gribble et al., 2003; van Dieën et al., 2003; Missenard et al., 2008a; Reeves et al., 2008). A few studies have looked at the role of cocontraction in improving accuracy after fatigue. Huysmans et al. (2008) found that subjects exhibited decreased accuracy in the minute following a fatigue task, but that accuracy was unchanged in the following minute. Their subjects did not exhibit increased muscle activity. Subjects in Missenard et al. (2008b)'s study did not cocontract and exhibited decreased movement accuracy and precision post-fatigue (Missenard et al., 2008b). Both of these studies were positional tasks where cocontraction is likely necessary to maintain end-point precision. In the present study, subjects presumably had to maintain movement stability while also accurately matching the metronome. Our subjects maintained constant accuracy post-fatigue (no changes in movement time or distance) and qualitatively similar movement patterns (Fig. 8) without increasing cocontraction. They also did not exhibit impaired stability. This might indicate that subjects used a feedback correction strategy to compensate for fatigue effects, as this is less energetically costly than cocontraction (Selen, 2006).

Like increased accuracy, it has often been suggested that cocontraction leads to increased stability. However, cocontracting too much could also make one more "rigid" and thus less able to respond to specific perturbations. While reaching in novel force field environments, people do not cocontract, but instead learn to specifically counteract the particular force field they anticipate (Shadmehr and Mussa-Ivaldi, 1994). Even when those forces vary unpredictably, people do not cocontract, but instead use learned information about perturbations and movement errors from immediately previous trials to adjust motor commands on subsequent trials (Scheidt et al. 2001).

The cocontraction index used here is not unique. Many other indices have been proposed (Falconer and Winter, 1985; Frost et al., 1997; Rudolph et al., 2001) and numerical values differ for different indices (Kellis et al., 2003). The CCI used here was applied only to the prime movers of the task. Thus, smaller accessory muscles might have either increased or decreased their activity, which could change the overall agonist to antagonist ratio. However, we expect that by quantifying cocontraction of the largest muscles, we captured the bulk of the activity. These indices may also be affected by noise levels in the EMG signal. While there is no way of directly quantifying the true quality of the EMG signal, care was taken to prepare the skin by cleansing with alcohol, shaving excess hair, placing the electrode over the muscle belly and holding it in place with tape and wrap.

Despite our attempt to make the tasks as similar as possible for the different subjects, significant differences in their responses remained, particularly for the MVC measures. The between-subject variability observed here was similar to many previous studies of multijoint fatigue (Nussbaum, 2001; von Tscharner, 2002; Madigan and Pidcoe, 2003). As this task was inherently redundant, subjects could compensate for fatigue by using different muscles or strategies that might allow them to maintain their stability. Different subjects fatigued to different degrees. Subjects could stop the fatigue task as soon as they felt they could no longer continue (RPE = 10). This “threshold” could be different for different subjects, depending on motivation level and other factors. Those with prior exercise experience may also have recovered quicker from the fatigue task. Some subjects showed rapid recovery from the fatigue task and their MVC values were completely restored after the ‘Post’ trial while others showed continued decreases.

In summary, subjects performed consistently accurate movements before and after fatigue. They did this in spite of significant muscle fatigue, imbalances between opposing muscles, and decreased cocontraction. Subjects’ shoulder movements became slightly more locally stable after targeted shoulder flexor fatigue. Shoulder and elbow movements became slightly more orbitally stable after general fatigue of the arm. Thus, when performing multi-joint redundant tasks, humans can compensate for muscle fatigue in ways that maintain task precision while increasing movement stability.

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Figure 1.

A) General protocol for the experiment. On both visits, all 20 subjects completed all activities. Only the fatigue task differed. **B)** During the sawing task subjects were seated in a high-back chair and restrained by a five-point harness (Corbeau, Sandy, UT) across their waist and shoulders. A handle with an adjustable weight stack was able to slide with low friction across a horizontal track. The device was adjusted so subjects sat with a knee angle of 90°. The height of the metal track was adjusted so the midpoint between the third and fourth finger was at the level of the xiphoid process. The front/back position of the chair was adjusted to be comfortable for the subject and allow for a full range of motion. This was defined as being at a maximum point when almost to full extension (no hyperextension) and at a minimum point at the level of the sternum. **C)** In the sawing (Saw) fatigue task subjects pushed 25% of their pushing/pulling MVC for four minutes. In the lifting (Lift) fatigue task, subjects lifted 10% of their shoulder flexion MVC from at their side to approximately 90 degrees in the sagittal plane for three minutes. In both tasks, subjects could stop at any point they felt they could no longer continue.

Figure 2.

EMG linear envelopes for one trial of a representative subject are shown. Here EMG_{ago} was the anterior deltoid, EMG_{ant} was the posterior deltoid and EMG_{min} was whichever had the lower value at that percent of the movement cycle. Subjects pushed the weight forward during the first 50 % of the movement cycle and then brought it back toward them during the last 50 %.

Figure 3.

The slope of the IMNF vs. cycle curves are shown for each condition. Errorbars represent 95% confidence intervals about the mean of the 20 subjects. Subjects showed significant fatigue in all muscles as a result of both fatigue protocols (95% confidence intervals do not include 0).

Figure 4.

MVC measures were taken at four time points in the experiment (Fig. 1A). **A)** MVCs are shown as a percent of maximum for Top: shoulder flexion and extension, Middle: shoulder internal and external rotation, Bottom: elbow flexion and extension. **B)** Balance ratios of shoulder flexion to extension and shoulder internal to external rotation MVCs are shown for each time point. Significant differences from MVC1 are represented as '*' for both fatigue protocols, '†' for the lifting protocol only, and '§' for the sawing protocol only. Significant differences between conditions at that time point are represented by 'c'.

Figure 5.

Mean values of λ_s^* are shown for the shoulder, elbow and wrist. Data are shown prior to fatigue 'o' and after 'x' either the sawing or lifting fatigue protocol. Errorbars are the 95% confidence intervals across subjects about the mean. "*" represent significant pre/post effects. $n = 20$.

Figure 6.

A) The MaxFM across the movement cycle is shown after the LIFT and SAW tasks. 0% was at the start of the pushing phase. At 50%, the subject began to pull the weight back toward them. B) The peak value of the MaxFM across the movement cycle is shown before and after each fatigue condition. 'o' represents the pre-fatigue sawing trials while 'x' denotes the post-fatigue sawing trials. Significant differences from pre to post-fatigue for that condition are denoted with '*'. n = 20.

Figure 7.

Cocontraction indices (CCI) are shown for the shoulder, elbow and wrist pre and post fatigue for each fatigue condition. 'o' represents the pre-fatigue sawing trials while 'x' denotes the post-fatigue sawing trials. Significant differences from pre to post-fatigue for that condition are denoted with '*'.

Figure 8.

The average movement pattern for the shoulder is shown pre and post fatigue for each condition. From right to left, these angles are shoulder elevation, humeral plane angle, and humeral rotation angle. Dashed lines represent the standard deviation across subjects for that condition.

Table 1**Positions of the arm during strength testing**

In each test, the subject was strapped into the seat used for the sawing task (Fig. 1B). The bar was raised to just below shoulder height so that the subject could rest their upper arm on the bar.

Shoulder Flexion	The arm was lifted to 90° in sagittal plane and the elbow was fully extended. The load cell was placed over the center of the anterior deltoid muscle.
Shoulder Extension	The arm was at the side of the body with the elbow fully extended. The load cell was placed approximately over the triceps muscle.
Shoulder Internal/External Rotation	The upper arm rested on the bar so that the shoulder was flexed 90 degrees. The elbow was flexed 90 degrees and in a neutral position. The load cell was placed just below the wrist on either the palmar (internal rotation) or dorsal (external rotation) surface.
Elbow Flexion/Extension	The upper arm rested on the bar so that the shoulder was flexed 90 degrees. The elbow was flexed 90 degrees and in a neutral position. The load cell was placed half way down the forearm either toward (flexion) or away (extension) from the body.