Muscle Fatigability and Control of Force in Men with Posttraumatic Stress Disorder

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ABSTRACT

KELLER-ROSS, M. L., B. SCHLINDER-DELAP, R. DOYEL, G. LARSON, and S. K. HUNTER. Muscle Fatigability and Control of Force in Men with Posttraumatic Stress Disorder. *Med. Sci. Sports Exerc.*, Vol. 46, No. 7, pp. 1302–1313, 2014. **Introduction**: Acute stress can increase fatigability and decrease steadiness of sustained low-force contractions that are required for functional tasks in upper limb muscles. Whether motor performance is more impaired in people with a chronic stress disorder is not known. **Purpose**: This study compared the fatigability and steadiness (force fluctuations) of handgrip muscles in veterans with posttraumatic stress disorder (PTSD) and civilian controls in the presence and absence of varying levels of cognitive demand. **Methods**: Eighteen veterans with PTSD and 21 healthy controls (33 ± 9 yr) attended three randomized experimental sessions to perform an isometric fatiguing contraction (20% of maximal strength) with the handgrip muscles. Two sessions involved performing a cognitive task during the fatiguing contraction: 1) difficult mental math task (stressor) and 2) a simple mental math task (mental attentiveness). A third session involved a fatiguing contraction with no mental task (control). **Results**: Stress elevated heart rate, blood pressure, and levels of anxiety in veterans with PTSD ($P < 0.05$) but blunted cortisol levels ($P < 0.05$). Time to failure was briefer (7.2 ± 2.5 vs 9.3 ± 5.2 min, $P = 0.03$), and force fluctuations increased at a greater rate for veterans with PTSD than for controls ($P < 0.05$). Cognitive stress did not influence time to failure or force fluctuations for either group ($P > 0.05$). **Conclusions**: Veterans with PTSD demonstrated greater fatigability and loss of steadiness (greater force fluctuations) of the handgrip muscles compared with healthy controls. **Significance**: Male veterans with PTSD demonstrated altered neuromuscular function of arm muscles that potentially affects functional tasks during daily, ergonomic, and military activities. **Key Words:** MUSCLE FATIGUE, STRESS, PTSD, HANDGRIP, CORTISOL, AROUSAL, MEN

In healthy young adults, an acute stressor can decrease steadiness (increase force fluctuations) and reduce time to task failure for low-intensity isometric contractions (7,27,40). Low-intensity contractions are foundational for activities of daily living, and the influence of stress on these types of tasks has important implications for musculoskeletal disorders. For example, exposure to an acute stressor will usually increase sympathetic outflow, which can result in excessive and inappropriate actions on motor control. Sympathetic activation exerts a number of actions at the periphery, including modulation of skeletal muscle contractility (4), reduction of blood perfusion to skeletal muscles (38), and modulation of the discharge of numerous receptors (i.e., muscle spindles that carry afferent feedback to the muscle for adequate motor control) (15).

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A clinical population with increased activation of the sympathetic nervous system such as people with posttraumatic stress disorder (PTSD) may therefore demonstrate greater muscle fatigability and alterations in motor control because of the actions of the sympathetic activity on skeletal muscle. Motor control and fatigability of people with PTSD is completely unexplored. PTSD can be caused by the threat of death or serious injury that leads to a reaction of intense fear, helplessness, or horror and causes a dysregulation in the stress systems (hypothalamic–pituitary–adrenal axis and sympathetic nervous system) (9). Symptoms that develop in those with PTSD include reexperiencing the traumatic event, avoidance of stimuli, and emotional numbing and hyperarousal (1). People with PTSD demonstrate elevated levels of sympathetic activation (increased plasma epinephrine, norepinephrine, and serotonin) (36), resulting in higher resting levels of heart rate (HR) and mean arterial pressure (MAP) (30). There is also evidence that those with PTSD have lower basal levels of cortisol that is, in part, due to an enhanced negative feedback system and reduced adrenal output (9). Such physiological adaptations that occur from an acute traumatic stressor can have detrimental and long-lasting psychological effects (24,31), but whether these adaptations result in alterations in motor behavior and fatigue of people with PTSD is not known.

Low-intensity contractions sustained for a long duration in the upper limb can result in substantial fatigability and

loss of steadiness (18,22,40) and are the foundation of stabilizing tasks performed in vocational and military settings. Hence, we chose to specifically study the handgrip muscles of war veterans with PTSD. Veterans of war often have a high prevalence of combat- or military-related PTSD, with a lifetime prevalence of PTSD of 15% in Iraq and Afghanistan war veterans (35). Given the rise in PTSD in veterans from recent combat experiences who are now attempting to assimilate into society and the workforce, there is significant clinical relevance for understanding motor behavior and potential impairments in people with PTSD. An aim of this study therefore was to compare time to task failure and steadiness (force fluctuations) of veterans with PTSD and healthy controls for a low-intensity contraction performed with the handgrip muscles. Because people with PTSD have elevated basal levels of sympathetic activation (measured by plasma catecholamines) (36), which may influence motor performance, we *hypothesized* that veterans with PTSD would fatigue more rapidly and be less steady than healthy control subjects.

Furthermore, many vocational and military tasks are executed while performing a cognitive task or under stressful conditions. Individuals with PTSD have altered physiological responses to acute stressors than healthy adults, including elevated HR and blood pressure and increased skin conductance (measures of the sympathetic nervous system activity) when exposed to a stressor (31). It is unknown whether the greater stress response demonstrated in individuals with PTSD would change muscle fatigability or the ability to maintain a steady contraction as occurs in elbow flexor muscles of healthy young adults (40). Therefore, a second aim of this study was to determine whether exposure to an acute cognitive stressor increases muscle fatigability and reduces steadiness of a low-intensity contraction with the handgrip muscles in veterans with PTSD. We *hypothesized* that fatigability would be greater and that steadiness was reduced more for veterans with PTSD when exposed to the acute stressor than healthy controls. We assessed potential neural and hormonal (HR, MAP, EMG, and cortisol) mechanisms that may have contributed to impaired motor performance with low-intensity contractions. These questions are equally important to men and women, and as both were actively recruited, only one woman qualified for and participated in the study. Because of the sex differences in response to stress (40), we removed the data of the woman subject from the sample until we are able to obtain enough women for a separate group.

METHODS

Study Overview

A total of 39 subjects participated in this study to investigate two aims. To address the first aim, 18 male veterans with PTSD (36 ± 9 yr) and 21 male control subjects who did not have PTSD (two veterans) (28 ± 9 yr) participated in a session to perform a fatiguing contraction (20% of the maximal voluntary contraction [MVC]) with handgrip muscles. To address the second aim, 18 male veterans with PTSD and 12 male control subjects attended an additional session to perform a difficult mental math task simultaneously during the fatiguing contraction. We have previously demonstrated that, in young healthy adults, a mental attentiveness task (a simple mental task for the purposes of distraction) while performing a fatiguing contraction does not result in greater fatigue or changes in steadiness (40). To determine whether mental attentiveness changes fatigue and steadiness in veterans with PTSD, 18 male veterans with PTSD and 7 control subjects attended a third session where they performed a simple mental math task during the fatiguing contraction (mental attentiveness session). Female veterans were actively recruited, and as a small number of women demonstrated interest, only one fit the criteria for the study and therefore her data were not included in the analysis.

All testing occurred in Milwaukee, WI, at the Veteran Affairs Medical Center for the veterans and at Marquette University for the nonveteran control subjects. The equipment setup was identical at each site, and the same investigators conducted the experiments at each site. The protocol was approved by the institutional review board at the Veteran Affairs Medical Center and Marquette University. Before participation in the study, each subject provided informed consent. At the initial familiarization session, all subjects completed health questionnaires, were familiarized to the equipment, and practiced experimental procedures.

Subjects answered questionnaires regarding their general anxiety levels (State Trait Anxiety Inventory [STAI-Trait]) (37), symptoms of PTSD using the PTSD Checklist – Civilian (PCL-C) (39), and symptoms of depression with the Beck Depression Inventory (BDI; n = 28, 10 control subjects completed the PCL-C and BDI) (2). The PCL-C and BDI were administered and evaluated by qualified psychologists, under the supervision of an M.D. at the VA Medical Center. Body mass index (BMI) was calculated from height and weight measurements, and physical activity levels for each subject were assessed with a questionnaire that estimated metabolic equivalent ($MET \cdot h \cdot wk^{-1}$; Table 1) (23). Hand dominance was estimated by the Edinburgh Handedness Inventory (28) (0.35 ± 0.5 vs 0.60 ± 0.4, P = 0.20) for PTSD and controls, respectively, with a ratio of 1 indicating complete right-handedness. Participants practiced MVCs and brief submaximal target matching contractions at 20% of MVC force with the left hand. The right hand of one veteran was tested because he had previously broken the left wrist. Subjects were instructed to abstain from caffeine, exercise, and smoking (seven veterans [one in the control group] smoked cigarettes) on the days of testing and alcohol 24 h before testing. All subjects were without known neurological or cardiovascular diseases and were naive to the protocol.

Veterans with PTSD were diagnosed by a physician using the *Diagnostic and Statistical Manual, Fourth Edition* (*DSM-IV*). The participating veterans served Operation Enduring Freedom or Operation Iraq Freedom theater era. The

TABLE 1. Subject characteristics for veterans with PTSD and healthy control subjects and types of medications and behavioral traits of veterans with PTSD.

Variable	PTSD	Healthy Controls	*P* (Group Effect)
Aim 1: no acute stress (n = 21 CTL)			
Age (yr)	36 ± 10	29 ± 10	0.03
Body mass index (kg·m^{-2})	29.7 ± 4.3	24.7 ± 4.0	0.003
PA (MET·h·wk^{-1})	38.5 ± 42.8	54.6 ± 55.0	0.40
Trait anxiety (STAI)	58.4 ± 11.4	32.3 ± 7.2	<0.001
PCL-C	63.1 ± 13.3	25.6 ± 12.9	<0.001
BDI	32.7 ± 13.9	6.4 ± 8.8	<0.001
Aim 2: mental stress (n = 12 CTL)			
Age (yr)		31 ± 11	0.18
Body mass index (kg·m^{-2})		25.8 ± 5.6	0.16
PA (MET·h·wk^{-1})		45.3 ± 32.7	0.67
Trait anxiety (STAI)		34.0 ± 8.2	<0.001
PCL-C		25.6 ± 12.9	<0.001
BDI		6.4 ± 8.8	<0.001
Mental attentiveness (n = 7 CTL)			
Age (yr)		28 ± 10	0.09
Body mass index (kg·m^{-2})		25.8 ± 5.6	0.16
PA (MET·h·wk^{-1})		38.6 ± 33.4	0.99
Trait anxiety (STAI)		33.3 ± 5.0	<0.001
PCL-C		26.2 ± 12.1	<0.001
BDI		6.2 ± 6.3	<0.001
	***n* (%)**		
Type of medication			
Antidepressant	12 (67)		
Anxiolytic	3 (17)		
Antipsychotic	4 (22)		
Insomnia Rx	10 (56)		
Analgesic	11 (61)		
Hypertensive Rx	4 (22)		
Smoking	7 (39)		
Alcohol use			
Excessive (treated while in study)	2 (11)		
Moderate	5 (28)		
Rarely	5 (28)		
None	6 (33)		

The *P* value for each variable is indicated in the last column of the table.
BDI, Beck Depression Inventory (<14 = mild, 15–30 = moderate, >30 = severe); CTL, control group; PA, physical activity questionnaire (metabolic equivalents [MET·h·wk^{-1}]); PCL-C, Posttraumatic Stress Disorder Checklist – Civilian (>44 indicates symptoms qualify for diagnosis of PTSD); STAI, State Trait Anxiety Inventory.

medications for the subjects in the PTSD group were not controlled but were documented at their initial session. Twenty-two percent of the veterans were medication-free, whereas the remaining veterans took prescribed medications (Table 1). Ten veterans were taking selective serotonin reuptake inhibitors (SSRIs) at the time of the study; one veteran was taking a selective norepinephrine reuptake inhibitor and one subject was taking a serotonin and norepinephrine reuptake inhibitor. The other most commonly prescribed medication was for pain relief, with 55% (11) of veterans taking a prescribed pain medication. The common location for pain complaint was chronic low back pain (55%), with 11% having combined knee or ankle pain. No subject reported pain associated with the exercising arm/hand.

Experimental Protocol for Control, Stressor, and Mental Attentiveness Session

Each experimental session began with assessments of baseline levels of anxiety using the visual analog scale (VAS) (19) and STAI (state). All procedures were performed in the following order for each experimental session (Fig. 1): 1) MVCs of the handgrip muscles, 2) assessments of cognitive and physiological arousal before and after either quiet sitting (control session) or 4 min (2 × 2-min bouts) of mental tasks (stressor and mental attentiveness sessions only), 3) performance of a fatiguing contraction at 20% MVC force with the handgrip muscles (simultaneous mental math task during stressor and mental attentiveness sessions), and 4) recovery MVCs and assessments of cognitive and physiological arousal immediately after the fatiguing contraction and 2, 5, and 10 min of recovery. The control, stressor, and mental attentiveness sessions were randomized for all subjects. Experimental sessions were ≥7 d apart.

Initial measures. Two to three MVCs were performed at the beginning of each session. The peak MVC was used to calculate the required force for the fatiguing contraction at 20% MVC. EMG of the finger flexors and extensors were recorded during the MVC and were used to normalize the EMG during the fatiguing contraction.

Fatiguing contraction. A fatiguing contraction was performed with the handgrip muscles at 20% MVC force during each session. The subject was required to match the target force displayed on the monitor and was verbally encouraged at the start of the task to sustain the force for as long as possible. To minimize the influence of transient fluctuations in motor output on the criteria for task failure, the task was terminated only after the force fell below 10% of the 20% MVC target force for two consecutive seconds.

The following variables were recorded during the fatiguing contraction: HR, blood pressure, finger flexor and extensor EMG, and rating of perceived exertion (RPE) as an index of perceived effort. For RPE, each subject was instructed to focus on assessing effort on the arm muscles performing the fatiguing task. The RPE scale was anchored so that 0 represented the resting state and 10 corresponded to the strongest contraction that the upper limb muscles could perform (CR–10) (3). RPE was recorded at the beginning of the fatiguing contraction and every minute thereafter until task failure. Recovery measures of MVCs and anxiety (VAS) were assessed immediately on task failure and at 2, 5, and 10 min after termination of the fatiguing contraction (Fig. 1).

Cognitive Tasks

Mental math is a well-established psychosocial technique to induce stress (20,27) and was used to increase levels of anxiety and stress in our previous studies (40). During the stressor session, each subject performed serial subtraction of 13 from a four-digit number, with a response required every 3 s. Once the subject made an error in the math or was not able to provide the correct answer within 3 s, he or she was provided with negative feedback regarding his or her performance and instructed to start the mental math again from a new number in the series. A few subjects in the group of veterans with PTSD were unable to perform the cognitive

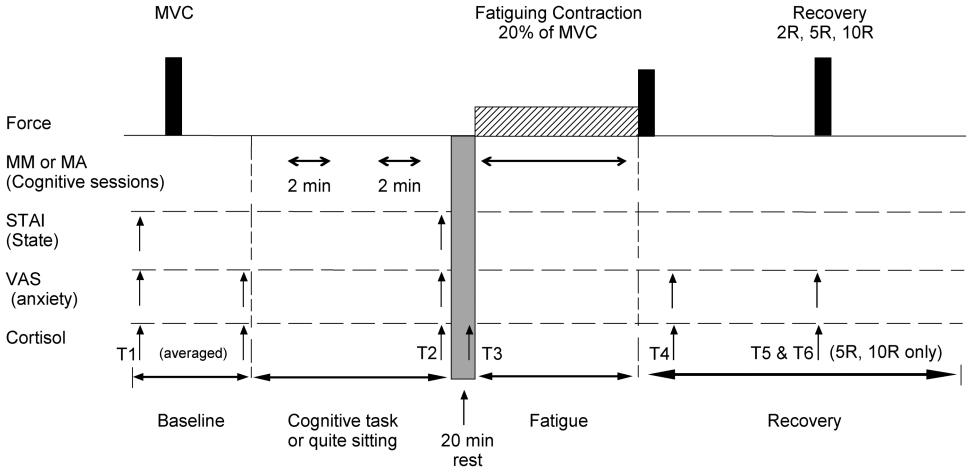

FIGURE 1—Experimental protocol: The *top panel* shows the order of force tasks performed by each subject with the handgrip muscles. Three MVCs were performed. This was followed by a fatiguing contraction at 20% of MVC and recovery MVCs at 2, 5, and 10 min (2R, 5R, and 10R). Mental math (MM), mental attentiveness (MA), or quiet rest (control session) were performed 2 × 2 min (total of 4 min) before and then during the fatiguing contraction for each respective session (*second row*). The state portion of the State-Trait Anxiety Inventory (STAI) questionnaire was assessed twice throughout the protocol. Levels of anxiety using the visual analog scale were assessed throughout the protocol. Cortisol was assessed several times throughout the protocol: twice at baseline and was averaged (T_1), after cognitive tasks (T_2), after 20 min of rest and immediately before the fatiguing contraction (T_3), immediately after fatiguing contraction (T_4), and at 5 and 10 min recovery (T_5 and T_6). Note that the schematic is not to scale for time or force.

task and were then prompted to subtract by 7 from a four-digit number. Each subject performed the mental math during the stressor session only. Moreover, each subject performed the mental math before the fatiguing contraction (2 × 2-min bouts) and during the fatiguing contraction.

The mental attentiveness task required subjects to perform a simple math task that was not designed to induce stress as we have done in a previous study (17,40). Participants continuously subtracted 1 from 50 during the 4 min (2 × 2-min bouts) while at rest before the fatiguing contraction and during the fatiguing contraction in the mental attentiveness session.

Mechanical Recordings

Each subject was seated upright in an adjustable chair with the left arm slightly abducted and the elbow, forearm, and wrist resting on a padded support at or slightly above heart level. The elbow joint was flexed to 90° so that the forearm was horizontal to the ground and the wrist was midway between supination and pronation. The motor task involved gripping a custom-made adjustable handgrip dynamometer as previously described (18). The tensile force detected by the transducer was recorded online at 1000 samples per second using Biopac Pro software (Biopac Systems Inc., Goleta, CA) and displayed on a 15-inch monitor 1.0 m in front of the subject. Each subject was asked to trace the horizontal force signal for as long as possible during the fatiguing contraction. Gain of the visual force feedback was consistent across sessions and participants. The force signal appeared on the screen from the left side of the monitor at 2 $cm \cdot s^{-1}$.

Electrical Recordings

EMG signals were recorded with circular bipolar surface disposable electrodes (Ag–AgCl, 8 mm in diameter, 16 mm between electrodes) that were placed over the flexor digitorum and extensor digitorum muscles. The bipolar electrode configuration was placed longitudinally over the muscle belly midway between the origin and the insertion for each muscle, according to the European recommendations for surface EMG (16). Reference electrodes were placed on a bony prominence at the elbow. Skin surface was abraded and prepared with alcohol and conductive gel before electrode placement. The EMG signal was amplified (1000×), with a band-pass, Butterworth, infinite impulse response filter (20–500 Hz) and an optimal Q of 0.707 and was sampled at 1000 samples per second with Biopac systems. Common mode rejection ratio was 110 dB, and differential input impedance was 2 MΩ.

Cardiovascular Measurements

HR and blood pressure were monitored at rest (baseline) and also during the fatiguing contraction. HR was recorded with an HR monitor (Polar F1, Oulu, Finland) placed against the skin around the subject's chest wall at heart level. Blood pressure was monitored with an automated wrist cuff (HEM-670IT; OMRON Electronic Components, Schaumberg, IL). The blood pressure cuff was placed around the wrist of the right hand, with the hand placed on a table adjacent to the subject at heart level. The automated blood pressure signal was calibrated to a manual blood pressure at each session. Blood pressure and HR were monitored and documented at the start of the contraction and every minute thereafter until task failure.

Cognitive Assessment of Arousal

Cognitive levels of anxiety were assessed throughout the protocol using VAS and the state portion of the STAI

questionnaire as we have detailed previously (22,40). In brief, the VAS involved a 10-cm line anchored at the far left by "not at all anxious" and at the far right by "very anxious." Anxiety was defined as the emotional changes perceived by the subject that was above and beyond the expectation for his or her level of exertion (7). The subject indicated his or her level of anxiety on the horizontal line of the scale. VAS scores for anxiety were recorded at seven time points during the protocol: two baseline assessments, immediately after the cognitive task or quiet sitting and before the start of the fatiguing contraction, immediately after the fatiguing contraction, and then at 2, 5, and 10 min after the fatiguing contraction (Fig. 1). The STAI-state questionnaire involved 20 statements that required a response on a four-point Likert-type scale. STAI was completed at baseline and after mental math tasks (Fig. 1).

Hormonal Assessment of Arousal: Salivary Cortisol

Salivary cortisol, a measure of adrenal output of free cortisol (32), was assessed during each experimental session (27). Saliva was collected using an oral swab (Salimetrics LLC, State College, PA) according to the manufacturer's recommendations and was stored at −20°C for later analysis. Free cortisol levels were measured using an enzymatic immunoassay (Salimetrics LLC). A previous study has established the reliability of this technique (32). Intrareliability and interreliability coefficients of variation (CV) of <6% have been established in this laboratory.

All experimental sessions were performed in the afternoon because of the circadian rhythm of glucocorticoid (cortisol) production and release from the hypothalamic–pituitary–adrenal axis (34). Eight salivary cortisol samples were collected throughout each experimental session: two baseline samples (averaged) before any intended arousal (T_1), a sample immediately after the cognitive task (2 × 2-min bouts of mental math or mental attentiveness) or quiet sitting (control session) (T_2), a sample after a 20-min rest and before the fatiguing contraction (T_3), immediately after the fatiguing contraction (T_4), and then 5 and 10 min after the fatiguing contraction (T_5 and T_6) (Fig. 1). Free cortisol takes approximately 10–20 min to peak in the saliva (after onset of stress) and eventual release from the adrenal glands (32), necessitating the rest after onset of cognitive tasks and the fatiguing contraction.

Data Analysis

The MVC force was quantified as the average force over a 0.5-s interval that was centered about the peak of the MVC. The maximal EMG for finger flexors and finger extensors was determined as the root mean squared (RMS) value over a 0.5-s interval about the same interval of the MVC force measurement. The maximal EMG value measured during the handgrip MVC was used to normalize the RMS EMG values recorded during the fatiguing contraction for both the finger flexor and finger extensor muscles. Force fluctuations were quantified by normalizing the standard deviation (SD) of the force to the mean of the force (coefficient of variation [CV] of force = [SD of force/ mean of force] × 100) (10). The RMS EMG of the finger extensor and flexor muscles and force fluctuations were measured during the fatiguing contraction at the following time intervals: the first and last 20 s of task duration and the 10 s at either side of 25%, 50%, and 75% of time to failure. Rates of change for several variables were calculated by subtracting the value at or immediately after task failure from the baseline value and normalizing the absolute change to the average time to failure.

HR and MAP were recorded at rest for 1 min and during the fatiguing contraction and were reported at the start and end of task and at 25%, 50%, and 75% of time to failure. MAP was calculated for each time point with the following equation: MAP = DBP + 1/3 (SBP−DBP) where SBP is systolic blood pressure and DBP is diastolic blood pressure.

Statistical Analysis

Data are reported as means ± SD within the text and displayed as means ± SEM in the figures. Depending on distribution of the data, an independent *t*-test or Mann–Whitney test was used to compare differences in mean values. We estimated the number of participants needed in each group from a power analysis to obtain >90% power and an $\alpha = 0.05$ level of significance.

Differences in means for control subjects and veterans with PTSD were compared for: 1) various physical characteristics including age, BMI, STAI (trait and state) anxiety levels, PCL-C, and depression (BDI) scores; 2) time to failure, MVC force, and percent reduction in MVC force; and 3) baseline levels of HR and MAP. Mixed factorial two-way ANOVAs with repeated measures over time and group (PTSD vs controls) as a between-subject factor were used to compare the various dependent variables. A separate analysis was also performed to compare control versus stressor and control versus mental attentiveness. Repeated-measure analyses were performed to determine differences from baseline to fatigue (fatigue effect) and from fatigue to recovery (recovery effect), as well as during the fatiguing contraction (time effect: 0%, 25%, 50%, 75%, and 100% of time to failure). Specifically, the statistical designs were as follows: 1) time × group for force fluctuations, EMG, MAP, and HR during the fatiguing contraction; and 2) fatigue × group or recovery × group for comparison of MVC and levels of anxiety (VAS) before and after fatigue. η^2 was calculated using the type III sum of squares (SPSS output) to demonstrate the percentage of variance in each of the effects or interaction. Pairwise comparisons were assessed using a *t*-test where appropriate with Bonferroni *post hoc* adjustment. Cohen *d* was calculated for each pairwise comparison. For within-subject comparisons, the correlation between the two means was used to correct for dependence among means. The strength of an association is reported as the Pearson product–moment correlation coefficient (r^2).

A significance level of $P < 0.05$ was used to identify statistical significance.

To determine whether antidepression or analgesic medications altered time to failure or reduction in strength, an ANOVA with repeated measures was performed, with session (control vs stressor) and group (medications vs no medications) as between-subject factors used to determine differences in time to failure, initial maximal strength (MVCs), reductions in strength (%), and force fluctuations (CV%). A separate ANOVA was performed for those taking pain medications and antidepression medications. Analysis was performed with SPSS (Version 19).

RESULTS

Subject Characteristics

Veterans with PTSD were older by 7 yr, had a greater BMI, levels of trait (general) anxiety, and symptoms of PTSD and depression ($P < 0.05$; Table 1). There was no difference in physical activity levels between the PTSD and control subjects ($P > 0.05$).

Aim 1: Control Session Comparison of Groups: Controls versus PTSD

Time to task failure and maximal strength. Time to task failure was briefer for veterans with PTSD compared with controls (24% difference between groups, $P = 0.02$; Fig. 2A). Time to failure was not associated with physical activity levels ($r = -0.05$, $P = 0.77$), BMI ($r = -0.26$, $P = 0.10$), or age of the individual ($r = 0.21$, $P = 0.13$). Maximal handgrip strength was similar across groups at baseline, after the fatiguing contraction, and throughout recovery ($P > 0.05$; Fig. 2B). Reduction in strength was similar for the controls (48% ± 15%) and veterans with PTSD (53% ± 14%, $P = 0.23$, $d = 0.34$). Maximal strength was significantly lower at 10 min of recovery than at baseline (recovery effect, $P < 0.001$) for both veterans with PTSD (14% ± 11%) and control group (17% ± 9%, $P = 0.4$, $d = 0.30$). Time to failure was similar for those taking antidepression medications (7.3 ± 2.0 min) compared with those not taking antidepression medications (7.3 ± 3.4 min, $P = 0.99$, $d = 0.00$). Similarly, in the control session, time to failure was similar for veterans taking pain medications (7.3 ± 2.0 min) compared with those not taking pain medications (7.4 ± 3.3 min, $P = 0.90$, $d = 0.04$). The reduction in strength was also similar between veterans taking antidepression medications (51.8% ± 15.4%) and those not taking antidepression medications (54.0% ± 13.3%, $P = 0.74$, $d = 0.15$) for the control session.

Force fluctuations. Force fluctuations (CV of force, %) increased during the fatiguing contraction (time effect, $P < 0.001$, $\eta^2 = 0.66$) with no group effect ($P = 0.13$, $\eta^2 = 0.06$). However, veterans with PTSD had a greater rate of increase in CV compared with the control group ($0.81 \pm 0.4\%\cdot min^{-1}$ and $0.48 \pm 0.3\%\cdot min^{-1}$, respectively, $P = 0.009$, $d = 0.92$; Fig. 2C). Force fluctuations were greater at task failure for the veterans with PTSD compared with the control subjects ($P = 0.03$, $d = 0.72$). The rate of increase in fluctuations was correlated with symptoms of PTSD (PCL-C) ($n = 31$, 10 controls, $r = 0.43$, $P = 0.005$): individuals with more PTSD symptoms had a greater rate of increase in CV of force. There were no main effects or interactions for veterans on antidepression or pain medication ($P < 0.05$).

Force fluctuations increased similarly for veterans taking antidepression medications compared with veterans not taking

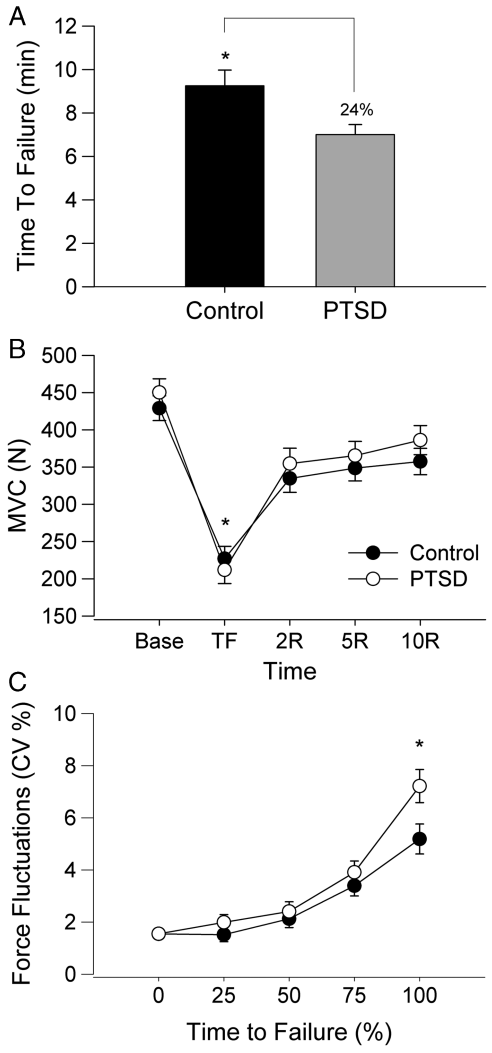

FIGURE 2—A, Time to task failure for veterans with PTSD and control subjects for the 20% fatiguing contraction with the handgrip muscles. Veterans fatigued more quickly than control subjects ($P < 0.01$). B, MVC force of the handgrip muscles for veterans with PTSD (*open symbols*) and control subjects (*closed symbols*) are shown at baseline (Base), at task failure (TF), and at 2, 5, and 10 min throughout recovery (2R, 5R, and 10R). Maximal strength was reduced after the low-intensity fatiguing contraction ($P < 0.05$, * indicates significance). C, Force fluctuations (coefficient of variation [CV]) for veterans with PTSD and control subjects throughout the fatiguing contraction. The rate of increase in force fluctuations was greater for veterans with PTSD (*$P < 0.01$). Values are presented as mean ± SE at 25% increments of the time to task failure.

antidepression medications (time × group, $P = 0.15$, $d = 0.02$). There was no overall group effect ($P = 0.77$, $d = 0.00$). Force fluctuations tended to increase more throughout the fatiguing contraction for veterans taking pain medications compared with veterans who were not taking pain medications (time × group, $P = 0.05$, $d = 0.02$).

EMG activity. Finger flexor and extensor EMG increased throughout the fatiguing contraction for the control group and veterans with PTSD (time effect, $P < 0.001$, $d = 0.52$ and $d = 0.29$, respectively), with no interactions ($P > 0.05$, $d = 0.01$ and $d = 0.02$, respectively) and no group effects ($P > 0.05$, $d = 0.02$, and $d = 0.00$, respectively). Finger flexor EMG increased from 17.9% ± 4.9% to 30.6% ± 10.2% in controls and from 12.5% ± 4.3% to 28.8% ± 15.5% in veterans with PTSD from the start to task failure. Extensor EMG increased from 22.0% ± 10.3% to 34.5% ± 11.7% in controls and from 24.2% ± 12.5% to 37.2% ± 16.4% in veterans with PTSD from the start to task failure.

Cardiovascular response during the fatiguing contraction. HR values were greater at baseline for veterans with PTSD compared with control subjects ($P = 0.03$, $d = 0.74$). HR increased throughout the fatiguing contraction (time effect, $P < 0.001$, $\eta^2 = 0.60$) similarly for both groups (from 72 ± 13 to 87 ± 18 bpm for controls and from 74 ± 11 to 89 ± 10 bpm for PTSD; time × group, $P = 0.84$, $\eta^2 = 0.00$) with no overall group effect ($P = 0.60$, $\eta^2 = 0.008$). Baseline HR correlated with symptoms of PTSD, quantified by the PCL-C ($n = 31$, 10 controls, $r = 0.46$, $P = 0.02$), suggesting that subjects with greater symptoms of PTSD have elevated levels of basal HR.

MAP increased throughout the fatiguing contraction (time effect, $P < 0.001$, $\eta^2 = 0.76$) similarly for both groups (from 95 ± 9 to 116 ± 10 mm Hg for controls and from 99 ± 9 to 124 ± 9 mm Hg for PTSD; time × group, $P = 0.67$, $\eta^2 = 0.01$) but was elevated more at task failure in veterans with PTSD compared with controls ($P = 0.02$, $d = 0.79$).

To understand the cardiac workload during the fatiguing contraction, rate pressure product was quantified (HR × MAP) (14). The rate pressure product increased throughout the fatiguing contraction (time effect, $P < 0.001$, $\eta^2 = 0.78$) but was overall greater for those with PTSD (from 8.1 ± 2.7 × 10^3 to 12.0 ± 3.3 × 10^3 mm Hg·bpm for controls and from 9.5 ± 1.8 × 10^3 to 14.1 ± 2.0 × 10^3 mm Hg·bpm for PTSD; group effect, $P = 0.04$, $\eta^2 = 0.05$).

RPE. This increased throughout the fatiguing contraction (time effect, $P < 0.001$, $\eta^2 = 0.96$) for both the control subjects and veterans with PTSD (time × group, $P = 0.92$, $\eta^2 = 0.00$) with no group effect ($P = 0.24$, $\eta^2 = 0.04$). The rate of increase was significantly greater for veterans with PTSD than for control subjects as the time to failure for those with PTSD was briefer (1.1 ± 0.14 and 0.84 ± 0.13 min^{-1} for control and PTSD subjects, respectively, $P < 0.001$, $d = 1.93$).

Cognitive assessments of anxiety. State anxiety (STAI) was elevated for the veterans with PTSD at baseline and before the fatiguing contraction compared with the control subjects (43.2 ± 9.8 vs 29.8 ± 8.7, respectively, $P < 0.001$, $d = 1.45$). Levels of anxiety assessed by the VAS increased after the fatiguing contraction (fatigue effect, $P = 0.01$, $\eta^2 = 0.27$) and were greater for veterans with PTSD (from 0.79 ± 0.64 to 1.6 ± 1.3 in controls and from 2.0 ± 2.3 to 2.3 ± 2.5 in PTSD, $P = 0.05$, $\eta^2 = 0.12$). Trait (general) levels of anxiety were greater for veterans with PTSD (Table 1) and also correlated with their symptoms on the PCL-C scale ($r = 0.92$, $P < 0.001$), indicating that the subjects who had greater symptoms of PTSD had greater levels of general anxiety.

Aim 2: Comparison of the Stressor and Control Session: Controls versus PTSD

Time to failure. Time to failure was similar for the control and stressor (session effect, $P = 0.16$, $\eta^2 = 0.07$) for both groups (session × group effect, $P = 0.43$, $\eta^2 = 0.02$; Fig. 3A). Time to failure was less for both the control and stressor sessions for the veterans with PTSD compared with the control subjects (24% for control session and 25% for stressor session; group effect, $P = 0.05$, $\eta^2 = 0.17$). The mental attentiveness task did not influence time to failure (7.4 ± 3 vs 7.8 ± 4 min, respectively; session effect, $P = 0.33$, $\eta^2 = 0.04$) for either group (session × group effect, $P = 0.50$, $\eta^2 = 0.02$). Similar to the control session, time to failure was not different in the stressor session for those taking medications compared with those who were not ($P > 0.05$).

Maximal strength. Maximal strength was similar between sessions (session effect, $P = 0.31$, $\eta^2 = 0.04$) for control subjects and veterans with PTSD (session × group effect, $P = 0.45$, $\eta^2 = 0.05$). Reduction in strength, however, was greater for the control session compared with the stressor session (session effect, $P = 0.02$, $\eta^2 = 0.18$; Fig. 3B) for both controls and veterans with PTSD (session × group, $P = 0.38$, $\eta^2 = 0.02$) with no group effect ($P = 0.92$, $\eta^2 = 0.00$). Neither maximal strength nor the reduction in strength was different for the control and mental attentiveness sessions ($P > 0.05$).

Force fluctuations. Force fluctuations (CV) increased for both control and stressor sessions (time effect, $P < 0.005$, $\eta^2 = 0.66$). Fluctuations increased at a greater rate for the veterans with PTSD compared with control subjects (group effect, $P = 0.01$, $\eta^2 = 0.15$; Fig. 3C).

Antidepressants did not influence CV of force for either group (time × group, $P = 0.32$, $\eta^2 = 0.00$) or session (session × group effect, $P = 0.74$, $\eta^2 = 0.00$). Force fluctuations did increase more throughout the fatiguing contraction for veterans taking pain medications for both sessions compared with veterans who were not taking pain medications (time × group, $P < 0.001$, $\eta^2 = 0.005$).

RMS EMG activity. Finger flexor and finger extensor EMG increased throughout the fatiguing contraction (time effect, $P < 0.001$) for all three sessions ($P > 0.05$), with the stressor having no effect and no differences between the veterans with PTSD and control subjects ($P > 0.05$). Finger flexor EMG increased from 14.2% ± 5.4% to 30.0% ± 13.9%

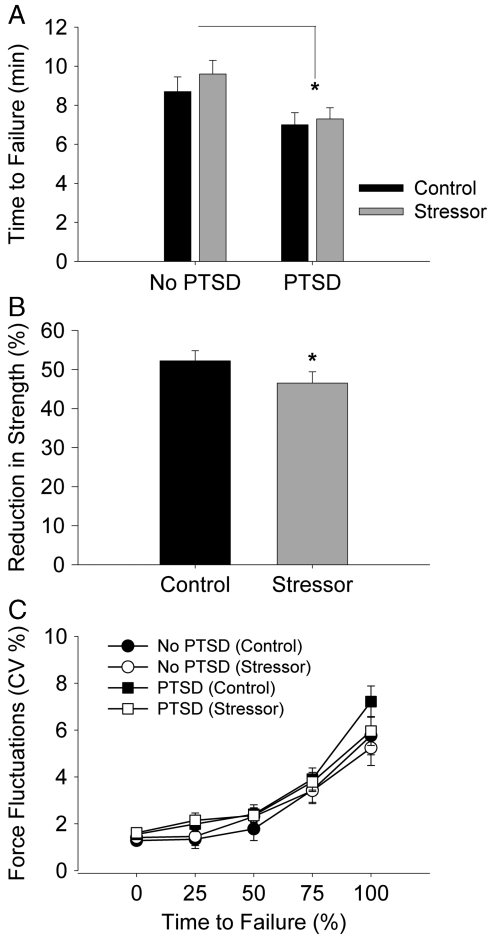

FIGURE 3—A, Time to failure was similar for the control and stressor sessions ($P > 0.05$) but, overall, was briefer for the veterans with PTSD ($P < 0.05$). B, Reductions in strength for the control and stressor sessions were combined for the two groups. Reductions in strength were less for the stressor session compared with the control session for both veterans with PTSD and control subjects ($P < 0.05$). C, Force fluctuations for veterans with PTSD (*square symbols*) and control subjects (*circle symbols*) throughout the fatiguing contraction for the control session (*closed symbols*) and the stressor session (*open symbols*). Coefficient of variation of force (CV, %) was greater for veterans with PTSD than controls ($P < 0.05$). Values are presented as mean ± SE at 25% increments of the time to task failure (*$P < 0.05$).

during the fatiguing contraction in the control session and from 17.6% ± 10.7% to 30.0% ± 11.2% during the fatiguing contraction in the stressor session. Mental attentiveness did not influence EMG during the fatiguing contraction ($P > 0.05$).

Cardiovascular response during the fatiguing contraction. HR values were overall greater during the fatiguing contraction for the stressor session compared with the control session (session effect, $P = 0.03$, $\eta^2 = 0.07$; Fig. 4A). HR increased at a greater rate for the stressor session compared with the control session (session × time, $P = 0.008$, $\eta^2 = 0.008$; Fig. 4A) for both the control subjects and the veterans with PTSD throughout the fatiguing contraction (session × time × group, $P = 0.22$, $\eta^2 = 0.006$). A pairwise correction indicated that there was a significant difference between the control and stressor session HR from the start of the task to 50% of task failure ($P < 0.01$). The standardized difference at the start of the contraction (time point 0) for HR was $d = 0.45$; 25% of task failure, $d = 0.68$; and at 50% of task failure, $d = 0.49$.

MAP increased throughout the fatiguing contraction (time effect, $P < 0.001$, $\eta^2 = 0.45$) similarly for both the control subjects and the veterans with PTSD (session × time × group, $P = 0.58$, $\eta^2 = 0.003$; Fig. 4B) with no effect of session ($P = 0.07$, $\eta^2 = 0.03$) or group ($P = 0.55$, $\eta^2 = 0.013$). A pairwise correction indicated that there was a significant difference in MAP between the control and stressor sessions from the start of the task to 50% of task failure ($P < 0.01$). The standardized difference at the start of the contraction (time point 0) for MAP was $d = 0.72$; 25% of task failure, $d = 0.72$; and at 50% of task failure, $d = 0.45$.

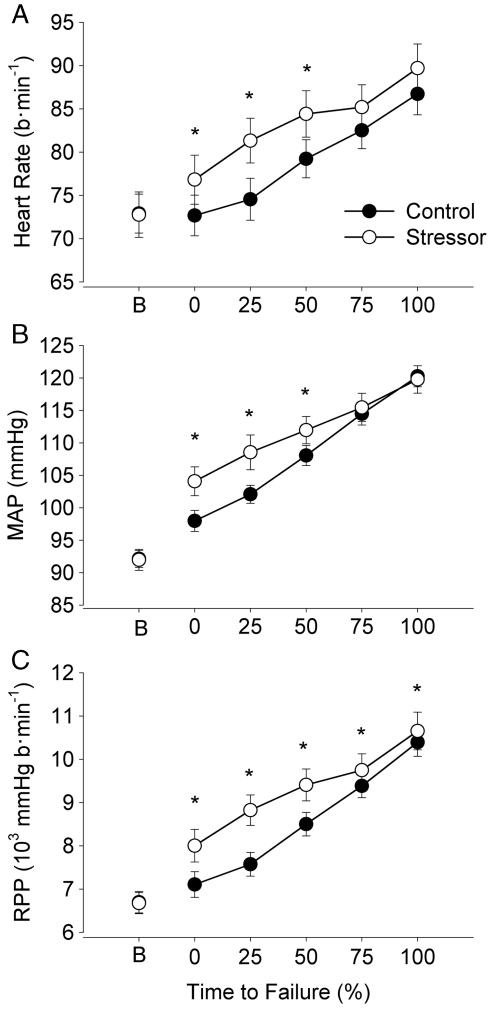

FIGURE 4—HR (A), mean arterial pressure (MAP; B), and rate–pressure product (RPP; C) for the control (*closed symbols*) and stressor sessions (*open symbols*) for the combined groups. HR and RPP were greater throughout the stressor session for both groups (group effect, $P < 0.05$). There was a trend for a greater MAP throughout the stressor session ($P = 0.07$) compared with the control session. Values are presented as mean ± SE at 25% increments of the time to task failure. The *disconnected symbol* indicates baseline (B) values for each variable (*$P < 0.01$).

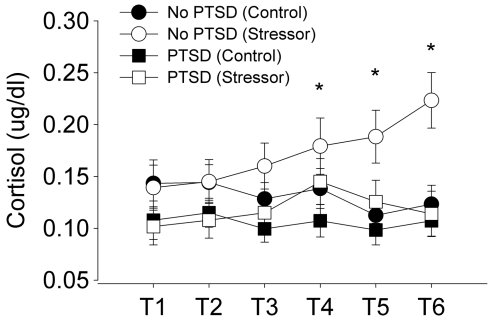

FIGURE 5—Cortisol for the control and stressor sessions in veterans with PTSD and healthy control subjects (no PTSD). Time points throughout the protocol are as follows: T_1 = average of two baseline time points before any experimental procedures; T_2 = immediately after exposure to stressful cognitive task; T_3 = after a 20-min wait, before the fatiguing contraction; T_4 = immediately after fatiguing contraction (with the addition of the cognitive task during the stressor session); T_5 = after 5 min of recovery; and T_6 = after 10 min of recovery (*$P < 0.05$, significant difference between control and stressor sessions).

The rate pressure product increased more for the stressor session than for the control session for both groups (session × time, $P = 0.03$, $\eta^2 = 0.02$; Fig. 4C) with no group effect ($P = 0.69$, $\eta^2 = 0.006$). A pairwise correction indicated that there was a significant difference between the control and stressor sessions' rate pressure product from the start of the task to task failure ($P < 0.01$). The standardized difference at the start of the contraction (time point 0) for the rate pressure product was $d = 0.75$; 25% of task failure, $d = 0.97$; at 50% of task failure, $d = 0.67$; at 75% of the task failure, $d = 0.25$; and 100% of task failure, $d = 0.07$.

Cognitive assessments of anxiety. For both sessions, state anxiety (STAI) was greater for veterans with PTSD (group effect, $P < 0.001$, $\eta^2 = 0.37$). Anxiety increased after exposure to the cognitive stressor (session × time, $P < 0.001$, $\eta^2 = 0.16$) similarly for the control subjects and veterans with PTSD (session × time × group, $P = 0.84$, $\eta^2 = 0.00$). Levels increased from 44.9 ± 10.6 to 56.6 ± 9.0 for veterans with PTSD and from 32.7 ± 10.6 to 41.8 ± 10.1 for the control subjects.

Anxiety (VAS) increased after the cognitive stressor compared with quiet sitting (36% for controls and 37% for veterans with PTSD; session × time, $P < 0.001$, $\eta^2 = 0.07$) and after the fatiguing contraction (compared to baseline measures) while simultaneously performing the stressor task (51% for controls and 47% for veterans with PTSD; session × fatigue, $P = 0.01$, $\eta^2 = 0.04$). Anxiety (STAI or the VAS) did not increase for either group for the mental attentiveness task ($P > 0.05$).

Hormonal assessment of arousal. Cortisol was similar at baseline for the control and stressor session (session effect, $P = 0.82$, $\eta^2 = 0.00$) for both PTSD and control participants (session × group, $P = 0.91$, $\eta^2 = 0.00$). Cortisol increased from baseline (T1) to 20 min after the stressor (T4) (session × time, $P = 0.02$, $\eta^2 = 0.03$) and after the fatiguing contraction (T5) (session × time, $P < 0.001$, $\eta^2 = 0.05$) for the stressor session and throughout recovery for the control participants only during the stressor session (session × time × group, $P = 0.04$, $\eta^2 = 0.02$; Fig. 5).

DISCUSSION

PTSD can be a debilitating cognitive and emotional disorder that significantly affects quality of life. This study demonstrates for the first time that PTSD can also cause decrements in motor performance, resulting in greater fatigability during performance of submaximal motor tasks of the upper limb. The novel findings from this study were 1) that time to failure was briefer and force fluctuations increased at a greater rate during a low-intensity contraction in handgrip muscles of veterans with PTSD than healthy control subjects and 2) that an acute cognitive stressor did not alter muscle fatigue for handgrip muscles of PTSD or healthy control subjects.

Despite the fact that the groups were fatigued to the same magnitude (similar reduction in strength), veterans with PTSD were unable to sustain the submaximal contraction for as long as the control subjects did and therefore fatigued more rapidly. In healthy young men and women, exposure to an acute cognitive stressor resulted in a briefer time to failure that was paralleled by greater indices of sympathetic activation and was associated with maximal strength (40). EMG patterns indicated that activation of the motor unit pools was similar at task failure, and therefore, the earlier task failure could not be attributed to the lack of muscle activation. Sympathetic activity, in contrast, is altered in those with PTSD (36), which may contribute to the inability to sustain a submaximal contraction. For example, greater sympathetic activity is associated with changes in the contractile force of the muscle (4), reduced blood perfusion to the muscle caused by excessive vasoconstriction (38), and altered proprioceptive feedback to the motoneuron in the spinal cord (15). Based on previous findings on elbow flexor muscles of young healthy men and women (40), one possibility for the greater muscle fatigability in veterans with PTSD is sympathetically induced vasoconstriction of the muscle, reducing the amount of blood perfusion and therefore oxygen supply to the exercising muscle.

An alternative explanation may be that greater fatigability in PTSD was mediated by adaptations within the motor cortex. PTSD is a centrally mediated disorder and has been associated with changes in brain structure, size, and function. Changes are observed in the hippocampus, amygdala, and prefrontal cortex (24), and these changes are known to be associated with a decrease in the norepinephrine and serotonin transmission within the brain in those with PTSD (36). The motor cortex, although outside the circuitry that is usually involved with the psychological manifestations of PTSD, is functionally connected to these affected regions. In fact, the motor cortex exhibits altered excitability and inhibition in resting conditions and during voluntary contractions of the first dorsal interosseous muscle

in patients with PTSD (33). In addition, compromised inhibitory control during executive functioning has been demonstrated under functional magnetic resonance imaging in those with PTSD (12). The central nervous system necessitates increases in both excitatory and inhibitory activities during motor tasks, and an imbalance in the corticomotor pathway in PTSD may lead to impairments in motor performance. Perceived exertion increased more for veterans with PTSD during the fatiguing contraction, indicating that a submaximal contraction similar in strength was sensed to be increasingly more difficult for veterans with PTSD. Perceived exertion is influenced by feedforward descending drive and peripheral afferent feedback mechanisms (6). The greater perceived effort may play an important role in the accelerated muscle fatigue in veterans with PTSD.

Physical activity levels are shown to be reduced in patients with PTSD (8); however, in this study, there were no differences in physical activity levels and physical activity was not associated with time to failure. In addition, BMI was higher in those with PTSD but was also not associated with the briefer time to failure demonstrated by PTSD. Thus, veterans with PTSD were not more fatigable because they were less active or less fit.

Motor performance in PTSD was not exacerbated by acute cognitive stress. Contrary to our expectations, neither control subjects nor the veterans with PTSD demonstrated a decrement in time to failure when exposed to the acute cognitive stressor. Possible reasons for this are that 1) this study involved only men and/or 2) the greater fatigability when exposed to a cognitive stress may be dependent on the muscle group. A previous study indicated that time to failure is reduced in young healthy individuals when exposed to an acute cognitive stressor for the elbow flexor muscles, and this difference was more prominent in women (40). Nonetheless, time to failure was reduced by ~25% in the veterans with PTSD compared with the control subjects, and this was similar across the two sessions. This may indicate a stronger influence of chronic stress condition (overactive sympathetic drive and/or corticomotor hyperexcitability) (33,36) on the performance of submaximal motor tasks compared with acute stress in men. Furthermore, mental attentiveness tasks did not influence motor performance in this study.

Interestingly, the reduction in maximal strength was less for the stressor session compared with the control session for both groups, despite no differences in time to failure across sessions. Anxiety and HR were greater during the fatiguing contraction, with MAP being marginally significant in the stressor session than in the control session. This indicates that the acute activation of sympathetic activity was probably greater for the stressor session than for the control session for the control and PTSD groups. Evidence suggests that increases in sympathetic activation can potentiate (increase force) Type II fibers (4) and increase the contractility of the muscle. Therefore, during a high-intensity contraction, where Type II fibers are activated in addition to Type I fibers, the stress-induced sympathetic activation may potentiate force leading to greater muscle activation, stronger MVCs after the fatiguing contraction, and therefore less of a reduction in maximal strength.

Influence of medications on motor performance in veterans with PTSD. Pain is comorbid with PTSD (26). Table 1 indicates the number and percentage of veterans who were taking prescribed medications for pain. Pain medications did not appear to influence maximal strength (MVC), the reduction in strength, or the time to failure of the submaximal fatiguing contraction. Veterans taking pain medication, however, were less steady during the fatiguing contraction compared with those who were not taking pain medication. It is unknown how pain medication modulated force, but evidence suggests that chronic pain may alter force fluctuations. Stimulation of nociceptors (pain receptors), for example, can decrease the discharge rate of motor units during sustained contractions (13), which can increase the variability of the discharge rate and consequently lead to larger force fluctuations. No chronic pain was reported in the upper extremity for any of the subjects; therefore, this explanation is unlikely.

SSRIs, which were the most common antidepressants prescribed to this group, reduce the reuptake of serotonin in the brain, enhancing brain serotonergic activity and improving mood and cognition (36). Serotonin is also important for motor function and has been implicated in improving motor performance in those who had stroke (25). For example, serotonin as well as other neurotransmitters modulate the excitability of cortical neurons, process sensory input, and coordinate motor output (25). Therefore, it might be expected that the veterans taking SSRIs would have enhanced performance (greater maximal strength, less reductions in strength, longer time to failure, and less motor output variability). We, however, did not find a difference in performance between those taking the medications and those who were not. This is consistent with a study that investigated the effect of both acute and chronic SSRIs on maximal strength and high-intensity exercise (cycling test) in individuals without neurally mediated motor impairment (29). The increased performance with serotonin use shown in this previous study therefore might be specific to the motor impairment and the task being performed. We, however, showed that use of SSRIs in veterans with PTSD did not improve motor performance.

Physiological and psychological indicators of stress. Veterans with a diagnosis of PTSD in this study demonstrated elevated basal levels of anxiety (VAS and STAI-state and trait), HR, and rate pressure product, which is an indicator of cardiac work and myocardial oxygen consumption (14). Importantly, a multisite study demonstrated that HR was a predictor for having and/or developing PTSD (5). Elevated HR and blood pressures are known to be indices of sympathetic activation (11) and are related to greater plasma levels of serotonin, norepinephrine, and epinephrine (neuromodulators) (36). Although these neuromodulators

were not measured in this study, it is likely that they were higher at baseline in the veterans with PTSD. MAP was greater at task failure in veterans with PTSD than in control subjects. This, however, was not associated with the briefer time to failure.

Cortisol increased during the stressor session only for the control subjects and tended to be less in the PTSD subjects. These findings are synonymous with those studies that found lower levels of basal cortisol in patients with PTSD, but contrast with those that have found elevated levels in patients with PTSD when exposed to an acute stressor (see de Kloet et al. [9] for review) because the veterans in this study demonstrated a blunted cortisol response to stress. Anger can contribute to low levels of cortisol (21), although anger in response to the difficult mental math task and/or the feedback from the tester was not quantified. Cortisol is an important stress hormone that contributes to normal functioning of several physiological functions, but the influence of low levels of cortisol during low-intensity fatiguing tasks is unknown but may not serve a significant role during these types of motor tasks because cortisol levels were not associated with motor performance in either group or condition.

Limitations. A limitation of this study was that the majority of the control group consisted of civilians and the subjects with PTSD were war veterans. An ideal comparison (control) group for the subjects with PTSD would be combat veterans without PTSD. Combat veterans without PTSD were actively recruited, but only two fit the criteria to participate in the study. Another limitation of the study is that the data are representative of only men. Female veterans were also actively recruited, and because a small number of women demonstrated interest, only one fit the criteria for the study and therefore her data were not included in the analysis because of the possible sex differences in response. Future studies will need to include women, especially because they can have larger decrements in motor performance to the stress response than men (7,40). A third limitation was the comorbidity of depression in the PTSD population. The comorbidity of depression with PTSD is high and recruitment of those without depression is difficult but may be warranted in future studies. Finally, we did not control for medications and smoking behavior because these would have severely limited recruitment of our patient population. Because results indicated that pain and antidepression medications did not contribute to the greater fatigability in veterans with PTSD, we believe that these medications did not bias this result.

CONCLUSIONS

In summary, this is the first study to demonstrate that fatigability is greater, and steadiness is reduced in male veterans with PTSD compared with healthy controls. Although the mechanisms are not understood, two possibilities for the greater fatigue and force fluctuations in veterans with PTSD are as follows: 1) dysfunctions in the balance of corticomotor excitability and inhibition and 2) sympathetically induced vasoconstriction and reduced blood perfusion to the exercising muscle. Time to failure of the submaximal handgrip task was less for veterans with PTSD in both the control and stressor sessions than that the control subjects. This finding suggests that the influence of chronic stress (long-term alterations in sympathetic activation) in PTSD may be stronger than the influence of an acute stressor on fatiguing motor tasks in men. This finding is important given the increasing prevalence of war veterans diagnosed with PTSD and the functional relevance of these findings to low-intensity motor tasks that are common during work-related and military activities. Because this is the first study to investigate motor performance during low-intensity tasks in veterans with PTSD, future studies are needed to determine the mechanisms for the greater reduction in time to failure in those with PTSD compared with controls and, in particular, the role of sympathetic activation.

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