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Fatigability, Coactivation, and Neuromuscular Responses of the Biceps Brachii and Triceps Brachii Following Sustained, Maximal, Isometric Forearm Flexion to Task Failure

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ABSTRACT

Smith RW, Neltner TJ, Anders JPV, Keller JL, Housh TJ, Schmidt RJ, Johnson GO. Fatigability, Coactivation, and Neuromuscular Responses of the Biceps Brachii and Triceps Brachii Following Sustained, Maximal, Isometric Forearm Flexion to Task Failure. **JEPonline** 2021;24(3):55-74. This study examined the fatigue-induced changes in torque, coactivation, and neuromuscular responses of agonist and antagonist muscles following a sustained, maximal, isometric forearm flexion task to failure. Eleven men (mean ± SD: age = 21.9 ± 2.1 yr; height = 180.1 ± 6.0 cm; body mass = 86.8 ± 18.4 kg) performed 2 randomly ordered 6-s forearm flexion and forearm extension maximal voluntary isometric contractions (MVIC) before and after a fatiguing task to failure. Electromyographic (EMG) and mechanomyographic (MMG) signals from the biceps brachii (BB) and triceps brachii (TB) were recorded. Repeated measures ANOVAs were used to examine mean differences in MVIC, EMG AMP, EMG MPF, MMG AMP, and MMG MPF. There were parallel decreases in MVIC from pre- to post-fatigue for the BB (9.5%) and TB (6.0%) following the forearm flexion fatiguing task. The EMG AMP from the BB decreased 28% with no changes in any other neuromuscular parameters for the BB or TB. The findings indicated parallel, fatigue-induced decreases in MVIC for forearm flexion and extension, which is likely due to peripheral fatigue during the forearm flexion fatiguing task.

Key Words: Coactivation, Electromyography, Fatigability

INTRODUCTION

A classic definition of fatigue from Bigland-Ritchie and Woods [8] is “an inability of a muscle or a group of muscles to sustain the required or expected force.” (p. 691) Furthermore, Enoka and Duchateau [20,21] indicated that the magnitude of fatigability can be assessed from the pre- to post-fatigue decrease in maximal voluntary isometric contraction (MVIC). The pre- to post-fatigue assessment of MVIC includes contributions from peripheral and central mechanisms and is considered a global performance-related measure of fatigability [3,54,56]. Recently, Dutra et al. [19] used pre- and post-fatigue assessments of MVIC, combined with peripheral nerve stimulation, to delineate changes in force from global, central, and peripheral factors following bouts of severe-intensity cycle ergometry. Furthermore, Pethick et al. [49] utilized pre- and post-fatigue assessments of MVIC combined with electrical stimulation of the femoral nerve to determine central and peripheral fatigue responses following submaximal, intermittent, isometric, leg extensions.

Sustained MVICs have been used to characterize rapidly developing fatigue of the entire motor pathway where, theoretically, the nervous system drives all motor units to maximal force production [56]. For example, previous studies by Carr et al. [12,13] used sustained MVICs to failure to examine the associations between neuromuscular responses and time to task failure. In addition, Butler et al. [11] used a sustained MVIC to investigate the mechanisms responsible for the decline in discharge frequency in motoneurons typically observed following task failure. Thus, sustained MVICs are suitable for the examination of various aspects of the neuromuscular responses that underly global fatigability.

In addition to pre- and post-fatigue assessments of MVIC, electromyographic (EMG) and mechanomyographic (MMG) responses can be used to examine the neuromuscular characteristics of a fatiguing task [12,13,20,31]. The amplitude (AMP) of the EMG signal represents muscle excitation [57] and the mean power frequency (MPF) has been related to muscle fiber action potential (i.e., conduction velocity) [4]. The MMG signal has been described as the mechanical counterpart of the motor unit electrical activity measured by EMG [26]. Under fatiguing conditions, MMG AMP can reflect motor unit recruitment and MMG MPF qualitatively reflects the global firing rate of unfused, activated motor units [6,47]. Thus, simultaneous assessments of EMG and MMG signals have been used to examine fatigue-induced neuromuscular responses and make inferences regarding the nature of motor unit activation strategies [55].

The simultaneous activation of agonist and antagonist muscles during a muscle action has been defined as coactivation [18,39]. Coactivation is typically quantified using the EMG AMP from the agonist and antagonist muscles and expressed as a ratio to evaluate the degree in which the antagonist muscle affects the force production of the agonist muscle [51,59]. For example, Psek and Cafarelli [51] suggested that the decrease in agonist force production following intermittent, isometric, leg extensions to task failure was due to an increase in the activation of the antagonist muscle reflected by a decrease in the agonist/antagonist ratio. In addition, during a fatiguing leg extension task, Weir et al. [59] suggested that the increased coactivation of the biceps femoris may have contributed to fatigue and affected the ability of the quadriceps to produce force. Thus, increased coactivation of the antagonist muscle may contribute to fatigue resulting in a decline in force output of the agonist muscle following a fatiguing task.

The influence of coactivation on fatigability, however, has primarily focused on the lower body across various muscle actions [44,48,51,59]. Only a few studies [10,30,40] have examined coactivation and fatigability of the upper body following a sustained, isometric fatiguing task. Therefore, the purpose of the present study was to examine the fatigue-induced changes in torque, coactivation, and patterns of neuromuscular responses of agonist and antagonist muscles following a sustained, maximal, isometric, forearm flexion task to failure. Based on the results of previous studies [18,39,51,59], it was hypothesized that increased muscle excitation of the antagonist would contribute to a fatigue-induced decrease in the force production of the agonist muscle. In addition, fatiguing the agonist muscle would result in a decrease in the force production of the non-fatigued antagonist muscle [33,41]. Furthermore, fatigability will reflect the typical neuromuscular responses assessed by EMG AMP, EMG MPF, MMG AMP, and MMG MPF following a sustained, maximal, forearm flexion task to failure [12,13].

METHODS

Subjects

Eleven men (mean ± SD: age = 21.9 ± 2.1 yr; height = 180.1 ± 6.0 cm; body mass = 86.8 ± 18.4 kg) volunteered to participate in this study. The subjects were recreationally trained and participated in resistance and/or aerobic exercise at least 3 $d \cdot wk^{-1}$ [2]. All subjects were free of upper body pathologies that would affect their performance. The subjects in the present study were part of a large multiple independent and dependent variable investigation, but none of the MVIC or neuromuscular responses have been published previously. The study was approved by the University Institutional Review Board for Human Subjects (IRB Approval #: 20200120007FB), and all subjects completed a Health History Questionnaire and signed written Informed Consent prior to testing.

Procedures

Time Course of Procedures

Table 1 includes the time course for the procedures of the study. The subjects visited the laboratory on 3 separate occasions (Orientation Session, Testing Visit 1, and Testing Visit 2) each separated by 24 to 48 hrs. The initial laboratory visit was an orientation session where demographic information was recorded, and the subjects were familiarized with the standardized warm-up as well as the testing procedures (Table 1). Testing Visit 1 included the standardized warm-up as well as forearm flexion MVICs and forearm extension MVICs with simultaneous recording of EMG and MMG signals. Testing Visit 2 included the standardized warm-up followed by the pre-fatigue forearm flexion MVICs and forearm extension MVICs, the fatiguing task, and the post-fatigue forearm flexion MVICs and forearm extension MVICs. The pre- and post-fatigue MVICs included the recording of EMG and MMG signals. The data from Testing Visit 1 and the pre-fatigue data from Testing Visit 2 were used to determine the test-retest reliability for the MVICs and neuromuscular parameters.

Table 1. Time Course of the Orientation Session and Testing Visits.

Orientation Session		Testing Visit 1		Testing Visit 2
1. Informed Consent. 2. Health History Questionnaire. 3. Age, height, and body mass were recorded. 4. Familiarized to testing procedures. 5. Standardized warm-up: a. 10 submaximal, reciprocal, concentric, isokinetic repetitions at 180°·s^{-1}. **b.** 2, 6 s forearm flexion MVICs.		1. Standardized warm-up. 2. Forearm flexion MVIC (n=2) and forearm extension MVIC (n=2) in random order.		1. Standardized warm-up. 2. Pre-fatigue forearm flexion MVIC (n=2) and pre-fatigue forearm extension MVIC (n=2) in random order. 3. Fatiguing Task: a. Isometric forearm flexion MVIC until task failure defined as a decrease in torque of 20% of pre-fatigue MVIC. 4. Post-fatigue forearm flexion MVIC (n=2) and post-fatigue forearm extension MVIC (n=2) in random order.

Determination of Maximal Voluntary Isometric Contractions and Forearm Flexion Fatiguing Task

The subjects were positioned in accordance with the Cybex 6000 user manual [16] on an upper body exercise table (UBXT) with the lateral epicondyle of the humerus of the dominant arm (based on throwing preference) aligned with the lever arm of the dynamometer. Once positioned, the subjects performed the standardized warm-up (Table 1) followed by 1-min of rest. The subjects then performed 2 forearm flexion MVICs and 2 forearm extension MVICs in randomized order, for 6 s with 5 s of rest between each repetition at elbow joint angles of 135° and 90°, respectively, on a calibrated Cybex 6000 dynamometer (Cybex, Division of Lumex, Inc., Ronkonkoma, NY, USA). The highest torque value for each movement was selected as the MVIC. The elbow joint angles of 135° for forearm flexion and 90° for forearm extension were selected to reflect the points in the range of motion that approximated maximal isometric torque production for each movement [5,34,37]. Following the MVIC tests during Testing Visit 2 (Table 1), the fatiguing task was performed which involved holding a continuous, isometric forearm flexion MVIC at an elbow joint angle of 135° to task failure. Task failure was defined as a decrease in torque of 20% of the pre-fatigue MVIC. Throughout the fatiguing task, the subjects were provided strong verbal encouragement that resulted in a precipitous end point. During the fatiguing task, torque was tracked and visually inspected by a member of the research team on a monitor that displayed the real-time, digitized torque signal overlayed onto a template identifying the target torque value. Immediately after the fatiguing task (within 5-s), the subjects performed the post-fatigue forearm flexion MVICs and forearm extension MVICs in an identical manner as the pre-fatigue MVICs.

Electromyographic, Mechanomyographic, and Torque Signal Acquisition

During the Testing Visits, bipolar (30-mm center-to-center) EMG electrodes (pregelled Ag/AgCl, AccuSensor; Lynn Medical, Wixom, MI) were attached to the biceps brachii (BB) and the long head of the triceps brachii (TB) of the dominant arm based on the recommendations of the Surface Electromyography for the Non-Invasive Assessment of Muscles [28]. A reference electrode was placed on the styloid process of the radius of the forearm. Prior to electrode placement, the skin was shaved, carefully abraded, and cleaned with alcohol. The electrodes were placed between the medial acromion and the fossa cubit, at one-third the distance from the fossa cubit over the BB. Additional electrodes were placed at 50% of the distance between the posterior crista of the acromion and the olecranon, at 2 finger widths medial to the line over the long head of the TB. Using double-sided adhesive tape, miniature accelerometers (Entras EGAS FT 10, bandwidth 0-200 Hz, dimensions 1.0 × 1.0 × 0.5 cm, mass 1.0 g, sensitivity 550 $mV \cdot g^{-1}$ for the BB, 501.7 $mV \cdot g^{-1}$ for the TB) were placed between the bipolar EMG electrodes to detect the MMG signals for both the BB and TB muscles.

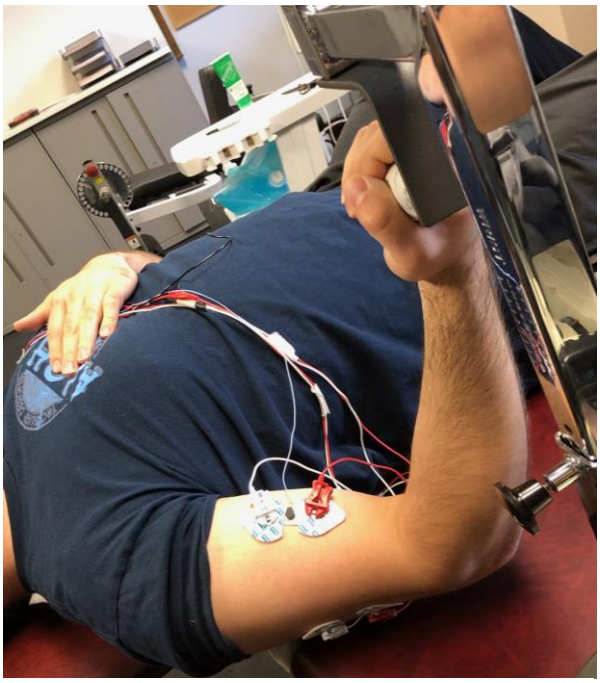

Figure 2. Experimental Set Up.

The raw EMG and MMG signals were digitized at 2000 Hz with a 12-bit analog-to-digital converter (Model MP150; Biopac Systems, Inc.) and stored on a personal computer (Acer Aspire TC-895-UA91 Acer Inc., San Jose, CA, USA) for analyses. The EMG signals were amplified (gain: × 1000) using differential amplifiers (EMG2-R Bionomadix, Biopac Systems, Inc. Goleta, CA, USA; bandwidth — 10-500 Hz). The EMG and MMG signals were digitally bandpass filtered (fourth-order Butterworth) at 10-500 Hz and 5-100 Hz, respectively. Signal processing was performed using custom programs written with LabVIEW programming software (version 20.0f1, National Instruments, Austin, TX, USA). Two second epochs from the center of the 6 s forearm flexion MVIC and forearm extension MVIC were used to calculate the EMG AMP (µVrms), EMG MPF (Hz), MMG AMP ($m \cdot s^2$) and MMG MPF (Hz). The MPF was selected to represent the power density spectrum and was calculated as described by Kwatny et al. [38]. The torque signal was sampled from the digital torque output of the Cybex 6000 dynamometer and stored on a personal computer (Acer Aspire TC-895-UA91 Acer Inc., San Jose, CA, USA) for statistical analysis.

Statistical Analyses

Test-retest reliability for the EMG AMP, EMG MPF, MMG AMP, MMG MPF and MVIC values were assessed with a repeated measures ANOVA to evaluate systematic error and a 2,1 model was used to determine the intraclass correlation coefficient (ICC) [58]. Mean differences for the neuromuscular parameters during forearm flexion MVIC and forearm

extension MVIC were determined by 4 separate 2 (Time: Pre-fatigue and Post-fatigue) × 2 (Muscle: Biceps Brachii and Triceps Brachii) × 2 (Action: Agonist and Antagonist) repeated measures ANOVAs. In addition, a 2 (Time: Pre-fatigue and Post-fatigue) × 2 (Contraction Type: Forearm Flexion and Forearm Extension) repeated measures ANOVA was used to determine the mean differences for the MVIC values. Significant interactions were decomposed with appropriate follow-up repeated measures ANOVAs and *post-hoc* pairwise comparisons. Effect size was reported as η_p^2 and Cohen's *d* for ANOVAs and *post-hoc* pairwise comparisons, respectively. All calculations and statistical analyses were carried out in IBM SPSS v. 26 (Armonk, NY, USA). A P-value ≤0.05 was considered statistically significant, and all the data were reported as mean ± SD.

RESULTS

Reliability

Table 2 includes the test-retest reliability parameters (P-value [systematic error], ICC, $ICC_{95\%}$, and SEM) for EMG AMP, EMG MPF, MMG AMP, MMG MPF, and MVIC. There were no mean differences (P>0.05) for test versus retest for the neuromuscular or MVIC values. The ICC values ranged from 0.367 (Biceps Brachii MMG MPF) to 0.731 (Biceps Brachii EMG AMP).

Table 2. Test-Retest Reliability Data for Maximal Voluntary Isometric Contraction (MVIC) Torque and Neuromuscular Parameters (EMG AMP, EMG MPF, MMG AMP, and MMG MPF) during Forearm Flexion MVIC and Forearm Extension MVIC from Visit 1 vs. Visit 2.

Variables MVIC (mean ± SD)	Test (Visit 1)	Retest (Visit 2)	P	ICC	$ICC_{95\%}$
Forearm Flexion (N·m)	52.4 ± 9.9	54.2 ± 7.1	0.443	0.624	0.084 - 0.882
Forearm Extension (N·m)	49.5 ± 41.0	50.6 ± 10.0	0.769	0.534	-0.095 - 0.851
Neuromuscular Parameters (m ± SD)					
EMG AMP Biceps Brachii (µVrms)	945.4 ± 546.3	789.7 ± 593.8	0.237	0.731	0.299-0.918
EMG MPF Biceps Brachii (Hz)	82.4 ± 9.4	79.4 ± 12.2	0.397	0.468	-0.138-0.821
MMG AMP Biceps Brachii ($m \cdot s^2$)	0.51 ± 0.22	0.45 ± 0.16	0.232	0.558	0.019-0.855
MMG MPF Biceps Brachii (Hz)	21.2 ± 6.0	21.2 ± 6.7	0.988	0.367	-0.326-0.785
EMG AMP Triceps Brachii (µVrms)	765.6 ± 346.9	953.7 ± 544.8	0.168	0.553	0.023-0.852
EMG MPF Triceps Brachii (Hz)	99.2 ± 30.4	91.8 ± 17.4	0.294	0.597	0.064-0.871
MMG AMP Triceps Brachii ($m \cdot s^2$)	0.72 ± 0.51	0.68 ± 0.63	0.789	0.698	0.186-0.910
MMG MPF Triceps Brachii (Hz)	17.0 ± 3.5	19.1 ± 4.9	0.116	0.540	0.011-0.846

P (ANOVA for systematic error), intraclass correlation coefficient (ICC), ICC 95% confidence interval ($ICC_{95\%}$), electromyography (EMG), mechanomyography (MMG), amplitude (AMP), mean power frequency (MPF).

MVIC

The 2 (Time: Pre-fatigue and Post-fatigue) × 2 (Contraction Type: Forearm Flexion and Forearm Extension) repeated measures ANOVA for MVIC indicated no significant Time × Contraction Type interaction (P = 0.423, η_p^2 = 0.65) or main effect for Contraction Type (P = 0.314, η_p^2 = 0.101). There was, however, a significant main effect (collapsed across Contraction Type) for Time (P < 0.001, η_p^2 = 0.724). Thus, the pre-fatigue MVIC (50.9 ± 8.5 N·m) was greater (P<0.001, *d* = 0.452) than the post-fatigue MVIC (46.9 ± 9.2 N·m) (Figure 1) following the forearm flexion MVIC fatiguing task in which the time to task failure was 33.63 ± 8.18 sec.

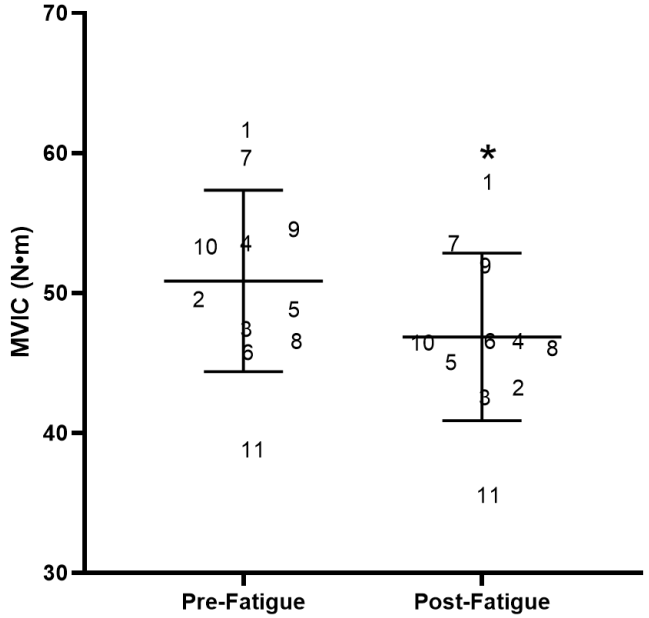

Figure 1. Mean (± SD) and Individual Maximal Voluntary Isometric Contraction (MVIC) Torque Values for Pre-Fatigue and Post-Fatigue (Collapsed Across Forearm Flexion and Extension). Note: Pre-fatigue > Post-fatigue at P<0.05.

Neuromuscular Parameters

Tables 3 to 6 include the individual subject and group (mean ± SD) EMG AMP, EMG MPF, MMG AMP, and MMG MPF parameters, respectively, from the pre- and post-fatigue forearm flexion MVIC and forearm extension MVIC recorded during testing visit 2.

EMG AMP

The 2 (Time: Pre-fatigue and Post-fatigue) × 2 (Muscle: Biceps Brachii and Triceps Brachii) × 2 (Action: Agonist and Antagonist) repeated measures ANOVA for EMG AMP resulted in significant Time × Muscle (P = 0.048, η_p^2 = 0.336) and Time × Action (*p* = 0.010, η_p^2 = 0.500) interactions. Separate, follow-up 2-way (Time × Action) repeated measures ANOVAs were performed for the BB and TB. For the BB, there was a significant 2-way (Time × Action) interaction (P = 0.008, η_p^2 = 0.525) and *post-hoc* paired *t*-tests showed that during the forearm flexion MVIC (when the BB was the agonist), the pre-fatigue EMG AMP (1021.8 ± 557.0 µV)

was greater (P<0.001, *d* = 0.552) than post-fatigue (735.7 ± 474.7 µV). During the forearm extension MVIC (when the BB was the antagonist), there was no significant (P = 0.722, *d* = 0.133) difference in pre-fatigue EMG AMP (80.2 ± 36.9 µV) versus post-fatigue (74.6 ± 46.8 µV). For the TB, there was no significant Time × Action interaction (P = 0.745, η_p^2 = 0.011) or main effect for Time (P = 0.703, η_p^2 = 0.015), but there was a significant (P = 0.002, η_p^2 = 0.652) main effect for Action. When collapsed across Time, EMG AMP from the TB was greater (P = 0.002, *d* = 1.837) during the forearm extension MVIC (when the TB was the agonist = 901.8 ± 594.6 µV) than during the forearm flexion MVIC (when the TB was the antagonist = 123.7 ± 72.1 µV).

Table 3. EMG AMP (µVrms) of the Biceps Brachii and Triceps Brachii during Maximal Voluntary Isometric Contraction (MVIC) Pre-Fatigue and Post-Fatigue.

	Biceps Brachii			
	Agonist		Antagonist	
Subjects	Pre-Fatigue	Post-Fatigue	Pre-Fatigue	Post-Fatigue
1	1831.2	1037.1	118.0	149.6
2	329.3	266.2	58.4	33.0
3	716.1	301.2	81.6	162.7
4	1267.3	619.9	90.2	51.6
5	613.4	402.8	43.4	57.1
6	1162.3	900.7	134.8	31.3
7	482.9	687.3	37.7	93.6
8	2018.8	1890.9	36.5	37.4
9	1205.6	997.7	60.7	36.7
10	1135.3	694.8	84.7	65.0
11	477.2	294.3	136.2	103.2
Mean ± SD	1021.8 ± 557.0	735.7 ± 474.4	80.2 ± 36.9	74.6 ± 46.8

Table 3. EMG AMP (µVrms) of the Biceps Brachii and Triceps Brachii during Maximal Voluntary Isometric Contraction (MVIC) Pre-Fatigue and Post-Fatigue, continued.

	Triceps Brachii		
Agonist		Antagonist	
Pre-Fatigue	Post-Fatigue	Pre-Fatigue	Post-Fatigue
2007.7	2596.5	149.9	116.1
277.0	175.9	60.6	58.7
857.6	814.1	60.1	53.3
1180.7	729.9	100.3	65.4
451.6	461.3	102.0	89.6
1047.7	953.5	164.8	110.3
530.9	828.7	68.5	328.2
1549.0	1488.4	124.5	110.1
576.6	581.9	270.7	220.8
1174.8	900.1	114.5	57.9
443.7	211.9	180.7	113.7
917.9 ± 534.9	885.7 ± 675.1	127.0 ± 62.7	120.4 ± 83.5

EMG MPF

The 2 (Time) × 2 (Muscle) × 2 (Action) repeated measures ANOVA for EMG MPF resulted in a significant 3-way interaction (P = 0.046, η_p^2 = 0.292). Separate, follow-up 2-way (Time × Action) repeated measures ANOVAs were performed for the BB and TB. For the BB, there was no significant Time × Action interaction (P = 0.772, η_p^2 = 0.009) or main effects for Time (P = 0.992, η_p^2 = 0.000) or Action (P = 0.114, η_p^2 = 0.231). For the TB, there was no significant Time × Action interaction (P = 0.277, η_p^2 = 0.117) or main effects for Time (P = 0.699, η_p^2 = 0.016) or Action (P = 0.470, η_p^2 = 0.053). Additional, separate, follow-up 2-way (Time × Muscle) repeated measures ANOVAs were performed for the agonist and antagonist. For the agonist, there was no significant Time × Muscle interaction (P = 0.616, η_p^2 = 0.026) or main effects for Time (P = 0.416, η_p^2 = 0.067) or Muscle (P = 0.055, η_p^2 = 0.320). For the antagonist, there was no significant Time × Muscle interaction (P = 0.282, η_p^2 = 0.115) or main effects for Time (P = 0.605, η_p^2 = 0.028), but there was a significant (P = 0.012, η_p^2 = 0.485) main effect for Muscle. When collapsed across Time, the EMG MPF was greater (P = 0.012, *d* = 0.963) in the TB when it was the antagonist (88.4 ± 11.4 Hz) versus the BB when it was the antagonist (73.0 ± 19.2 Hz).

Table 4. EMG MPF (Hz) of the Biceps Brachii and Triceps Brachii during Maximal Voluntary Isometric Contraction (MVIC) Pre-Fatigue and Post-Fatigue.

	Biceps Brachii			
	Agonist		Antagonist	
Subjects	Pre-Fatigue	Post-Fatigue	Pre-Fatigue	Post-Fatigue
1	85.2	91.0	79.8	79.5
2	72.4	78.8	42.9	38.6
3	82.0	85.7	70.0	72.6
4	71.8	69.4	74.3	71.5
5	96.4	94.9	121.3	99.7
6	96.9	101.4	49.4	82.8
7	73.5	63.8	83.8	41.1
8	101.0	103.9	100.1	102.4
9	69.6	67.0	56.3	76.7
10	63.4	63.5	76.3	72.8
11	84.2	85.2	55.6	63.1
Mean ± SD	81.5 ± 12.5	82.2 ± 14.8	68.7 ± 16.7	72.8 ± 20.1

Table 4. EMG MPF (Hz) of the Biceps Brachii and Triceps Brachii during Maximal Voluntary Isometric Contraction (MVIC) Pre-Fatigue and Post-Fatigue, continued.

Triceps Brachii			
Agonist		Antagonist	
Pre-Fatigue	Post-Fatigue	Pre-Fatigue	Post-Fatigue
90.4	91.4	87.4	89.1
102.9	91.0	79.1	79.5
82.6	109.3	93.9	95.3
76.2	81.4	81.9	91.2
109.5	102.7	88.2	81.9
83.5	83.3	72.5	85.1
68.7	64.4	115.7	39.3
110.3	117.9	115.4	120.5
90.3	86.1	81.0	83.2
98.9	98.3	96.2	80.7
73.5	87.8	94.0	93.9
88.9 ± 13.3	92.1 ± 14.6	91.4 ± 13.9	85.4 ± 19.1

MMG AMP

The 2 (Time) × 2 (Muscle) × 2 (Action) repeated measures ANOVA for MMG AMP resulted in a significant 3-way interaction (P = 0.025, η_p^2 = 0.355). Separate, follow-up 2-way (Time × Action) repeated measures ANOVAs were performed for the BB and TB. For the BB, there was no significant Time × Action interaction (P = 0.082, η_p^2 = 0.272) or main effect for Time (P = 0.278, η_p^2 = 0.116), but there was a significant (p < 0.001, η_p^2 = 0.799) main effect for Action. Collapsed across Time, MMG AMP was lower (P < 0.001, d = 1.895) during the forearm flexion MVIC (when the BB was the agonist = 0.42 ± 0.15 m·s^2) than during the forearm extension MVIC (when the BB was the antagonist = 0.98 ± 0.39 m·s^2). For the TB, there was no significant 2-way interaction (P = 0.115, η_p^2 = 0.229) or main effects for Time (P = 0.590, η_p^2 = 0.030) or Action (P = 0.423, η_p^2 = 0.065).

Table 5. MMG AMP (ms^2) of the Biceps Brachii and Triceps Brachii during Maximal Voluntary Isometric Contraction (MVIC) Pre-Fatigue and Post-Fatigue.

	Biceps Brachii			
	Agonist		Antagonist	
Subjects	Pre-Fatigue	Post-Fatigue	Pre-Fatigue	Post-Fatigue
1	0.61	0.47	1.30	1.93
2	0.66	0.43	1.72	1.48
3	0.45	0.37	0.96	0.83
4	0.62	0.37	0.94	1.24
5	0.66	0.25	0.70	0.61
6	0.58	0.37	0.83	0.81
7	0.36	0.28	0.99	0.77
8	0.28	0.27	0.60	0.62
9	0.63	0.49	0.89	1.47
10	0.34	0.14	0.97	0.83
11	0.30	0.33	0.64	0.44
Mean ± SD	0.50 ± 0.15	0.34 ± 0.10	1.01 ± 0.32	1.00 ± 0.46

Table 5. MMG AMP (ms^2) of the Biceps Brachii and Triceps Brachii during Maximal Voluntary Isometric Contraction (MVIC) Pre-Fatigue and Post-Fatigue, continued.

Triceps Brachii			
Agonist		Antagonist	
Pre-Fatigue	Post-Fatigue	Pre-Fatigue	Post-Fatigue
0.45	0.87	0.96	0.86
2.01	2.77	0.76	0.30
0.70	1.13	0.27	0.25
0.44	0.63	1.09	0.40
0.41	0.52	0.41	0.30
0.48	0.73	0.81	0.51
0.53	0.94	0.73	0.48
0.29	0.32	0.45	0.47
1.48	1.83	1.49	2.81
0.59	0.36	0.59	0.35
0.83	0.39	0.59	0.32
0.75 ± 0.53	0.95 ± 0.74	0.74 ± 0.34	0.64 ± 0.74

MMG MPF

The 2 (Time) × 2 (Muscle) × 2 (Action) repeated measures ANOVA for MMG MPF resulted in no significant 3-way or 2-way interactions (P = 0.221, η_p^2 = 0.145; P>0.05) or main effects for Time (P = 0.068, η_p^2 = 0.295) or Muscle (P = 0.604, η_p^2 = 0.028). There was, however, a significant (P = 0.007, η_p^2 = 0.538) main effect for Action. When collapsed across Time and Muscle, the MMG MPF was higher (P = 0.016, *d* = 0.862) as the agonist (19.3 ± 5.2 Hz) than the antagonist (15.7 ± 2.8 Hz).

Table 6. MMG MPF (Hz) of the Biceps Brachii and Triceps Brachii during Maximal Voluntary Isometric Contraction (MVIC) Pre-Fatigue and Post-Fatigue.

	Biceps Brachii			
	Agonist		Antagonist	
Subjects	Pre-Fatigue	Post-Fatigue	Pre-Fatigue	Post-Fatigue
1	15.3	28.6	14.6	15.6
2	14.2	15.7	15.5	15.0
3	29.7	23.3	14.9	16.6
4	16.5	16.1	11.7	13.3
5	15.5	19.3	15.8	16.9
6	16.2	22.6	14.9	14.3
7	27.1	24.0	13.2	15.6
8	22.5	26.9	16.5	15.7
9	14.3	15.4	14.0	11.1
10	18.1	26.0	14.9	16.2
11	21.2	20.3	14.7	17.7
Mean ± SD	19.1 ± 5.3	21.6 ± 4.7	14.6 ± 1.3	15.3 ± 1.8

Table 6. MMG MPF (Hz) of the Biceps Brachii and Triceps Brachii during Maximal Voluntary Isometric Contraction (MVIC) Pre-Fatigue and Post-Fatigue, continued.

Triceps Brachii			
Agonist		Antagonist	
Pre-Fatigue	Post-Fatigue	Pre-Fatigue	Post-Fatigue
20.0	14.9	14.2	12.7
15.6	14.8	13.4	17.7
18.2	16.1	25.3	24.6
17.6	19.4	15.2	19.2
20.1	16.7	16.9	18.6
18.1	17.4	15.7	17.5
18.3	18.1	13.5	16.6
33.0	28.2	15.3	17.1
13.2	8.6	12.2	13.8
15.0	22.5	19.0	13.8
10.5	22.7	11.6	17.7
18.1 ± 5.7	18.1 ± 5.1	15.7 ± 3.8	17.3 ± 3.3

DISCUSSION

The test-retest reliability analyses for the MVICs and neuromuscular parameters in the present study are presented in Table 2. For forearm flexion MVIC and forearm extension MVIC, there were no significant mean differences for test versus retest and the ICCs ranged from R = 0.534 – 0.624. These ICCs reflected fair to good reliability [14], but were somewhat lower than those (R = 0.65 – 0.96) reported by Colombo et al. [15] and Gaudet et al. [25]. There were also no significant mean differences between test and retest for the neuromuscular parameters (EMG AMP, EMG MPF, MMG AMP, and MMG MPF) from the BB and TB during the forearm flexion MVIC and forearm extension MVIC, respectively. The ICCs for the neuromuscular parameters ranged from (R = 0.367 – 0.731) and were consistent with those (R = 0.36 – 0.99) from previous studies[1,25,27,45,52] during various modes of muscle actions.

Pre-fatigue versus post-fatigue assessments of MVIC represent a global, performance-related measure of fatigability [19,49] from central and peripheral mechanisms [3,56]. Furthermore, the performance of a sustained MVIC can be used to examine fatigue-related changes throughout "…the entire motor pathway" and "…the task for the nervous system is 'maximal' throughout the exercise (i.e., to drive all motor units to produce maximal force)" (p. 543) [56]. In the present study, the time to task failure (33.63 ± 8.18 sec) was similar to those (approximately 20 to 60 sec) of Bigland-Ritchie et al. [9], Carr et al. [12,13], and Moritani et al. [43] during sustained forearm flexion MVICs. Furthermore, there were parallel decreases in the MVIC for forearm flexion (9.5%) and forearm extension (6.0%) following the sustained forearm flexion MVIC to task failure (Figure 1). These findings suggested that both the agonist (BB) and antagonist (TB) muscles experienced fatigue-induced declines in MVIC, even though the level of muscle excitation (EMG AMP) of the antagonist muscle was only 20% of MVIC during the fatiguing task. Furthermore, EMG AMP from the BB decreased

(28%) from pre- to post-fatigue as a result of the sustained forearm flexion MVIC with no change in EMG AMP from the TB which served as the antagonist. Thus, coactivation as defined by the TB EMG AMP versus BB EMG AMP ratio, increased following the fatiguing task due solely to the decrease in EMG AMP from the agonist (BB) with no change in the EMG AMP of the antagonist (TB). Therefore, the parallel decline in MVIC of the BB and TB was not a function of increased excitation of the antagonist (TB).

Neuromuscular parameters from surface EMG and MMG signals have been used to characterize the electrical and mechanical aspects of muscular activity and make inferences regarding motor unit activation strategies during fatiguing tasks [12,13,22,47]. Sustained MVICs are typically characterized by fatigue-induced decreases in EMG AMP and EMG MPF that reflect decreases in muscle excitation and slowing of the muscle fiber action potential conduction velocity (MFAP CV), respectively [4]. Furthermore, during sustained MVICs, MMG AMP, and MMG MPF potentially reflect de-recruitment of high threshold motor units or increases in intra-muscular fluid pressure, as well as reductions in the firing rate of unfused, activated motor units [6,47].

Previous studies [12,36,43,46] have reported fatigue-induced decreases in EMG AMP from the BB as the result of sustained, forearm flexion MVICs. For example, Carr et al. [12] reported significant, negative, slope coefficients for the EMG AMP from the BB versus time relationships for 16 of the 18 subjects during a sustained, forearm flexion MVIC to task failure which was defined as a 45% decrease in MVIC. In the present study, 10 of the 11 subjects demonstrated fatigue-induced decreases in EMG AMP from the BB following the sustained, forearm flexion MVIC. This was true even though the decrease in MVIC torque of 20% at task failure in the present study was less than one-half of that of Carr et al. [12]. Furthermore, like the current study (Table 3) and that of Carr et al. [12], previous studies by Kranz et al. [36], Moritani et al. [43], and Orizio [46] reported mean decreases in EMG AMP from the BB of approximately 10% to 35% as the result of forearm flexion MVIC sustained for 40 to 60 s.

Fatigue during a sustained MVIC may be attributable to peripheral and/or central mechanisms. It has been suggested [12] that during sustained MVICs, blood flow occlusion can lead to the buildup of metabolic byproducts which interferes with excitation-contraction coupling within the muscle fiber (peripheral fatigue). In addition, the buildup of metabolic byproducts can cause inhibitory feedback from type III and IV muscle afferents that can lead to a decrease in central motor drive and synaptic nerve responsiveness via supraspinal and spinal mechanisms (central fatigue) [56]. There is often a mismatch between EMG AMP and central motor drive [23], and, therefore, it is questionable whether the fatigue-induced decreases in MVIC and EMG AMP from the BB in the present study reflected central fatigue. Furthermore, the presence of central fatigue is typically characterized by a decrease in motor unit firing rate due to a decrease in excitatory input and an increase in inhibitory input, as well as a decrease in motoneuron responsiveness [56]. In the present study, there was no mean fatigue-induced change in MMG MPF from the BB during the post-fatigue MVIC following the sustained forearm flexion MVIC. The MMG MPF provides a qualitative assessment of fatigue-induced changes in the global firing rate of the activated, unfused motor units [47]. Thus, the lack of change in MMG MPF from the BB following the sustained forearm flexion MVIC suggested that there was no change in the global motor unit firing rates. These findings did not support a contribution to the decrease in MVIC from central fatigue.

It is possible that the magnitude of fatigue in the present study for the agonist (BB) was not sufficient to be manifested in the neuromuscular responses. Task failure in the present study was defined as a 20% reduction in isometric torque during the sustained, maximal forearm flexion task which translated to a 9.5% decrease in MVIC. Typically, fatigue is characterized by a decrease in EMG MPF which is sensitive to the buildup of metabolic byproducts within the muscle fiber and reflects a decrease in muscle fiber action potential conduction velocity. In the present study, there was no fatigue-induced decrease in EMG MPF in the BB, even though the MVIC decreased by a mean of 9.5%. The EMG MPF is not only affected by changes in the muscle fiber milieu, but also muscle temperature [50]. Perhaps the lack of change in EMG MPF from pre- to post-fatigue for the BB reflected the small level of overall fatigue associated with a 9.5% decrease in MVIC and the competing influence of temperature which increases EMG MPF and the fatigue-induced buildup of metabolic byproducts which decrease EMG MPF [23,24,50].

Also, there was no fatigue-induced change in MMG AMP from the BB following the sustained forearm flexion MVIC. At submaximal levels of force up to approximately 60% of MVIC, changes in MMG AMP likely reflect motor unit recruitment [47]. At higher force levels, however, MMG AMP tends to plateau due to increases in muscle stiffness and intra-muscular fluid pressure [7]. The lack of change in MMG AMP in the present study likely reflected factors related to muscle compliance and not motor unit activation. Thus, in total, the neuromuscular responses from the BB suggested that the small fatigue-induced decrease in MVIC following the sustained forearm flexion task was likely due to peripheral fatigue from the buildup of metabolic byproducts that interfered with aspects of excitation contraction coupling, but was not sufficient to cause central fatigue or neuromuscular manifestations of fatigue for EMG MPF and MMG MPF.

The 6.0% decrease in forearm extension MVIC following the sustained forearm flexion fatiguing task was statistically parallel to the 9.5% decrease in forearm flexion MVIC, but was not associated with changes for any of the neuromuscular parameters from the agonist TB or antagonist BB muscles. Hence, the lack of fatigue-induced changes for EMG AMP from the TB (agonist) or BB (antagonist) indicated that coactivation did not contribute to the decrease in forearm extension MVIC.

Previous studies [17,29,53] have suggested that mechanical compression of blood vessels due to an increase in intramuscular pressure during sustained isometric muscle actions may reduce muscle blood flow and contribute to the buildup of metabolic byproducts resulting in peripheral fatigue [32]. In a sample of men, Keller et al. [32] reported fatigue-induced decreases in mean force, EMG AMP, EMG MPF, and MMG MPF, as well as an increase in MMG AMP during a 300 s, unilateral, sustained, isometric leg extension task anchored to a rating of perceived exertion (RPE) of 2 on a 10-point scale. Furthermore, there was a 30.9% decrease in MVIC and approximately a 50% increase in femoral artery blood flow from pre-fatigue to post-fatigue [32]. Typical mean neuromuscular and blood flow responses during a sustained, submaximal isometric muscle action when anchored to force were characterized by an increase in the amplitude and a decrease in the frequency content of the EMG signal, as well as a decrease in both the amplitude and frequency content of the MMG signal, and an increase in the average blood flow to the muscle [6,35,42,43].

The normalized EMG AMP at RPE = 2 from Keller et al. [32], corresponded to approximately 10% of MVIC and, therefore, was less than the 20% of MVIC for the EMG AMP from the antagonist (TB) during the sustained forearm flexion MVIC in the present study. During the first 10% (30 sec) of the sustained isometric leg extension task utilized by Keller et al. [32], there were slight decreases for MMG AMP and EMG MPF, but no changes for EMG AMP or MMG MPF. In the present study, following the fatiguing forearm flexion task in which the time task failure was 33.63 ± 8.18 sec, there were no mean changes for any of the neuromuscular parameters from the TB during the post-fatigue forearm extension MVIC. It is possible that differences in the neuromuscular responses of Keller et al. [32] and the present study were attributable to the differences in the intensities of the fatiguing tasks as well as the anchoring procedures.

Keller et al. [32] suggested that the decrease in EMG MPF was likely attributable to the accumulation of metabolic byproducts [K^+] and [H^+] which may also have contributed to the fatigue-induced decrease in MVIC. Peripheral fatigue may have also contributed to the decrease in forearm extension MVIC following the forearm flexion fatiguing task in the present study. This hypothesis is consistent with the findings of Kennedy et al. [33] who reported approximately a 9% decrease in forearm flexion MVIC following a 120 sec, sustained forearm extension MVIC, but no change in voluntary activation or evidence of central fatigue of the antagonist BB. Thus, it is not likely that central fatigue contributed to the decrease in forearm extension MVIC following the forearm flexion fatiguing task in the current study.

CONCLUSIONS

There were parallel decreases in MVIC from pre- to post-fatigue following the forearm flexion fatiguing task for forearm flexion MVIC (9.5%) and forearm extension MVIC (6.0%). Furthermore, there was a decrease in EMG AMP (28%) from the BB with no changes for any of the other neuromuscular parameters from the BB or the TB. Coactivation did not account for the fatigue-induced decreases in forearm flexion MIVC or forearm extension MVIC. The findings for the neuromuscular responses suggested that the fatigue-induced decreases in forearm flexion MVIC and forearm extension MVIC were due to peripheral, but not central fatigue.

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RWS contributed to the data collection, analyses, manuscript writing, and accepts responsibility for the integrity of the data analysis. TJH, TJN, RJS, and GOJ conceived and designed the study. RJS and GOJ provided administrative oversight of the study. TJN primarily collected the data while RWS contributed to interpretation of this data set. All authors contributed to the final drafting and approved the final submission of this manuscript. There was no external funding for this project.

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