Original Article

Task-specific performance fatigability and the bilateral deficit during isokinetic leg extensions

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Abstract

Objectives: The purpose of the present study was to compare the fatigue-induced changes in performance fatigability, bilateral deficit, and patterns of responses for the electromyographic (EMG) and mechanomyographic (MMG) amplitude (AMP) and mean power frequency (MPF), during unilateral and bilateral maximal, fatiguing leg extensions. **Methods**: Nine men (Mean$\pm$SD; age $=21.9\pm2.4$ yrs; height $=181.8\pm11.9$ cm; body mass $=85.8\pm6.2$ kg) volunteered to perform 50 consecutive maximal, bilateral (BL), unilateral dominant (DL), and unilateral non-dominant (NL) isokinetic leg extensions at $180°\cdot s^{-1}$, on 3 separate days. Electromyographic and MMG signals from both vastus lateralis (VL) muscles were recorded. Repeated measures ANOVAs were utilized to examine mean differences in normalized force, EMG AMP, EMG MPF, MMG AMP, MMG MPF and the bilateral deficit. **Results**: The results demonstrated a Condition $\times$ Repetition interaction for normalized force ($p=0.004$, $\eta^2_p=0.222$) and EMG MPF ($p=0.034$, $\eta^2_p=0.214$) and main effects for Repetition for EMG AMP ($p=0.019$, $\eta^2_p=0.231$), MMG AMP ($p<0.001$, $\eta^2_p=0.8550$), MMG MPF ($p=0.009$, $\eta^2_p=0.252$), and the bilateral deficit ($p<0.001$, $\eta^2_p=0.366$). **Conclusions**: The findings demonstrated less performance fatigability during the BL than the unilateral tasks, likely due to a reduced relative intensity via interhemispheric inhibition that attenuated the development of excitation-contraction coupling failure during the BL task.

Keywords: Bilateral Deficit, Electromyography, Isokinetic, Mechanomyography, Performance Fatigability

Introduction

The development of fatigue during exercise is a complex process involving perceptual, neuromuscular, and metabolic changes that manifest as reduced force production. Recent studies[1,2] have utilized performance fatigability to objectively assess the magnitude of exercise-induced fatigue. The present study defines performance fatigability as the exercise-induced reductions in force production. Performance fatigability is generally task-specific[2], however, the mechanisms underlying differences in performance fatigability between

tasks have yet to be elucidated[1,3,4]. Hypotheses regarding the mechanisms regulating performance fatigability include the amount of muscle mass activated[1], a peripheral fatigue threshold[3,4], and a sensory tolerance limit[3].

Previous studies[5-8] have utilized electromyographic (EMG) and mechanomyographic (MMG) signals to make inferences regarding the neuromuscular control strategies during unilateral, fatiguing, dynamic leg extensions. Specifically, the EMG signal provides insight regarding muscle excitation (EMG AMP) and muscle fiber action potential conduction velocity (EMG MPF)[9,10], while the MMG signal provides insight regarding motor unit recruitment (MMG AMP) and global, unfused motor unit firing rates (MMG MPF)[11,12]. The task-specificity of performance fatigability[2] suggests unilateral and bilateral (BL) muscle actions may exhibit unique fatigue-induced changes to performance and neuromuscular control, however, studies comparing unilateral and BL fatiguing muscle actions are limited[13-16]. In general, these studies have reported a greater performance fatigability during unilateral compared to BL submaximal[13] and maximal[16] isometric leg

extensions, as well as submaximal[14], and maximal[15] dynamic leg extensions. The neural mechanism(s) underlying the differences in performance fatigability, however, remain to be explained.

The bilateral deficit is a unique phenomenon characterized by reduced force production of a muscle during BL contractions compared to the sum of the muscle actions performed unilaterally[17]. It has been hypothesized that the bilateral deficit is the result of interhemispheric inhibition between the primary motor cortices (M1) during the BL task[18,19]. Specifically, Ferbert et al.[20] demonstrated that both evoked and voluntary activation of the M1 elicited inhibitory signaling in the contralateral M1 during first dorsal interosseous (FDI) muscle actions, resulting in attenuated muscle activation. Oda and Moritani[21,22] also reported reduced movement-related cortical potentials (MRCPs) during BL compared to unilateral FDI muscle actions. Thus, the presence of the bilateral deficit may be indicative of unique neural control strategies during BL compared to unilateral muscle actions, however, it remains unclear how the bilateral deficit is affected by fatigue[23,24]. Therefore, the purpose of the present study was to compare the fatigue-induced changes in performance fatigability, patterns of neuromuscular responses, and the bilateral deficit during unilateral and BL maximal, fatiguing leg extensions. Based on previous studies[13-16,23,24], we hypothesized that: 1) the unilateral conditions would exhibit a greater performance fatigability than the bilateral condition; 2) unilateral and BL muscle actions would be characterized by unique patterns of neuromuscular responses and; 3) the bilateral deficit would be attenuated throughout the fatiguing task.

Materials and Methods

Subjects

Nine men (Mean±SD; age=21.9±2.4 yrs; height=181.8±11.9 cm; body mass=85.8±6.2 kg) volunteered to participate in this study. All subjects were recreationally trained and reported performing 6.2±2.6 hours of resistance training per week and 1.9±1.7 hours of aerobic training per week. All subjects were right leg dominant as determined by kicking preference. The subjects were free from any injuries that would influence their performance. The present study was approved by the University Institutional Review Board for Human Subjects (IRB Approval #: 20191019755FB) and, prior to participation, the subjects signed a written Informed Consent and completed a health history questionnaire.

Protocol

The subjects visited the laboratory on four occasions, each separated by 3-7 days. The first visit was an orientation session to familiarize the subjects with the exercise protocol and they performed submaximal and maximal, BL and unilateral concentric, isokinetic leg extensions at 180°·s⁻¹. All muscle actions were performed on a calibrated Cybex 6000 dynamometer (Cybex, Division of Lumex Inc., Ronkonkoma,

NY, USA). At the subsequent test visits, the subjects completed 50 consecutive maximal BL, unilateral dominant leg only (DL), or unilateral non-dominant leg only (NL) (in random order on separate days) isokinetic leg extensions at 180°·s⁻¹.

Electromyography, Mechanomyography, and Force Acquisition

Electromyography was assessed in accordance with SENIAM recommendations[25] utilizing bipolar (30-mm center-to-center) surface EMG electrode arrangements (circular 4-mm diameter silver/silver chloride; Biopac Systems, Inc., Santa Barbara, CA, USA) on the vastus lateralis (VL) of both limbs. The electrodes were placed 66% of the distance between the anterior superior iliac spine and the lateral portion of the patella and were orientated at an angle of 20° to align with the angle of pennation of the VL muscle fibers[26]. A reference electrode was placed over the anterior superior iliac spine. Prior to electrode placement, the locations were shaved, abraded, and cleaned with an alcohol wipe. Between both bipolar electrode arrangements, miniature accelerometers (ENTRAS EGAS FT 10, Bandwidth 0-200 Hz, dimension 1.0×1.0×0.5, mass 1.0 g) were placed using double-sided adhesive tape to assess the MMG signal of both VL.

The raw EMG and MMG signals were digitized at 2,000 Hz with a 12-bit analog-to-digital converter (Model MP 150; Biopac Systems, Inc., Goleta, CA, USA) and the data were stored on a personal computer (G5 Dell Inc., Round Rock, TX, USA) for later analyses. The EMG signal was amplified (gain: × 1,000) with differential amplifiers (EMG100C; Biopac Systems, Inc., Goleta, CA, USA). The signals were bandpass filtered (fourth-order Butterworth) at 10-500 Hz for EMG and 5-100 Hz for MMG. The EMG and MMG signals were processed using customized programs in LabVIEW programming software (Version 18.0f2; National Instruments, Austin, TX, USA). For each repetition of the fatiguing task, the EMG (μV root mean square, μVrms) and MMG (m·s²) amplitude (AMP) and mean power frequency (Hz, MPF) values were calculated for a epoch corresponding to the middle 30° range of motion that avoids the acceleration and deceleration phases of the leg extension muscle action[27]. The isokinetic torque and neuromuscular values from the 50 maximal isokinetic leg extensions were averaged across 5 consecutive repetitions (i.e. average of repetitions 1-5=5, 6-10=10, 11-15=15, 16-20=20, 21-25=25, 26-30=30, 31-35=35, 36-40=40, 41-45=45, 46-50=50) to generate a total of 10 data points. To examine the patterns and timing of changes throughout the fatiguing task, the isokinetic torque and neuromuscular parameters were normalized to the respective value at repetition 5.

Performance fatigability was calculated from the absolute peak torque values from repetitions 1-5 (initial) and repetitions 46-50 (final) as:

$$Performance\ Fatigability\ (\%) = \frac{(Initial\ Peak\ Torque - Final\ Peak\ Torque)}{(Initial\ Peak\ Torque)} \times 100$$
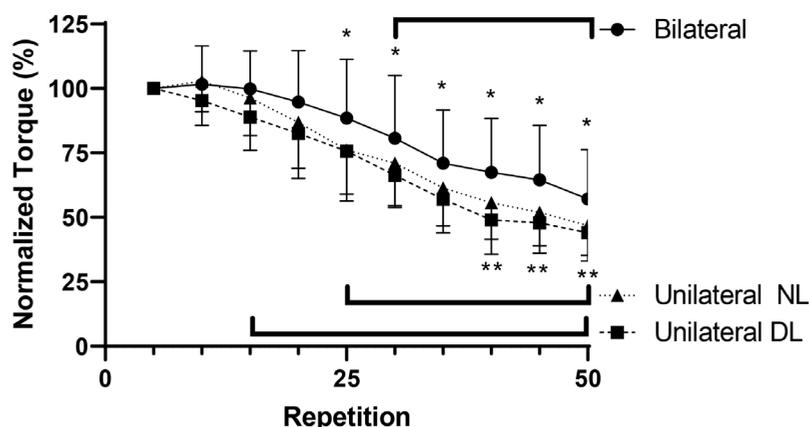
Figure 1. Mean (± SD) isokinetic toque during maximal, bilateral, unilateral dominant leg (DL), and unilateral non-dominant leg (NL), isokinetic leg extensions. All torque values were normalized as a percentage of the mean of repetitions 1-5. (Note: $p<0.05$. Bilateral: ⊓ Repetition 5> repetitions 30-50. *Bilateral >Unilateral DL at repetitions 25-50. **Bilateral >Unilateral NL at repetitions 40-50. Unilateral DL: ⊔ Repetition 5> repetitions 15-50. Unilateral Right Leg=Unilateral Left Leg for repetitions 5-50. Unilateral NL: ⊔ Repetition 5> repetitions 25-50).

To examine changes in the bilateral deficit throughout the fatiguing task, the Bilateral Deficit Index (BDI) from the absolute isokinetic peak torque was calculated as described by Howard and Enoka[17]:

$$BDI\ (\%) = \left[100 \times \frac{Bilateral}{(Right\ Leg + Left\ Leg)} \right] - 100$$

A negative BDI value indicates a bilateral deficit and a positive BDI value indicates a bilateral facilitation.

Statistical Analyses

For normalized isokinetic torque, mean differences were examined with a 3 (Condition [BL, DL, NL]) × 10 (Repetition [5, 10, 15, 20, 25, 30, 35, 40, 45, 50]) repeated measures ANOVA. Mean differences for each normalized neuromuscular parameter (EMG AMP, EMG MPF, MMG AMP, and MMG MPF) were examined by separate 2 (Leg [Dominant, Non-dominant]) × 3 (Condition [BL, DL, NL]) × 10 (Repetition [5, 10, 15, 20, 25, 30, 35, 40, 45, 50]) repeated measures ANOVA. Mean differences for the BDI throughout the fatiguing task were examined with a 1 × 10 (Repetition [5, 10, 15, 20, 25, 30, 35, 40, 45, 50]) repeated measures ANOVA across repetitions. Follow up 1-way repeated measure ANOVAs and paired samples t-tests were performed when appropriate. The time course of changes for the mean normalized isokinetic torque and normalized neuromuscular parameters where examined with paired samples *t*-tests between repetition 5 and repetitions 10-50. The Greenhaus-Geisser correction was applied when sphericity was violated as determined by Maulchy's Tests of Sphericity. Effect sizes (partial eta squared (η^2_p) and Cohen's *d*) were calculated for all ANOVAs and paired samples t-tests, respectively. All statistics were examined with IBM SPSS v. 25 (Armonk, NY, USA) and an alpha of $p<0.05$ was considered statistically significant for all comparisons.

Results

Isokinetic Peak Torque

The initial absolute peak torque values (average of repetitions 1-5) for the fatiguing tasks were BL=298.4±61.5 N·m, DL=185.0±40.6 N·m, and NL=177.6±38.3 N·m. The final absolute peak torque values (average of repetitions 46-50) for the fatiguing tasks were BL=164.8±50.7 N·m, DL=80.3±23.4 N·m, and NL=81.9±25.3 N·m. Thus, the performance fatigability for the BL, DL, and NL conditions were 42.8±19.1%, 56.0±10.9%, and 53.2±11.6%, respectively. The results of the 3×10 repeated measures ANOVA demonstrated a significant Condition by Repetition interaction ($p=0.004$, $\eta^2_p=0.222$; Figure 1). The follow-up 1-way repeated measures ANOVA for the BL condition demonstrated a significant effect for Repetition ($p<0.001$, $\eta^2_p=0.739$). Post-hoc pairwise comparisons demonstrated that repetition 5 (100.0±0.0%) was significantly greater than repetitions 30 (80.7±24.3%, $p=0.045$, $d=1.12$), 35 (71.0±20.6%, p=0.003, d=1.99), 40 (67.5±20.9, $p=0.002$, $d=2.20$), 45 (64.5±21.1%, $p=0.001$, $d=2.38$) and 50 (57.2±19.1%, $p<0001$, $d=3.17$).

The follow up 1-way repeated measures ANOVA for the DL condition demonstrated a significant effect for Repetition ($p<0.001$, $\eta^2_p=0.907$; Figure 1). Post-hoc pairwise comparisons demonstrated that repetition 5 (100.0±0.0%) was significantly greater than repetitions 15 (88.9±12.9%, $p=0.032$, $d=1.22$), 20 (82.6±17.4%, $p=0.017$, $d=1.41$), 25 (75.6±16.6%, $p=0.002$, $d=2.08$), 30 (66.3±12.4%, $p<0.001$, $d=3.84$) , 35 (57.1±13.0%, $p<0.001$, $d=4.67$), 40 (49.0±13.3%, $p<0.001$, $d=5.42$), 45 (47.8±11.8%, $p<0.001$, $d=6.26$), and 50 (44.1±10.9%, $p<0.001$, $d=7.25$).

The follow-up 1-way repeated measures ANOVA for the NL condition demonstrated a significant effect for
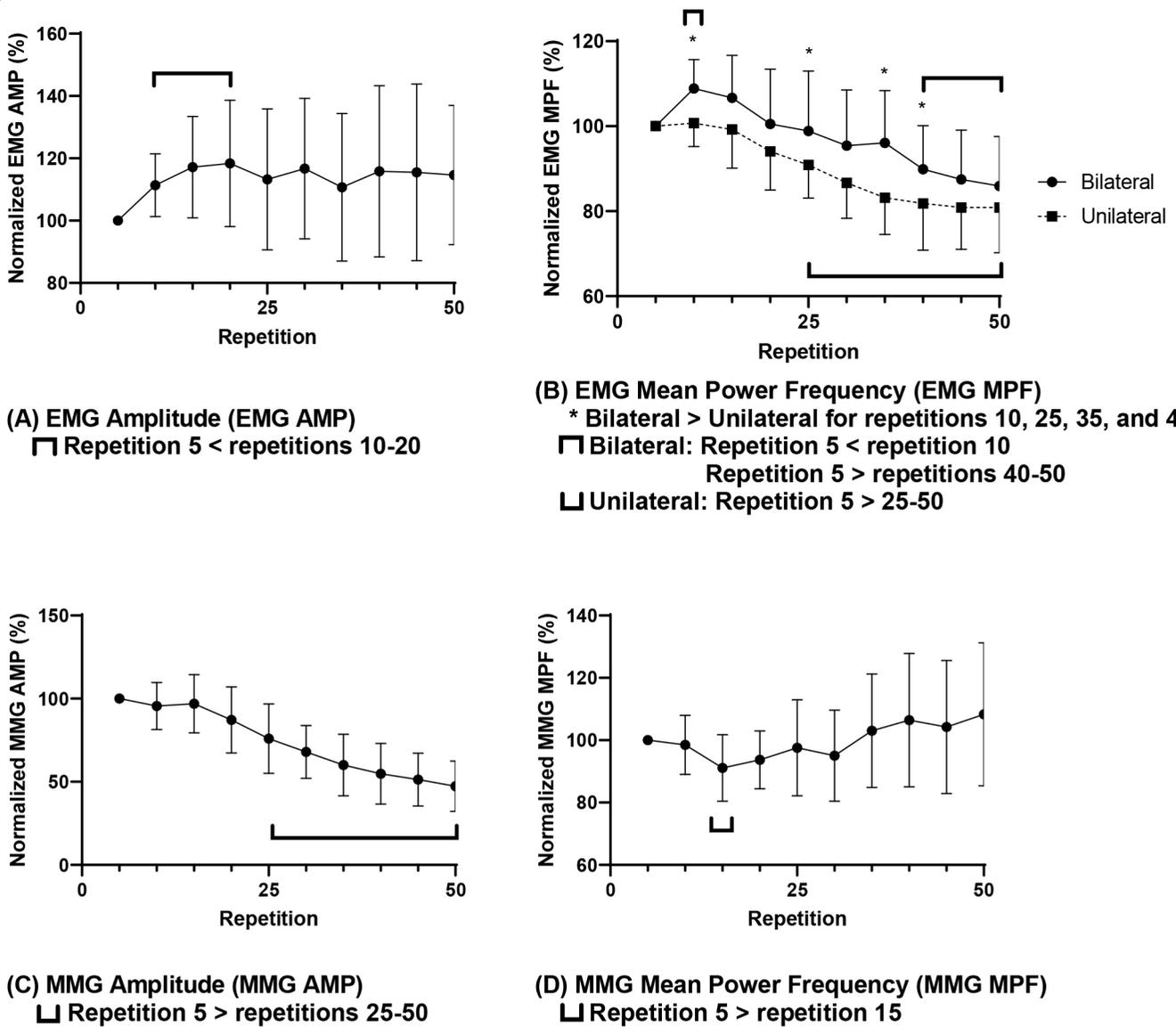
Figure 2. Mean (± SD) EMG amplitude (A), EMG mean power frequency (B), MMG amplitude (C) and MMG mean power frequency (D) during maximal, isokinetic leg extensions. All neuromuscular parameters were normalized as a percentage of the mean of repetitions 1-5. For A, C, and D, the neuromuscular parameters have been collapsed across Leg and Condition due to the significant (p<0.05) main effect for Repetition only. For B, both the bilateral and unilateral conditions (collapsed across Leg) have been included due to the significant (p<0.05) Condition by Repetition interaction. See Results section for the outcomes of the ANOVA and follow-up analyses for each variable. (Note: p<0.05).

Repetition (p<0.001, η^2_p=0.920; Figure 1). Post-hoc pairwise comparisons demonstrated that repetition 5 (100±0.0%) was significantly greater than repetitions 25 (76.1±19.6%, p=0.006, d=1.73), 30 (71.0±16.5%, p=0.001, d=2.49), 35 (61.5±14.7%, p<0.001, d=3.36), 40 (55.7±14.2%, p<0.001, d=4.41), 45 (51.9±12.9%, p<0.001, d=5.27), and 50 (46.8±11.6%, p<0.001, d=6.49).

Follow-up 1-way repeated measures ANOVAs demonstrated significant differences between BL, DL, and NL at repetitions 25 (p=0.026, η^2_p=0.365), 30 (p=0.040, η^2_p=0.332), 35 (p=0.026, η^2_p=0.368), 40 (p=0.004, η^2_p=0.498), 45 (p=0.002, η^2_p=0.538), and 50 (p=0.008, η^2_p=0.538; Figure 1). Post-hoc pairwise comparisons demonstrated that at repetition 25, BL (88.5±22.7%) was significantly greater than DL (75.6±16.6%, p=0.008, d=0.65), at repetition 30, BL (80.7±24.3%) was significantly greater than DL (66.3±12.4%, p=0.033, d=0.75), at repetition 35, BL (71.0±20.6%) was significantly greater than DL (57.1±13.0%, p=0.028, d=0.81), at repetition 40, BL (67.5±20.9%) was significantly greater than DL
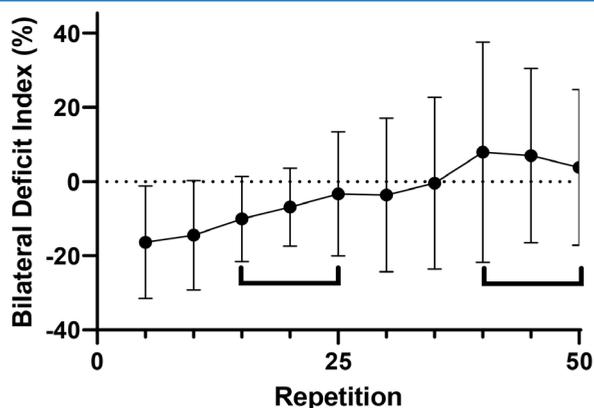
Figure 3. The Bilateral Deficit Index (Mean ± SD) during maximal, isokinetic leg extensions. (Note: $p<0.05$. ⊔ Repetition 5< repetitions 15-25, 40-50).

(49.0±13.3%, $p=0.011$, $d=1.06$) and NL (55.7±14.2%, $p=0.023$, $d=0.66$), at repetition 45, BL (64.5±21.1%) was significantly greater than DL (47.8±11.8%, $p=0.003$, $d=0.98$) and NL (51.9±12.9%, $p=0.029$, $d=0.72$), and at repetition 50, BL (57.1±19.1%) was significantly greater than DL (44.0±10.9, $p=0.009$, $d=0.84$) and NL (46.8±11.6%, $p=0.043$, $d=0.65$).

Neuromuscular Parameters

The patterns of neuromuscular responses are depicted in Figure 2. The results of the $2 \times 3 \times 10$ repeated measures ANOVA for EMG AMP demonstrated no significant 3- ($p=0.186$) or 2- ($p=0.199-0.535$) way interactions, but a significant main effect for Repetition ($p=0.019$, $\eta^2_p=0.231$; Figure 2A). Post-hoc pairwise comparisons demonstrated that repetition 5 (100±0.0%) was significantly less than repetitions 10 (111.4±10.1%, $p=0.010$, $d=1.60$), 15 (117.2±16.2%, $p=0.013$, $d=1.50$), and 20 (118.4±20.3% $p=0.026$, $d=1.28$).

The results of the $2 \times 3 \times 10$ repeated measures ANOVA for EMG MPF demonstrated no significant 3-way interaction ($p=0.374$) but a significant Condition by Repetition interaction, collapsed across Leg ($p=0.034$, $\eta^2_p=0.214$; Figure 2B). The follow-up 1-way repeated measures ANOVA for the BL condition demonstrated a significant effect for Repetition ($p<0.001$, $\eta^2_p=0.619$). Post-hoc pairwise comparisons demonstrated that repetition 5 (100.0±0.0%) was significantly less than repetition 10 (108.9±6.8%, $p=0.004$, $d=1.85$) and was significantly greater than repetitions 40 (89.9±10.3%, $p=0.018$, $d=1.39$), 45 (87.5±11.6%, $p=0.012$, $d=1.52$), and 50 (86.0±11.6%, $p=0.007$, $d=1.71$). The follow-up 1-way repeated measures ANOVA for the unilateral condition demonstrated a significant effect for Repetition ($p=<0.001$, $\eta^2_p=0.619$). Post-hoc pairwise comparisons demonstrated that repetition 5 (100.0±0.0%) was significantly greater than repetitions

25 (90.9±7.8%, $p=0.008$, $d=1.65$), 30 (86.7±8.4%, $p=0.001$, $d=2.24$), 35 (83.2±8.7%, $p<0.001$, $d=2.73$), 40 (81.8±11.0%, $p=0.001$, $d=2.34$), 45 (80.9±9.9%, $p<0.001$, $d=2.73$), and 50 (80.9±10.6%, $p=0.001$, $d=2.55$).

Follow-up pairwise comparisons demonstrated that for EMG MPF, the BL condition was significantly greater than the UL conditions at repetitions 10 (BL=108.9±6.8%, UL=100.7±5.5%, $p=0.037$, $d=1.33$), 25 (BL=98.9±14.1%, UL=90.9±7.8, $p=0.042$, $d=0.70$), 35 (96.1±12.3%, UL=83.2±8.7%, $p=0.003$, $d=1.21$), and 40 (89.9±10.3%, UL=81.8±11.0%, $p=0.012$, $d=0.76$).

The results of the $2 \times 3 \times 10$ repeated measures ANOVA for MMG AMP demonstrated no significant 3- ($p=0.250$) or 2- ($p=0.438-0.780$) way interactions, but a significant main effect for Repetition ($p<0.001$, $\eta^2_p=0.8550$; Figure 2C). Post-hoc pairwise comparisons demonstrated that repetition 5 (100±0.0%) was significantly greater than repetitions 25 (76.0±20.6%, $p=0.009$, $d=1.65$), 30 (68.0±15.6%, $p<0.001$, $d=2.90$), 35 (60.1±18.5, $p<0.001$, $d=3.05$), 40 (54.8±18.3%, $p<0.001$, $d=3.49$), 45 (51.3±16.0%, $p<0.001$, $d=4.31$), and 50 (47.4±15.2%, $p<0.001$, $d=4.89$).

The results of the $2 \times 3 \times 10$ repeated measures ANOVA for MMG MPF demonstrated no significant 3- ($p=0.191$) or 2- ($p=0.130-0.992$) way interactions, but a significant main effect for Repetition ($p=0.009$, $\eta^2_p=0.252$; Figure 2D). Post-hoc pairwise comparisons demonstrated that repetition 5 (100±0.0%) was significantly greater than repetition 15 (91.1±10.7%, $p=0.037$, $d=1.76$).

Bilateral Deficit Index

The results of the 1×10 repeated measures ANOVA for the BDI demonstrated a significant effect for Repetition ($p<0.001$, $\eta^2_p=0.366$; Figure 3). Post-hoc pairwise comparisons demonstrated repetition 5 (-16.4±15.2%) was significantly less than repetitions 15 (-10.1±11.5%, $p=0.049$, $d=0.47$), 20 (-6.9±10.5%, $p=0.023$, $d=0.72$), 25

(-3.3±16.7%, p=0.023), 40 (7.9±29.7%, p=0.018, d=1.03), 45 (7.0±23.5%, p=0.004, d=1.18), and 50 (3.8±21.0%, p=0.011, d=1.10).

Discussion

The purpose of this study was to examine the fatigue-induced changes in isokinetic torque and neuromuscular parameters during unilateral and BL leg extensions. The results demonstrated that the unilateral leg extensions exhibited 56.0±10.9% and 53.2±11.6% performance fatigability for the DL and NL conditions, respectively, while the BL condition exhibited a 42.8±19.1% performance fatigability. These findings were consistent with previous studies that have demonstrated greater performance fatigability for unilateral than BL isometric[13,16] and dynamic[14,15,28] leg extensions. These results, as well as those of previous studies[13-16,28], were consistent with the hypothesis of Thomas et al.[1] that the magnitude of performance fatigability "... depends on the muscle mass engaged during the task..." (pg. 241). Specifically, Thomas et al.[1] suggested that engaging more muscle mass (i.e. BL versus unilateral muscle actions) poses a greater risk to homeostasis for systems such as the cardiovascular and respiratory systems and, thus, performance will be attenuated to mitigate severe perturbations to homeostasis. Unilateral tasks, however, pose less risk to homeostatic conditions which, "... permits the exerciser to endure greater perturbations to contractile function..." (pg. 242) and, therefore, greater performance fatigability for the unilateral than the BL muscle actions.

It has been proposed that the total fatigue-induced threat to homeostasis and task failure is determined by a global negative feedback loop comprised of the sum of feedforward (i.e. corollary discharge) and feedback (i.e. from group III/IV afferents) signaling called the sensory tolerance limit[3,29]. Theoretically, feedforward signals provide the central motor drive to the activated muscle mass as well as an efferent copy or "corollary discharge" to the sensory cortex to influence the perception of exertion and the development of central fatigue[3]. The feedback aspect of the sensory tolerance limit reflects group III/IV afferent feedback from the activated muscle mass which sense metabolic perturbations of the intramuscular milieu associated with muscle contractions[3,4]. Hureau et al.[3] hypothesized that compared to unilateral muscle actions, BL muscle actions reach the sensory tolerance limit with similar overall sensory feedback, less metabolic perturbation, greater corollary discharge, and greater central motor drive. In theory, it is possible that these factors translated into less performance fatigability for the BL than the unilateral conditions in the present study.

It is also possible that the BL and unilateral performance fatigability findings of the present study can be explained by the "critical threshold of peripheral fatigue" hypothesis[3]. This hypothesis proposes that exercise tolerance is modulated by the build-up of metabolic byproducts in the activated muscle mass which induces group III/IV afferent feedback to inhibit central motor drive and, thereby, limits performance fatigability to a critical level[3,14]. A central tenant of the critical threshold of peripheral fatigue hypothesis is that the threshold for exercise tolerance is specific to individuals and tasks. In the present study, the unilateral leg extensions for the DL and NL conditions involved the same tasks and the magnitude and timing of performance fatigability were nearly identical (Figure 1). On the other hand, it could be argued that even though they both involved leg extension muscle actions, the BL condition represented a substantially different task which could explain the differences in performance fatigability between the unilateral and BL conditions. For example, Oda[18] suggested that interhemispheric inhibition functions to coordinate the central motor drive to the activated muscles during BL tasks. Oda and Moritani[21,22] examined the potential neural mechanisms associated with the bilateral deficit by comparing MRCP during BL and unilateral handgrip maximal voluntary isometric contractions (MVIC). The MRCP precede voluntary muscle actions and arise from neural circuits associated with motor preparation and initiation[21]. By comparing the same hemisphere in the brain between conditions, Oda and Moritani[21,22] demonstrated that MRCP during the BL task were smaller than those during the unilateral task. It was hypothesized[21,22] that interhemispheric inhibition during BL muscle actions reduced the neural drive to the muscle, resulting in a bilateral deficit. In the present study, the subjects demonstrated a 16.4±15.2% bilateral deficit at repetition 5 which suggested the BL condition was likely influenced by interhemispheric inhibition at the onset of the task that may have lead to a unique critical threshold and, therefore, resulted in a different level of performance fatigability than the unilateral conditions.

The present study examined the time course of fatigue-induced changes in force and neuromuscular parameters throughout the unilateral and BL conditions. Matkowski et al.[13] and Rossman et al.[13,14] examined the differences in force and peripheral fatigue between unilateral and BL leg extensions by comparing the changes from pre- to post-exercise for both conditions, but not during the fatiguing task. Koral et al.[16] reported no differences in force declines between unilateral and BL isometric leg extensions during the one minute MVIC fatiguing task, but a greater reduction in post-exercise MVIC force for the unilateral compared to the BL condition. The present findings demonstrated that the unilateral conditions exhibited an earlier decline in force (repetitions 15 and 25 for the DL and NL, respectively) than the BL condition (repetition 30; Figure 1). The EMG MPF also exhibited earlier declines during the unilateral conditions (repetition 15) than the BL condition (repetition 40; Figure 2B). Fatigue-induced decreases in EMG MPF reflect decreases in motor unit action potential conduction velocity[30,31] and have been attributed to the build-up of metabolic byproducts in peripheral tissue, such as extracellular potassium, intracellular calcium, and intracellular hydrogen ions[30,32,33]. Thus, the task-specific declines in force and EMG MPF in the present study indicated that the unilateral and BL conditions were characterized by distinct patterns of fatigue. It is

possible that interhemispheric inhibition exhibited during the BL condition reduced the relative intensity of the muscle actions, which mitigated the declines in force and EMG MPF and contributed to the reduced performance fatigability compared to the unilateral conditions.

The current findings demonstrated that there were initial increases in EMG AMP from repetitions 10-20 that returned to baseline for the remainder of the fatiguing tasks for the unilateral and BL conditions (Figure 2A). These findings were consistent with previous studies that demonstrated similar responses in EMG AMP for unilateral and BL leg extensions during submaximal[13] and maximal[16] fatiguing tasks. The findings of the present study were generally consistent with previous studies that have demonstrated an initial increase followed by a plateau in EMG AMP[5,6], indicating muscle excitation remains relatively stable[9] during maximal, unilateral, leg extensions. Furthermore, we have previously reported no differences in the fatigue-related patterns of EMG AMP responses between unilateral and BL maximal leg extensions at $60°·s^{-1}$ in men that were characterized by an initial increase followed by a plateau throughout the fatiguing tasks[15]. Thus, the current findings, as well as our previous findings[15], suggested that performance fatigability as the result of maximal, unilateral and BL leg extensions were associated with excitation-contraction coupling failure because torque production decreased at a stable level of muscle excitation. Excitation-contraction coupling failure results from fatigue-induced alterations in the metabolic milieu within the muscle, such as derangements in Ca^{2+} release and reuptake from the sarcoplasmic reticulum and impaired cross-bridge binding dynamics, that occur independent of changes in muscle excitation[34,35] and neural drive to the muscle.

The present study demonstrated no significant differences between the unilateral and BL muscle actions for either MMG AMP or MMG MPF (Figure 2C, D). Under some conditions, the amplitude of the MMG signal reflects motor unit recruitment[11,12,36], but can be affected by reductions in muscle compliance due to increases in muscle stiffness[37,38] and intramuscular pressure[39,40]. In the present study, the lack of changes in muscle excitation (EMG AMP) suggested that the declines in MMG AMP were likely due to a greater restriction on the lateral oscillations of the muscle fibers as a result of reduced muscle compliance during the maximal unilateral and BL leg extensions. These findings were consistent with our previous findings[15] that demonstrated fatigue-induced decreases in MMG AMP following unilateral and BL leg extensions at $60°·s^{-1}$.

The frequency content of the MMG signal reflects the global firing rate of the unfused, activated motor units[12,36]. The present study demonstrated no significant differences in the MMG MPF between the unilateral and BL condition and, in general, no changes throughout the fatiguing task (Figure 2D). These findings were not consistent with those of Anders et al.[15] who reported significant decreases in MMG MPF throughout the fatiguing isokinetic leg extensions at $60°·s^{-1}$ for the unilateral and BL conditions. The present findings may,

therefore, represent velocity-dependent differences in global motor unit firing rate modulation during maximal, dynamic leg extensions. It has been reported, however, that MMG MPF may also be affected by changes in muscle compliance[12,41] and additional research is needed to determine whether the lack of change in global motor unit firing rate (MMG MPF) is a neural control strategy for higher velocity muscle actions or the result of reduced muscle compliance throughout the fatiguing task.

In addition to examining the time course of changes in the neuromuscular parameters, the present study examined the changes in the bilateral deficit throughout the fatiguing task (Figure 3). The bilateral deficit has been extensively reported during maximal muscle actions[19], however, few studies[23,24] have examined fatigue-induced changes to the bilateral deficit. These studies[23,24] have reported equivocal findings, demonstrating both decreases[23] and increases[24] in the disparity of force production of a muscle during unilateral and BL muscle actions. Similarly, limited research has examined the influence of fatigue on interhemispheric inhibition[42-44]. Matsuura and Ogata[43] examined the effects of fatiguing, unilateral plantar flexions on interhemispheric inhibition for FDI muscles actions and demonstrated that interhemispheric inhibition from the affected (associated with the fatigued muscle) M1 to the unaffected M1 was reduced following the fatiguing task. Takahashi et al.[44] reported reduced interhemispheric inhibition in both upper (FDI, biceps brachii) and lower limbs (quadriceps femoris) following repeated BL leg extension muscle actions at 50% of maximal voluntary contraction. Additionally, Alhassani et al.[42] recently reported attenuated interhemispheric inhibition to the unaffected FDI following hypertonic saline-induced pain in the contralateral FDI. The findings of the present study demonstrated a bilateral deficit (16.4±15.2%) that was attenuated by the end of the fatiguing task (Figure 3). These findings, in conjunction with previous studies[42-44] suggested that reductions in the bilateral deficit throughout the fatiguing task may be associated with decreased interhemispheric inhibition during BL leg extensions. Thus, BL muscle actions may be characterized by unique changes in central neural processes that manifest as attenuated rates of fatigue and performance fatigability compared to unilateral muscle actions. Further research is warranted to examine the fatigue-induced changes in the bilateral deficit and interhemispheric inhibition.

Conclusion

In conclusion, the present study reported greater performance fatigability for the unilateral than BL leg extensions. The BL condition was associated with an initial bilateral deficit, a phenomenon associated with interhemispheric inhibition which has been demonstrated to reduce the maximal force production of a muscle during BL muscle actions. The reduced relative intensity throughout the BL task, therefore, may have delayed the build-up of metabolic byproducts and attenuated the development of

excitation-contraction coupling failure compared to the unilateral tasks. These findings suggested differences in performance fatigability between unilateral and BL muscle actions are likely due to task-specific differences in the neural control strategies between unilateral and BL modalities. Future studies are warranted to further elucidate the neural mechanisms underlying unilateral and BL modalities and how these mechanisms are influenced by fatigue.

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