

Original Article

Acute Effects of Sprint Interval Training and Blood Flow Restriction on Neuromuscular and Muscle Function

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Abstract

Objectives: This investigation examined the effects of continuous (CBFR) and intermittent (IBFR) blood flow restriction (BFR) applied during sprint interval training (SIT) on performance and neuromuscular function. **Methods:** Fifteen men completed a randomized bout of SIT with CBFR, IBFR, and without BFR (No-BFR), consisting of 2, 30-s maximal sprints on a cycle ergometer with a resistance of 7.5% of body mass. Concentric peak torque (CPT), maximal voluntary isometric contraction (MVIC) torque, and muscle thickness (MT) were measured before and after SIT, including surface electromyography (sEMG) recorded during the strength assessments. Peak and mean revolutions per minute (RPM) were measured during SIT and power output was examined relative to physical working capacity at the fatigue threshold (PWC_{FT}). **Results:** CPT and MVIC torque decreased from pre-SIT (220.3 ± 47.6 Nm and 355.1 ± 72.5 Nm, respectively) to post-SIT (147.9 ± 27.7 Nm and 252.2 ± 45.5 Nm, respectively, all $P < 0.05$), while MT increased (1.77 ± 0.31 cm to 1.96 ± 0.30 cm). sEMG mean power frequency decreased during CPT ($-12.8 \pm 10.5\%$) and MVIC ($-8.7 \pm 10.2\%$) muscle actions. $\%PWC_{FT}$ was greater during No-BFR ($414.2 \pm 121.9\%$) than CBFR ($375.9 \pm 121.9\%$). **Conclusion:** SIT with or without BFR induced comparable alterations in neuromuscular fatigue and sprint performance across all conditions, without affecting neuromuscular function.

Keywords: BFR, PWC_{FT} , sEMG, SIT, Threshold

Introduction

The application of blood flow restriction (BFR) during resistance exercise has been well studied, but less is known regarding the effects of BFR during maximal intensity activities. Specifically, BFR may provide a robust training stimulus when combined with sprint interval training (SIT). SIT is a form of high intensity interval training that utilizes maximal or supramaximal sprints interspersed

with rest periods^{1,2}. SIT has been demonstrated to improve maximal aerobic speed, mean power, peak power, and time to exhaustion³. Furthermore, there was a 4.7% increase in maximal oxygen uptake (VO_{2max}) following a 4-week intervention of SIT with BFR applied intermittently (IBFR) during passive rest, compared to a 1.1% increase with no BFR (No-BFR)⁴. Additionally, acute SIT with BFR applied continuously (CBFR) results in lower mean power (530 ± 160 W vs. 543 ± 135 W), total work (42 ± 32 kJ vs. 162 ± 81 kJ), maximal heart rate (171 ± 20 bpm vs. 185 ± 9 bpm), and fatigue index ($-23.1 \pm 7.7\%$ vs. $-26.5 \pm 7.2\%$) than SIT without BFR⁵. A greater rating of perceived exertion (RPE) for the legs (19.5 ± 0.6) but a lower RPE for breathing (15.5 ± 2.5) was observed during SIT with CBFR compared to No-BFR⁵. Together, these studies^{4,5} indicated that SIT with BFR, whether applied intermittently or continuously, elicits both chronic positive (increase in VO_{2max}) and adverse acute (decrease in mean power and total work) physiological and performance responses.

The authors have no conflict of interest.

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Other studies have also examined the acute responses of SIT with CBFR and/or IBFR⁶⁻⁸. For example, relative to non-restricted conditions, SIT combined with CBFR results in fewer completed 10-s cycling sprints, lower muscle excitation, and resting twitch force⁷. Furthermore, SIT combined with IBFR (during rest) reduced peak power, oxyhemoglobin concentration, and tissue saturation, but increased heart rate to a greater extent across five 10-s cycling sprints compared to SIT with No-BFR⁶. There is less available information, however, that has examined the effects of IBFR versus CBFR during SIT. Specifically, IBFR may elicit comparable physiological responses as CBFR but achieved at a lower perceived effort⁹. Therefore, it is important to examine the differences in physiological responses to SIT between IBFR and CBFR to develop more efficient training modalities to elicit desired adaptations.

The examination of SIT with and without BFR has been decomposed using a multitude of outcomes^{4-6,10}. The application of BFR, however, induces unique sensations and physiological perturbations in the occluded limb that are typically quantified by systemic or subjective measures of fatigue or performance. Thus, local measures of fatiguing exercise such as acute muscle swelling and neuromuscular function would provide valuable insight to the physiological responses associated with SIT and SIT combined with IBFR or CBFR. For example, acute changes in muscle thickness are associated with muscle swelling which may mediate chronic changes in muscle hypertrophy^{11,12}. Acute changes in neuromuscular function as evaluated with surface electromyography (sEMG), including muscle excitation (sEMG amplitude), action potential conduction velocity (sEMG frequency) and maximal strength, can be used to evaluate the efficacy of exercise¹³ which may mediate chronic adaptations in neuromuscular function¹⁴.

Local muscle fatigue can be evaluated by determining the physical working capacity at the fatigue threshold (PWC_{FT}). Specifically, the sEMG-based PWC_{FT} can be defined as an individual's theoretical neuromuscular fatigue threshold where exercise can be sustained without accruing neuromuscular fatigue. The sEMG-based PWC_{FT} can also be applied to distinguish between heavy and moderate intensity exercise^{15,16}. For example, exercise performed at or below the sEMG-based PWC_{FT} does not induce a linear increase in muscle excitation as demarcated by sEMG amplitude. Exercise performed above sEMG-based PWC_{FT} is associated with linear increases in sEMG amplitude which is thought to reflect a fatigue-induced increase in motor unit recruitment¹⁷. During maximal sprinting exercise, it is possible that BFR would adversely affect power output which would result in an individual sprinting at a lower percentage of their previously determined PWC_{FT}. For example, applying BFR during exercise accelerates muscle fatigue by trapping metabolic byproducts within the exercising muscle(s)¹⁸. It has yet to be determined how BFR may affect maximal sprinting performance as it relates to an individual's PWC_{FT}. Thus, the collective assessment of local physiological changes would provide valuable information regarding the effects of

SIT with and without BFR. No previous investigations have examined the effects of IBFR or CBFR during SIT training on PWC_{FT}. Therefore, the purpose of this investigation was to examine the effects of CBFR and IBFR applied during SIT on performance and neuromuscular function. Based on previous findings^{6,7}, it was hypothesized that SIT with IBFR and CBFR would result in greater neuromuscular fatigue and reduce the % of PWC_{FT} achieved during SIT relative to No-BFR.

Material and methods

Experimental Approach

College aged men performed a graded exercise test on a cycle ergometer (Visit1) and then randomly completed on separate days (≥ 48 hours), 3 separate SIT protocols with IBFR, CBFR, and No-BFR (Visits 2-4). Each SIT protocol consisted of 2, 30s sprints with 2 minutes of rest between sprints at a resistance of 7.5% of body mass. Prior to (pre-SIT) and after (post-SIT) each SIT protocol, maximal strength, neuromuscular function, and muscle thickness (MT) were measured. During each sprint, peak and mean revolutions per minute (RPM) as well as % of PWC_{FT} were determined.

Subjects

Fifteen actively trained men (mean age \pm SD = 23 \pm 2 yrs; height = 176.0 \pm 6.2 cm; body mass = 78.8 \pm 13.0 kg) participated in this study and performed SIT with CBFR, IBFR, and No-BFR. During the strength assessments, sEMG data from 11 of the 15 subjects was used for analysis as there were four incomplete datasets (Subjects: 7,9,10, and 15) due to hardware malfunction. Subjects had no known cardiovascular, pulmonary, metabolic, muscular, and or coronary heart disease, as well as no daily use of prescription medications or dietary supplements. Actively trained was defined as a tier 2 on the participant classification framework, described as regularly training approximately three times per week, identify with a specific sport, and train with a purpose to compete¹⁹. Subjects visited the laboratory on four separate occasions: one familiarization and baseline graded exercise test (GXT) and three randomly allocated testing visits. All subjects completed a health history questionnaire and signed a written informed consent prior to all testing. Subjects were told to refrain from exhaustive lower body exercise at least 48 hours prior to testing and asked at the beginning of their visit if they felt physically able to perform maximal intensity exercise, if unable, their visit was rescheduled.

Familiarization and Baseline

The first laboratory visit consisted of a written informed consent and health history questionnaire followed by a familiarization of the testing protocols. During the familiarization, subjects' BFR arterial occlusion pressure was determined using Doppler ultrasound and a rapid-cuff-inflator (Hokanson E20 Rapid Cuff Inflator; Hokanson Inc., Bellevue, WA, USA). Subjects then performed maximal strength testing

on an isokinetic dynamometer (Biodex system 3, Shirley, NY, USA) and practiced performing maximal sprints on a cycle ergometer (894 E, Monark, Vansbro, Sweden). Subjects then performed a GXT to determine both PWC_{FT} and aerobic capacity.

Maximal Isometric Strength Familiarization

Following a 5-minute warm up on a cycle ergometer (Corval 400, Groningen, The Netherlands), subjects completed three maximal voluntary isometric contraction (MVIC) leg extension muscle actions on an isokinetic dynamometer (Biodex system 3, Shirley, NY, USA). For each MVIC attempt, the leg was positioned at a knee joint angle of 90° , where 180° corresponded to full extension at the knee and a 90° hip angle. Each MVIC muscle action was initiated as rapidly and forcibly as possible and maintained for a duration of three seconds. A rest period of 90 seconds was provided between attempts and the highest MVIC torque (Nm) produced during the three attempts was used for further analyses.

Maximal Sprinting Familiarization

Following the assessment of MVIC torque, subjects were seated on a mechanically braked ergometer (894 E, Monark, Vansbro, Sweden) and practiced performing maximal sprints. Each practice sprint was performed for a duration of four seconds at a resistance of 7.5% of total body mass^{4,6}. Subjects performed 2-5 sprints until they were comfortable with the SIT protocol.

Graded Exercise Test

Subjects were equipped with a heart rate monitor (Polar H10, Polar Electro Oy, Kempele, Finland) and fitted to a silicone facemask (7450 V2, Hans Rudolph, Inc., Shawnee, KS, USA) with a two-way non-rebreathing valve prior to being seated and fitted on a cycle ergometer (Corval 400, Groningen, The Netherlands). Maximal aerobic capacity was examined using a breath-by-breath gas analysis (10-second rolling average) recorded on a metabolic cart (TrueMax 2400, ParvoMedics, Sandy UT, USA) that was used to determine the maximum volume of oxygen consumption in ml/kg/min ($\dot{V}O_{2max}$). sEMG sensors were placed on the subjects' dominant leg to determine the sEMG-based PWC_{FT} . The GXT began at 90 watts with a pedal cadence of 70 ± 5 rpm and increased by 30 watts every three minutes until volitional exhaustion, that was determined when subjects could no longer sustain the pedal cadence for a 5-second period despite strong verbal encouragement.

Muscle Thickness

Upon entering the laboratory and a brief rest period, subjects lied supine on a padded table to measure the thickness of the vastus lateralis (VL) muscle of their dominant leg. MT was measured using a portable brightness mode Doppler ultrasound imaging device (Logiq e, General

Electric, Chicago IL, USA) and a multifrequency linear-array probe. All ultrasound assessments were determined at 50% of the distance of the subjects' greater trochanter to the lateral aspect of their patella with their leg internally rotated²⁰. The ultrasound images were captured using a gain of 50 dB and a frequency of 12 Hz with a default depth of 5 cm. These settings were kept consistent across subjects, only adjusting depth on a subject-by-subject basis. To enhance acoustic coupling and reduce near-field artifacts, water-soluble transmission gel was applied to the skin for each of the ultrasound assessments. Assessment of MT was also determined following post-SIT strength testing for each visit.

Maximal Strength Testing

Following a 5-minute warm up on a cycle ergometer (Corval 400 Groningen, The Netherlands), subjects performed maximal concentric leg extension muscle actions followed by MVIC muscle actions on an isokinetic dynamometer (Biodex system 3, Shirley, NY, USA) to determine concentric peak torque (CPT) and MVIC torque, respectively. Subjects completed one set of three maximal concentric leg extension muscle actions at a velocity of $90^\circ/s$, performed through a 90° range of motion (90° - 180° of knee extension, where 180° corresponded to full extension at the knee). Following a 60-s rest period, subjects then completed three MVIC muscle actions performed at 90° sustained for a period of 3-s, each separated by 90-s of rest. Prior to post-SIT MT assessments, maximal strength testing was performed following each SIT protocol where rest between attempts was limited to 3-5 seconds to minimize recovery. Specifically, CPT muscle actions were performed once participants were resealed on the isokinetic dynamometer (~15s) after the second 30-s sprint, and MVICs were performed immediately after the CPT muscle actions (~30s after the second sprint).

Sprint Interval Protocol

The SIT protocol consisted of 2, 30-second sprints performed on a mechanically braked cycle ergometer (894 E, Monark, Vansbro, Sweden) with 2 minutes of rest between the first and second sprint. Specifically, during pilot testing, no subject performed more than two sprints with both IBFR and/or CBF due to accumulated fatigue and discomfort. Each sprint utilized a resistance of 7.5% of the subjects' body mass and was initiated from a rolling start that progressively increased in pedal cadence in the three seconds preceding each sprint^{4,6}. The participant remained seated on the ergometer during each sprint and the passive rest period. Specifically, during each rest period, subjects were instructed to remain seated with their dominant foot extended and to avoid pedaling. Peak RPM was recorded for each sprint for all conditions as the highest 5-s value recorded. Mean RPM was derived from six, 5-s averages recorded during each 30-s sprint for each condition as well.

Blood Flow Restriction

Subjects completed a SIT protocol with three randomly ordered BFR conditions: IBFR, CBFR, and No-BFR. During the CBFR intervention, bilateral BFR was applied for the duration of the two, 30-second sprints and during the 2-minute rest period allotted between sprints. Bilateral IBFR was applied during the 2-minute rest period between sprints but not applied during the sprints. For the No-BFR condition, bilateral BFR was not applied during the sprints or rest period. Both IBFR and CBFR conditions were performed at 60% of arterial occlusion pressure that was determined during each visit²¹. Specifically, Doppler ultrasound was used to examine muscle blood flow from the posterior tibial artery and to determine when total arterial occlusion was reached. Total arterial occlusion pressure was achieved by rapidly inflating (increments of 1-30 mmHg) and deflating (completely) the cuff until total arterial occlusion was identified. Each inflation and deflation was approximately five seconds in duration to avoid accentuated vasodilation from an increase in arterial nitric oxide secretion that is enhanced under ischemic/hypoxic conditions²².

Electromyography

sEMG was assessed from the dominant leg using a Delsys Avanti Trigno sensor (Delsys Inc.). Before electrode placement, the area was shaved and cleaned with an alcohol swab. The sensor was placed on the VL muscle in accordance with SENIAM recommendations²³. Specifically, the sensor was placed at 66% of the distance from the anterior superior iliac spine to the lateral aspect of the patella and oriented towards the pennation angle of the muscle (20°) with the leg internally rotated¹⁶. The analog sEMG signals were digitized at 2,148 Hz with EMGworks-acquisition software (Delsys Inc., Natick MA, USA) and stored on a laboratory computer (i7 Dell XPS 15 9570, Round Rock, TX) for subsequent offline analysis. The signals were amplified x1,000,000 and digitally bandpass filtered (zero-phase shift, fourth-order Butterworth) at 10-500 Hz (Basmajian, 1978). sEMG amplitude (root-mean-squared: RMS) and mean power frequency (MPF) were processed offline using a custom-written LabVIEW, 2021 (National Instruments, Austin TX, USA) program. RMS and MPF were calculated and normalized to the MVIC and CPT attempts that produced the highest torque. MPF was derived using a Hamming window on the power density spectrum and using discrete Fourier transform algorithm. Signals were expressed as a % change relative to pretest values, where pretest was 100%. The equation used to determine % change was:

$$\Delta\% = \left(\frac{\text{Posttest value}}{\text{Pretest value}} \times 100 \right) - 100.$$

Physical Working Capacity at the Fatigue Threshold (PWC_{FT})

PWC_{FT} was determined from the GXT performed during visit 1. During each 3-min stage of the GXT, intermittent

10-s epochs were selected for a total of nine 10-s samples collected during each stage²⁴. To determine PWC_{FT} , sEMG amplitude (μV) was determined for each 10-s epoch. The nine 10-s epochs for each stage were then plotted across time and a slope coefficient was determined via linear regression ($\mu\text{V}/10\text{s}$). PWC_{FT} was identified as the combined average of the power output from the last stage that resulted in a non-significant increase in the slope coefficient of the linear regression analysis with the power output from the first stage that resulted in a significant increase in the slope coefficient of a linear regression analysis²⁵. $\%PWC_{FT}$ was determined from the mean power output during the sprints relative to the PWC_{FT} determined from the GXT.

Statistical Analysis

To examine the acute effects of each SIT protocol, separate 3-way (Condition [Continuous BFR, Intermittent BFR, No-BFR]) x 2 (Time [Pre-SIT, Post-SIT]) repeated measures analyses of variance (ANOVA) were used to analyze CPT, MVIC torque, MT, as well as RMS and MPF responses during the CPT and MVIC muscle actions. During each sprint, $\%PWC_{FT}$, peak RPM, and mean RPM were examined using separate 2-way (Sprint [Sprint 1, Sprint 2]) x 3 (Condition [Continuous BFR, Intermittent BFR, No-BFR]) repeated measures ANOVAs. Greenhouse-Geisser corrections were used if sphericity was not met according to Mauchly's test of sphericity. Significant ($p < 0.05$) interactions were decomposed, and follow-up Bonferroni-corrected dependent samples t-tests were performed when necessary. Partial eta squared effect sizes (η_p^2) were calculated for each ANOVA, and classified as small ($\eta_p^2 = 0.01$), medium ($\eta_p^2 = 0.06$), and large ($\eta_p^2 = 0.14$)²⁶. All statistical analyses were performed using IBM SPSS v. 27 (Armonk, NY) and an alpha of $p \leq 0.05$ was considered statistically significant for all comparisons.

Results

GXT

Subjects' $\dot{V}O_{2\text{max}}$ and PWC_{FT} were determined from the GXT performed during the baseline visit. Mean $\dot{V}O_{2\text{max}}$ was 38.0 ± 6.1 ml/min/kg ($n=14$, one subject did not reach maximal capacity), and mean power output for PWC_{FT} was 143 ± 39.6 W ($n=15$).

RPM

As shown in Figure 1, there were significant interactions for peak ($p=0.005$, $\eta_p^2=0.315$) and mean ($p=0.030$, $\eta_p^2=0.221$) RPM. Follow-up analyses of simple main effects indicated that peak RPM decreased from Sprint 1 to Sprint 2 for No-BFR (157.7 ± 12.5 to 147.5 ± 12.8 RPM), CBFR (153.9 ± 14.5 to 129.2 ± 13.5 RPM) and IBFR (158.0 ± 14.4 to 134.1 ± 15.7 RPM). Similarly, Mean RPM also decreased from Sprint 1 to Sprint 2 for No-BFR (110.4 ± 7.1 to 85.5 ± 9.9 RPM), CBFR (105.2 ± 11.5 to 73.6 ± 14.0 RPM) and IBFR (110.3 ± 8.6 to 81.2 ± 12.5 RPM). Additionally, there were no differences

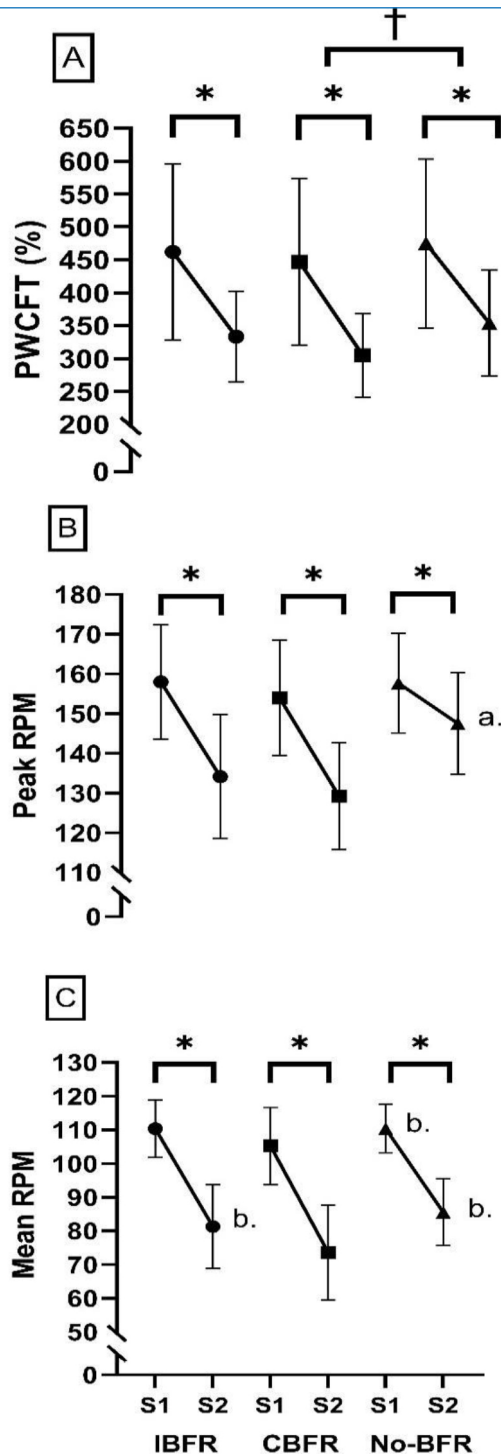


Figure 1. Mean \pm SD values for physical working capacity at the fatigue threshold (PWC_{FT} ; A), peak revolutions per minute (RPM; B), and mean (RPM; C) determined during sprint interval training (SIT). PWC_{FT} , peak, and mean RPM were determined for both Sprint 1 and Sprint 2. SIT was performed with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (No-BFR). *Significant ($p \leq 0.05$) main or simple main effect for Sprint (Sprint 1 > Sprint 2). †Significant ($p \leq 0.05$) main effect for Condition (No-BFR > CBFR). a. Significant ($p \leq 0.05$) simple main effect for Condition (>CBFR & IBFR during sprint 2 alone). b. Significant ($p \leq 0.05$) simple main effect for Condition (> CBFR).

between conditions for peak RPM during Sprint 1, while during Sprint 2 No-BFR was greater than IBFR and CBFR (i.e., No-BFR > IBFR and CBFR). Furthermore, during Sprint 1, mean RPM was greater for No-BFR than CBFR, while during Sprint 2, both No-BFR and IBFR were greater than CBFR (i.e., Sprint 1, No-BFR > CBFR; Sprint 2, No-BFR and IBFR > CBFR).

Muscle Thickness and Maximal Strength

There were no significant Condition \times Time interactions for MT ($p=0.792$, $\eta_p^2=0.016$), CPT ($p=0.203$, $\eta_p^2=0.111$), or MVIC torque ($p=0.519$, $\eta_p^2=0.039$). There were, however, main effects for Time, but not Condition for MT ($p<0.001$, $\eta_p^2=0.591$), CPT ($p<0.001$, $\eta_p^2=0.761$) and MVIC torque ($p<0.001$, $\eta_p^2=0.793$). Specifically, collapsed across Condition, MT increased from pre-SIT (1.77 ± 0.31 cm) to post-SIT (1.96 ± 0.30 cm), while CPT and MVIC torque decreased from pretest (220.3 ± 47.6 Nm and 355.1 ± 72.5 Nm, respectively) to posttest (147.9 ± 27.7 Nm and 252.2 ± 45.5 Nm, respectively).

sEMG Amplitude and sEMG Mean Power Frequency

There were no significant interactions for RMS or MPF during the CPT or MVIC muscle actions. Indicated in Figure 2, there were, however, main effects for Time, but not Condition for MPF during the CPT ($p<0.001$, $\eta_p^2=0.778$) and MVIC ($p=0.005$, $\eta_p^2=0.564$) muscle actions. Specifically, collapsed across Condition, CPT and MVIC MPF decreased (-12.8 ± 10.5 and -8.7 ± 10.2 %, respectively) from pre-SIT to post-SIT.

% PWC_{FT}

There was no significant interaction ($p=0.258$, $\eta_p^2=0.092$) for % PWC_{FT} , but there were significant main effects for Sprint ($p<0.001$, $\eta_p^2=0.784$) and Condition ($p<0.001$, $\eta_p^2=0.409$). Specifically, collapsed across Condition, % PWC_{FT} was greater during Sprint 1 ($461.2 \pm 126.9\%$) than Sprint 2 ($330.7 \pm 72.7\%$). Furthermore, collapsed across Sprint, % PWC_{FT} was greater during No-BFR ($414.2 \pm 121.9\%$) than CBFR ($375.9 \pm 121.9\%$), while IBFR ($397.7 \pm 123.2\%$) was not different from CBFR and No-BFR, collectively indicated in Figure 1.

Discussion

The application of IBFR and CBFR elicits similar acute physiological responses as No-BFR when applied during SIT. Specifically, there were similar fatigue-induced reductions in maximal strength (i.e., CPT and MVIC) and an increase in MT among all three conditions. The reductions in maximal strength were unrelated to changes in muscle excitation (RMS) but may have been due, in part, to excitation-contraction coupling failure as evidenced by the reduction in MPF. Despite a lack of differences among these physiological responses, % PWC_{FT} as well as peak and mean RPM were higher during No-BFR relative to CBFR. Collectively, these

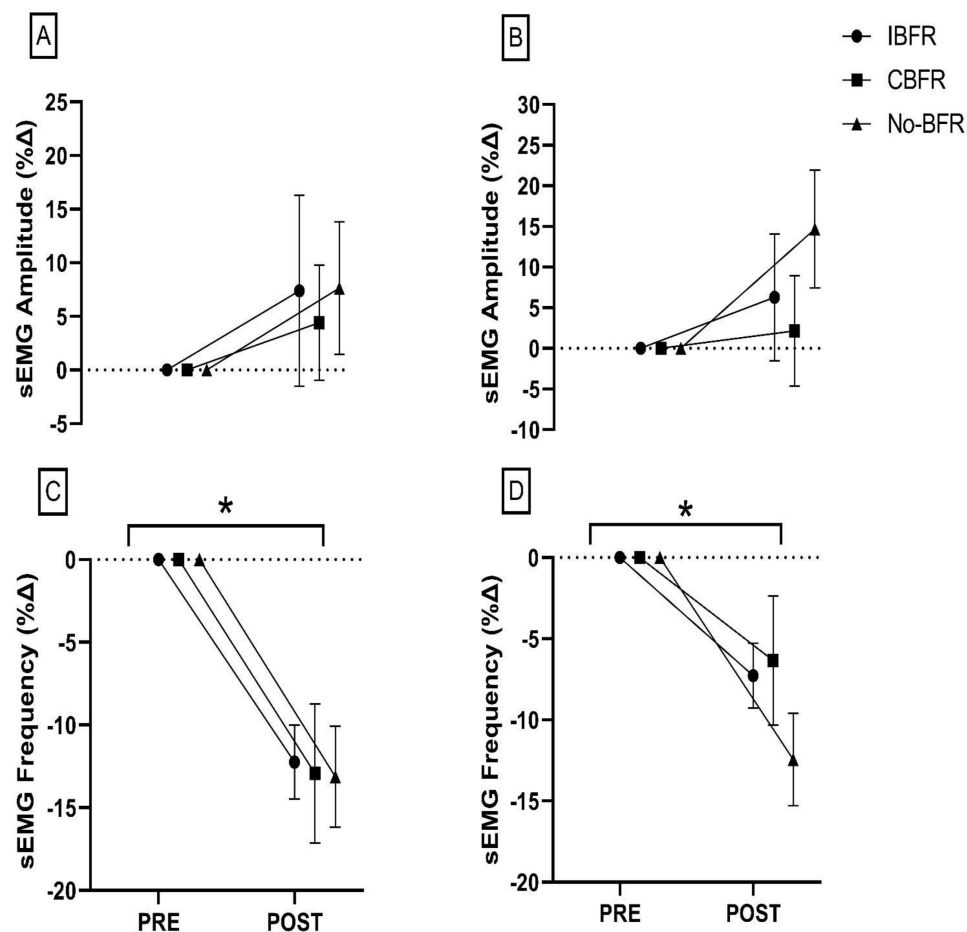


Figure 2. Mean \pm SD values (% Δ) for surface electromyographic (sEMG) amplitude and frequency obtained during the concentric peak torque (CPT: A and C) and maximal voluntary isometric contraction (MVIC: B and D) muscle actions. CPT and MVIC sEMG amplitude and frequency were recorded prior to (PRE) and immediately after (POST) sprint interval training (SIT). SIT was performed with continuous blood flow restriction (CBFR), intermittent blood flow restriction (IBFR), and no blood flow restriction (No-BFR). *Significant ($p \leq 0.05$) main effect for Time (POST < PRE).

findings suggested that SIT with or without CBFR and IBFR elicited comparable physiological responses, although there were differences in some of the performance measures (i.e., %PWC_{FT} and RPM) among conditions.

Performance Measures

In the present study, SIT elicited comparable individualized average percent reductions in CPT ($-31.0 \pm 14.2\%$) and MVIC torque ($-27.6 \pm 12.6\%$) and similar individualized average percent increases in MT ($11.4 \pm 11.0\%$) from pre-SIT to post-SIT across all 3 conditions. Furthermore, there were reductions in peak and mean RPM from Sprint 1 to Sprint 2 for all conditions that, in general, decreased by a greater extent for CBFR than No-BFR. The findings of the present study were partially consistent with previous investigations^{6,7}

that have examined the acute effects of SIT with and without BFR. For example, like the present investigation, MVIC torque decreased similarly for CBFR ($-10.5 \pm 12.1\%$) and No-BFR ($-16.5 \pm 9\%$) from pre-SIT to post-SIT following 10-s sprints of arm cycling to exhaustion with 20 seconds of active recovery between sprints⁷. Contrarily, following three separate sessions of 10-s maximal cycling sprints to exhaustion (cadence < 70RPM), MVIC torque was lower for CBFR (138 ± 82 Nm) than No-BFR (255 ± 114 Nm) relative to pre-SIT values (263 ± 124 Nm and 279 ± 140 Nm, respectively)⁵. Additionally, Kojima et al. reported decreases (approximately 16-23%) in mean power output relative to body mass after 3-5 10-s sprints (40-s passive rest), although there were no differences between BFR and No-BFR conditions. The inconsistency among the performance measures between the present study and previous investigations⁵⁻⁷ are likely

due, in part, to the heterogeneity among protocols (number of sprints, duty cycle), modality, and BFR pressure (45%, 60%, 140 mmHg).

Neuromuscular Responses

In the present study, SIT with and without BFR did not affect RMS, but elicited similar reductions in MPF assessed during the CPT and MVIC muscle actions. Specifically, there were no changes in RMS during the CPT ($+6.5 \pm 22.5\%$ change from pretest) or MVIC ($+7.7 \pm 24.1\%$ change from pretest) muscle actions, while MPF decreased by $12.8 \pm 10.5\%$ during the CPT and $8.7 \pm 10.2\%$ during the MVIC muscle actions across all three conditions. The lack of change in RMS suggested that the SIT intervention did not adversely affect muscle excitation or the ability to maximally excite all available motor units²⁷. The reduction in MPF, however, suggested there were fatigued-induced decreases in action potential conduction velocity (APCV) possibly due to the build-up of metabolites. For example, fatigue-induced reductions in MPF have been attributed to the build-up of metabolites which has been shown to adversely affect APCV²⁸⁻³⁰. Specifically, MPF is sensitive to changes in APCV which is inversely related to metabolic increases of H^+ and lactate. It is of note how SIT with CBFR did not exhibit a greater decrease in MPF compared to IBFR and No-BFR, possibly due to the high intensity of SIT (highly anaerobic) that results in naturally increased concentrations of H^+ and lactate that are comparable during occluded and non-occluded conditions^{5,8}. Collectively, the findings of the present study indicated that SIT with or without BFR did not affect muscle excitation but did induce comparable reductions in APCV across all conditions.

%PWC_{FT}

In the present study, collapsed across condition, %PWC_{FT} was greater during sprint 1 ($461.2 \pm 126.9\%$) than sprint 2 ($330.7 \pm 72.7\%$) which was consistent with the fatiguing nature of repeated sprint exercise³¹⁻³⁴. Furthermore, collapsed across Sprint, %PWC_{FT} was greater during No-BFR ($414.2 \pm 121.9\%$) than CBFR ($375.9 \pm 121.9\%$) with no difference between IBFR ($397.7 \pm 123.2\%$) and No-BFR. The sEMG-based PWC_{FT} has been applied to examine neuromuscular function during progressive exercise bouts to demarcate the onset of neuromuscular fatigue (i.e., linear increase in muscle excitation possibly due to increased recruitment of additional motor units). Thus, theoretically, 100% of PWC_{FT} represents the highest power output that could be achieved prior to power- and/or fatigue-induced increases in muscle excitation. Therefore, in the present study, regardless of condition, each 30-s sprint achieved approximately 3-5 times the power output of the PWC_{FT}, likely eliciting a potent stimulus on neuromuscular and cardiovascular function. Furthermore, despite lower %PWC_{FT} for CBFR than No-BFR, collapsed across Sprint, all sprints substantially exceeded the PWC_{FT} threshold suggesting that all SIT protocols necessitated robust increases in muscle

excitation which were not augmented in either BFR protocol. The lack of change in muscle excitation across conditions may be related to metabolite concentration achieved during SIT. When an individual sustains a contraction, muscle excitation increases to recruit additional motor units and maintain power output³⁵. These increases in excitation relate to an increase in metabolite concentration that results in recruitment of higher threshold motor units. Muscle actions performed with BFR have previously demonstrated greater increases in muscle excitation however they were performed submaximally³⁶. Therefore, performing maximal muscle actions during SIT may elicit enough metabolite concentration to plateau muscle excitation regardless of occlusion. Collectively, the results of the present study suggested that SIT exhibits a potent effect on muscle function, without affecting excitation, and achieved a power output that of 3 to 5-fold larger than PWC_{FT}.

Conclusion

In the present study, SIT elicited comparable reductions in leg extension strength and similar increases in MT from pre-SIT to post-SIT across all three conditions. There were also differences in reductions in peak and mean RPM from Sprint 1 to Sprint 2 for all conditions. Specifically, during Sprint 2 peak RPM was greater during No-BFR than IBFR and CBFR while mean RPM was greater for No-BFR and IBFR than CBFR. Regardless of condition, each 30-s sprint achieved approximately 3-5 times the power output of the PWC_{FT}. SIT with and without BFR did not affect RMS but elicited similar reductions in MPF during both CPT and MVIC muscle actions although %PWC_{FT} was greater during sprint 1 than sprint 2 and greater during No-BFR than CBFR. Collectively, the findings of the present study indicated that SIT with or without BFR did not affect neuromuscular function and induced comparable alterations in neuromuscular fatigue across all conditions. However, SIT with CBFR exhibited lower sprint performance with similar neuromuscular responses, suggesting an increased difficulty when performed that is not reflected by neuromuscular recruiting strategies, but potentially due to increased localized pain or discomfort from continuous inflation during high intensity work-bouts.

Ethics Approval

This study was approved by the University of Central Florida Institutional Review Board in compliance with their regulations. IRB Number: STUDY00003396.

Consent to participate

All subjects signed a written informed consent prior to all testing.

Authors' Contributions

DHGR, AMW, ECH, MSS and JRS conceived the study concept and design. DHGR, AMW, PMR and CEP carried out data acquisition. DHGR performed data analysis and was the primary author. AMW, PMR, CEP, JEL, MSS, JRS, FB, and ECH aided in manuscript development and editing. All authors approved the final version of this manuscript.

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