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INVITED REVIEW

The effect of blood flow occlusion during acute low-intensity isometric elbow flexion exercise

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Abstract

Purpose Blood flow restriction (BFR) with low-intensity (< 30% of 1 repetition maximum strength) muscle contraction has been used chronically (> 4 weeks) to enhance resistance training. However, mechanisms underlying muscle adaptations following BFR are not well understood. To explore changes related to chronic BFR adaptations, the current study used blood flow occlusion (BFO) during an acute bout of low-intensity isometric fatiguing contractions to assess peripheral (muscle) factors affected.

Methods Ten males completed separate fatiguing elbow flexor protocols to failure; one with BFO and one with un-restricted blood flow (FF). Baseline, post-task failure, and 30 min of recovery measures of voluntary and involuntary contractile properties were compared.

Results BFO had greater impairment of intrinsic measures compared with FF, despite FF lasting 80% longer. Following task failure, maximal voluntary contraction and 50 Hz torque decreased in both protocols (~ 60% from baseline). Voluntary activation decreased ~ 11% from baseline at failure following both protocols, but recovered at a faster rate following BFO, whereas MVC recovered to ~ 90% of baseline in both protocols. The 10/50 Hz torque ratio was decreased by ~ 68% and ~ 21% from baseline, for BFO and FF, respectively ($P < 0.01$). 50 Hz half-relaxation-time (HRT) was significantly longer immediately following BFO (~ 107% greater than baseline), with no change following FF.

Conclusions Thus, greater peripheral fatigue that recovers at a similar rate compared to conventional exercise is likely driving muscle adaptations observed with chronic BFR exercise. Likely BFO alters energy demand and supply of working muscle similar to chronic BFR, but is exaggerated in this paradigm.

Keywords Blood flow restriction · Elbow flexion · Fatigue · Intrinsic muscle properties · Low-frequency fatigue · Peripheral fatigue

Introduction

It is generally accepted that resistance training with contractile intensities of ~ 70% of maximal voluntary contraction (MVC), are required to stimulate muscle hypertrophy and strength gains (ACSM 2009; Ratamess et al. 2009). However, evidence supports the use of blood flow-restricted (BFR) exercise, sometimes referred to as occlusion exercise, as a training technique that elicits hypertrophy and strength gains by incorporating low-intensity muscular contraction (~ 20–30% of MVC) in combination with BFR (Yasuda et al. 2008, 2011). Although it is commonly reported that strength and hypertrophy gains are maximized at relatively high-intensity, other studies report low levels of intensity (< 50% of MVC) performed to failure that will elicit similar gains in strength and hypertrophy (Kumar et al. 2009;

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Burd et al. 2010). These results would indicate that sufficient protein synthesis, and therefore, muscle hypertrophy can occur at lower exercise intensities than previously suggested (Ratamess et al. 2009). BFR is most often achieved through the application of external pressure around the limb segment over the working muscle. External pressure can be applied using a sphygmomanometer that applies a quantifiable pressure (mmHg), or by using elastic bands that cannot provide an exact measure of restriction. Whereas the physiological mechanisms are not fully understood, BFR training has been observed to facilitate positive muscular adaptations in both rehabilitation (Ohta et al. 2003) settings and athletes (Cook et al. 2014). Specifically, blood flow-restricted exercise has been explored from a chronic adaptation perspective over 4 or more weeks of exercise training in both young and elderly populations, as well as in healthy and rehabilitation settings (Ozaki et al. 2011; Abe et al. 2010). These studies observed increases in hypertrophy, strength (Manimmanakorn et al. 2013a; Abe et al. 2010; Ozaki et al. 2011), and muscle endurance (Manimmanakorn et al. 2013a). Hypertrophy has been assessed by changes in muscle girth, and through magnetic resonance imaging to evaluate CSA (cross-sectional area) (Manimmanakorn et al. 2013a; Ozaki et al. 2011; Abe et al. 2010). Restricting or occluding blood flow essentially attenuates oxygenated blood and blood-borne substrates (glucose and free fatty acids) from reaching the working muscle (Moritani et al. 1992), with greater levels of restriction causing greater attenuation. This localized hypoxic stimulus may be an important mechanism that in combination with low-load exercise contributes to positive strength adaptations (Scott et al. 2015). In addition, the effect of BFR distal to the restriction, likely causes a larger accumulation of metabolic by-products due to the greater hypoxic state (Scott et al. 2015). This presumably causes aerobic fibers (type I slow motor units), to metabolize ATP in a low-oxygen anaerobic environment rather than a preferred oxygen-rich environment, and therefore recruitment of anaerobic fibers (type II fast motor units) is enhanced to offset and maintain a given contraction intensity. Type II fibers produce more force per fiber than type I, but are far less fatigue resistant (Greising et al. 2012). Although this concept has not been directly assessed, indirect measures would manifest as shorter time to fatigue, and relatively greater neural activation of muscle reflected in the electromyographic (EMG) signal (Manimmanakorn et al. 2013b; Yasuda et al. 2008). A study by Karabulut et al. (2010) reported in the thigh muscle (vastus lateralis) an increase in EMG amplitude across isotonic submaximal repetitions with blood restriction set at 44% greater than systolic blood pressure. Towards the end of the intervention contractions EMG declined indicating some neural drive failure at the muscle level, although it must be recognized there are surface EMG limitations during dynamic contractions (see Farina et al. 2004). Also, they did not go to failure, or explore the recovery profile. In another study, a continuous increase in EMG indicated contractile output during complete blood flow occlusion was maintained by greater neural output (Moritini et al. 1992), Furthermore, Yasuda et al. (2009) noted that under complete blood flow occlusion (BFO) with low-level fatiguing contractions there was an extreme mismatch in energy demand (increasing muscle activation, or neural compression) and energy supply (no blood flow) that resulted in feelings of maximal exertion and complete mechanical failure.

Neuromuscular fatigue has both voluntary and involuntary components. The involuntary (peripheral) component can be assessed from electrically evoked contractions of the muscle, which bypasses the central nervous system. During prolonged contraction of a moderate to high intensity, the muscle fibers become weaker and contract slower, often due to muscle damage (Alway et al. 1990) or excitation–contraction (E–C) failure (Edwards et al. 1977). Excitation–contraction coupling failure is one of a number possible causes of fatigue (Jones 1996), and can be evaluated indirectly following fatiguing contractions by comparing the response of the muscle at lower frequencies of tetanic stimulation (i.e., <20 Hz) to the response from maximal frequencies of excitation (i.e., 50 Hz), (Jones 1996). An increase in this ratio (10–50 Hz) after fatiguing contractions is an indication of peripheral fatigue and is referred to as low-frequency fatigue (LFF). Central components of fatigue are more difficult to evaluate because they include assessing the degree of voluntary drive generated from spinal and supraspinal factors. Voluntary activation (VA) of the system can be indirectly assessed using the interpolated twitch technique (ITT) (Behm et al. 1996). Following fatiguing low-intensity blood flow-restricted exercise VA was decreased (Karabulut et al. 2010) and EMG, as a measure of voluntary neural activation, was increased. These changes were interpreted as an increase in central fatigue following low-intensity blood flow-restricted exercise (Karabulut et al. 2010).

Thus, assessing changes in neural activation and muscle properties using an acute bout of fatiguing exercise with a high level of occlusion should provide further understanding of relevant peripheral factors affected during chronic BFR training paradigms. Therefore, the purpose of this experiment was to quantify the degree of fatigability in response to prolonged steady-state contraction of the elbow flexors at low-intensity (20% MVC) with a high level of external pressure (250 mmHg) to failure, compared with the responses to the same exercise at 20% MVC during unrestricted blood flow to failure (FF). We hypothesized, H1: that BFO exercise will cause exercise induced voluntary failure in a shorter absolute time, and H2: that BFO will cause greater peripheral fatigue compared to the unrestricted exercise. H3: that the recovery following a greater amount of peripheral fatigue caused by BFO, will be attenuated compared to the

FF protocol. Electrically evoked tetanic responses were used to specifically target intrinsic (peripheral) changes in the muscle and voluntary activation in combination with time to fatigue and EMG were used as measures to assess overall fatigability of the system.

Methods

Subjects

Ten healthy male subjects (see Table 1 for characteristics) participated in two testing trials administered in a random order and each was separated by at least 48 h, with both completed within 7 days. All procedures were approved by the local institutional ethics committee (REB# 107212, WREM at The University of Western Ontario) and conformed to the declaration of Helsinki. Written and verbal consent were obtained from each participant. All subjects were right-hand dominant and therefore to minimize limb dominance effects, the non-dominant left arm was tested in each participant.

Experimental set-up

Participants were supine on an examination plinth with their legs supported by a large wooden box (to minimize extraneous movement of the lower limbs) placing their hip and knee joints at ~ 90°. To record elbow flexion, the left arm was in the dependent position and the elbow joint was flexed to ~ 90° with the supinated wrist secured to a linear force transducer (SST-700-100A; AS Technology, Haliburton, ON, Canada) using a velcro strap. The force transducer was adjusted so that force was measured at the wrist in a standardized fashion for all participants. A brace was adjusted to press down firmly on the left shoulder to minimize any extraneous shoulder or torso movements, as well as in inelastic strap was fastened securely across the chest. EMG of the flexors was recorded, via adhesive Ag–AgCl electrodes (Kendall, H59P cloth electrodes) arranged in a monopolar fashion. Recording electrodes were placed on the skin over the mid-belly of the muscle and reference electrodes were secured over the ulna on the posterior forearm. For elbow flexor muscle stimulation custom made aluminum foil electrode pads (~ 2 × 5 cm) covered in damp paper towel were placed over the distal and proximal 1/3 of the arm flexors.

In all sessions, torque and EMG data were recorded using an A/D converter (CED 1401; Cambridge Electronic Design Ltd, Cambridge, UK) in conjunction with Spike2 software (v. 7.02; Cambridge Electronic Design). The torque and EMG data were sampled at 500 and 5000 Hz, respectively. EMG data were amplified (× 1000) and bandpass filtered (10 Hz—20 KHz, with a 60 Hz notch filter) using Neurolog; NL844, Digitimer, Welwyn Garden City, UK.

Table 1 Values are means ± SD

Parameters	Participants ($n = 10$ males)
Age (years)	27 ± 4
Height (cm)	178 ± 7
Mass (kg)	84.9 ± 10.2
BFO time to failure (s)	234 ± 44[a]
FF time to failure (s)	1026 ± 752
BFO MVC (Nm)	337.1 ± 76.2
FFfail MVC (Nm)	322.6 ± 66.2

BFO, blood flow occlusion at 20% intensity. FF, no blood flow occlusion at 20% intensity. MVC, maximal voluntary isometric contraction

[a]Denotes significant difference between BFO and FF time to failure in seconds

Experimental protocol

Participants were randomly assigned to complete either first the low-intensity BFO to failure protocol or the low-intensity unrestricted blood flow (FF) to failure protocol. Except for the addition of the blood pressure cuff to restrict blood flow all procedures were identical in both trials. The blood pressure cuff (adult sized sphygmomanometer, 10 cm in width), was applied over the most proximal portion of the biceps muscle belly and pressure was maintained at 250 mmHg during the BFO protocol for all participants. This pressure was sufficient to abolish the radial pulse during the resting state for all participants, To determine MVC strength and voluntary activation of the elbow flexors maximal electrical stimulation doublets (200 µs pulse width; 400 V; 100 Hz doublet; range 90–168 mA) were applied using a constant current stimulator (DS7AH; Digitimer, Hertfordshire, UK) by increasing current intensity until the torque response no longer increased with an increase in current intensity, or coactivation of other muscles impeded the elbow flexor torque. For the interpolated twitch technique (ITT) (Behm et al. 1996) doublets were used to assess voluntary activation in the elbow flexors. Participants were instructed to perform 2–3 brief (~ 3–5 s) elbow flexion MVCs, which included a superimposed doublet at the peak torque, and a potentiated doublet was applied at rest immediately following the MVC. If variability in maximal torque was 5% or more between MVCs then a third MVC was performed. It has been reported that inexperienced participants may produce less than maximal MVCs when expecting electrical stimuli (Button and Behm 2008), thus multiple MVCs were conducted for familiarization. Two to three minutes of rest was given between each MVC. Strong verbal encouragement and visual feedback were provided during all voluntary contractions. The greatest elbow flexion MVC was selected as the

baseline value. Tetanic 50 Hz (200 µs pulse width; 400V; 1 s duration; range 35–90 mA) stimulation was applied for 1 s to elicit 30% of elbow flexor MVC torque which from pilot testing was tolerable and did not activate antagonist muscles of the arm. Stimulations at 1 Hz (twitch) and 10 Hz (same stimulation parameters as 50 Hz) were applied using this same intensity. After baseline measures were acquired (at the beginning of each testing day), participants completed one of the two intervention protocols. Participants were instructed to maintain 20% of MVC constant isometric contraction, while elbow flexor MVCs (~3–5 s) with ITT were assessed every minute during the fatiguing intervention until failure. Failure was defined as the point at which participants were unable to maintain the 20% testing contraction after two consecutive attempts. During a 30-min recovery period at each time point a 1 Hz, 10 Hz and 50 Hz response was elicited followed by an MVC with ITT. Parameters were assessed immediately following the fatiguing task (at failure point, FP; and ~2–3 s following failure (R0), which accounted for the time needed to remove the blood pressure cuff) and at 2, 5, 10, 20, and 30 min of recovery.

Data and statistical analyses

Off-line quantification of measures consisted of time-to-peak torque (TPT), peak torque (PT), and half-relaxation time (HRT) for the 1-Hz stimulation, and PT and HRT for the 50- and 10-Hz stimulations. Peak torque (Nm) was measured in the MVCs. Voluntary activation was calculated using the interpolated twitch equation: $1-(\text{superimposed/potentiated twitch})\times 100$ (Belanger and McComas 1981; Gandevia 2001). The superimposed twitch refers to the doublet stimulation applied at the MVC peak, and the potentiated twitch refers to the doublet stimulation applied following the MVC at rest. Data are described as mean ± SD, while displayed as means ± SE in all figures. A two-way repeated measures ANOVA with a modified Bonferroni was performed to determine between-group differences of time and protocol. Cohen's d effect sizes (ES) were also calculated for main findings. A post hoc power analysis was completed for the 10-Hz tetanic stimulation and found that the sample size of 10 was sufficient. When only a main effect of time was observed, paired sample *t* tests were used in conjunction with a Dunnett's table test for multiple comparisons. Paired *t* tests were used to compare group differences in time-to-failure as well as MVC strength. All statistical analyses were performed using SPSS version 25. Statistical significance accepted at $\alpha < 0.05$.

Results

Voluntary characteristics

Time to failure point (FP) was 80% longer for FF compared with BFO (see Table 1). There was a main effect of time for MVC ($P < 0.01$) which decreased similarly in both protocols by ~60% at FP (see Fig. 1). Following 30 min of recovery MVC rebounded at similar rates to ~87% of baseline following both protocols, but was significantly lower than baseline. During both protocols voluntary activation decreased to ~89% of baseline at FP. There was a main effect of time ($P < 0.01$) and protocol ($P = 0.012$) and FF was ~5% less than BFO at the mid-point of task failure ($P = 0.018$, ES = 0.41) (see Fig. 2). The FF protocol remained significantly lower compared to BFO at recovery time points R–R10. At the completion of recovery (30 min) voluntary activation for both protocols was not different from baseline at ~94% and ~91%, respectively (see Fig. 2).

Twitch properties

For the twitch there was a significant decrease in peak torque following BFO compared to FF at R0 of ~75% and ~31%

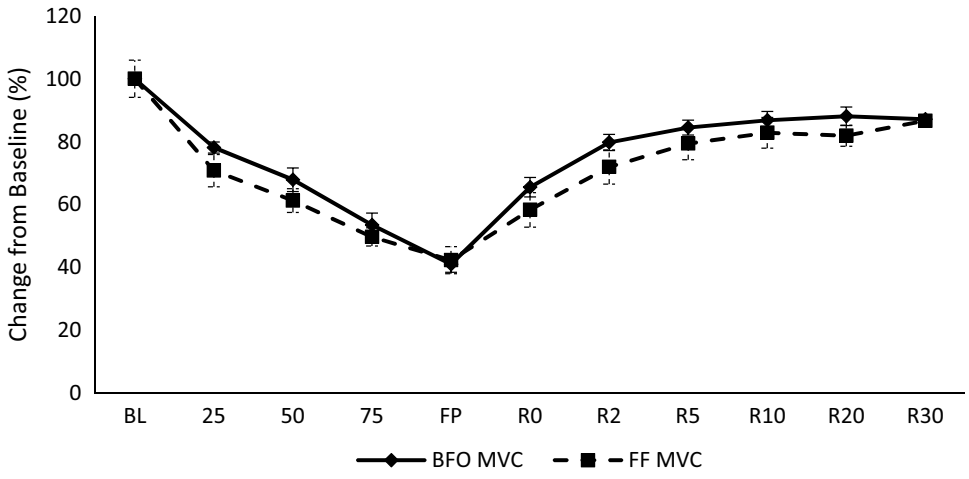

Fig. 1 Maximal voluntary contraction normalized to time displayed as 25%—failure point (FP) for both BFO and FF trials. Recovery time points from R0–R30. Values represented as percent change from baseline and displayed as means ± SE

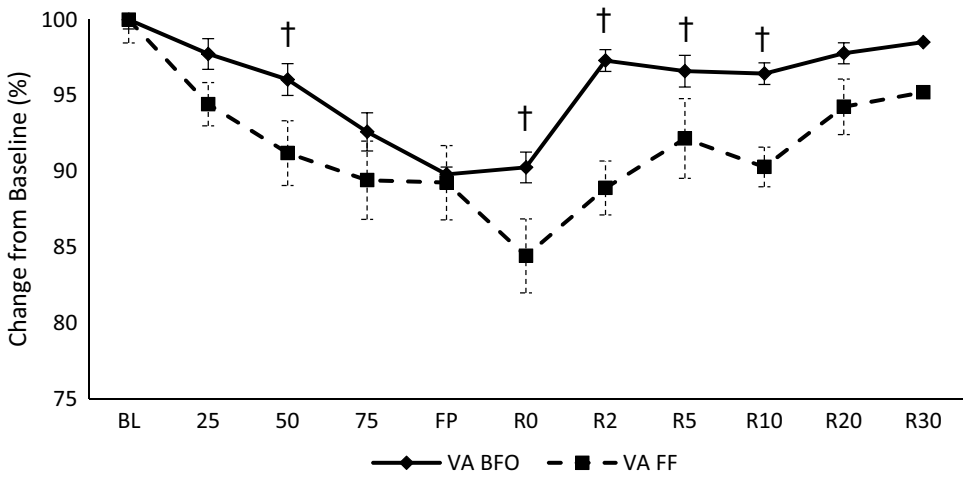

Fig. 2 Voluntary activation values normalized to time displayed as 25%—fail point (FP) for both BFO and FF trials. Recovery time points from R0–R30. Values represented as percent change from baseline and displayed as means ± SE. *Dagger* denotes significant difference between BFO and FF conditions

from baseline, respectively ($P < 0.01$, ES 0.88), as well as a main effect of time ($P < 0.01$). However, no statistical difference was observed between trials at R2 or for the remainder of the 30-min recovery period (see Fig. 3). The twitch (1 Hz) following the 30-min recovery period, remained depressed for both protocols at ~ 55% and ~ 64%, respectively, of baseline. TPT and HRT of the twitch (1 Hz) response were unchanged following the task and throughout recovery (data not displayed).

Tetanic properties

The 10-Hz tetanic stimulation had a greater decrease during BFO (~ 80% decrease from baseline) compared to FF (~ 45% from baseline) at R0 ($P < 0.01$, ES 0.56). Both the BFO and FF protocols had a main effect of time ($P = 0.01$) and 10 Hz remained depressed by ~ 40% from baseline following 30 min of recovery (see Fig. 4). The 10 Hz HRT was unchanged throughout both protocols and recovery with no difference between protocols (data not displayed). The 50-Hz peak torque was not significantly different between groups, but was significantly reduced from baseline following both protocols and throughout the duration of recovery (see Fig. 5a). The changes in 50-Hz torques were similar to changes observed for MVC torques. However, the 50-Hz HRT had a main effect of time ($P < 0.01$) and was significantly longer following the BFO at R0 and R2, by ~ 107% ($P < 0.01$, ES 0.95) and ~ 18% ($P < 0.01$, ES 0.74), respectively, compared to baseline. In contrast, 50-Hz HRT during FF remained unchanged from baseline at R0 (~ 96%) and R2 (~ 91%). Following 5 min of recovery 50-Hz HRT was similar to baseline for both protocols (see Fig. 5b). The 10 Hz to 50 Hz peak torque ratio between protocols was significantly different ($P < 0.01$, ES 0.51) immediately following both protocols (at R0), but was reduced more following BFO (to ~ 32% of baseline) compared to the FF (~ 79% of baseline) (see Fig. 6).

EMG

With BFO maximal EMG at the 25% normalized time-to-fatigue value was statistically greater than baseline,

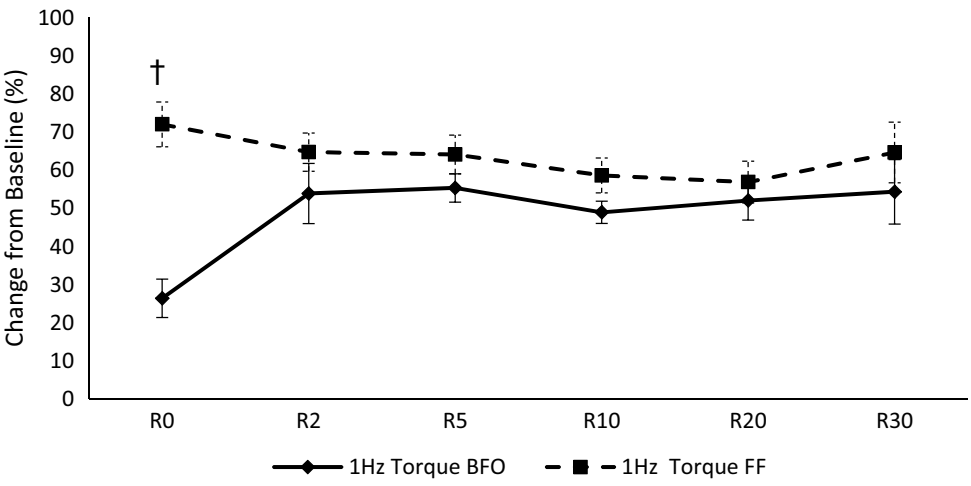

Fig. 3 Represents values of 1 Hz torque percent changes during R0–R30 minutes of recovery compared to baseline. Dagger denotes significant difference between BFO and FF conditions. Values displayed as mean ± SE

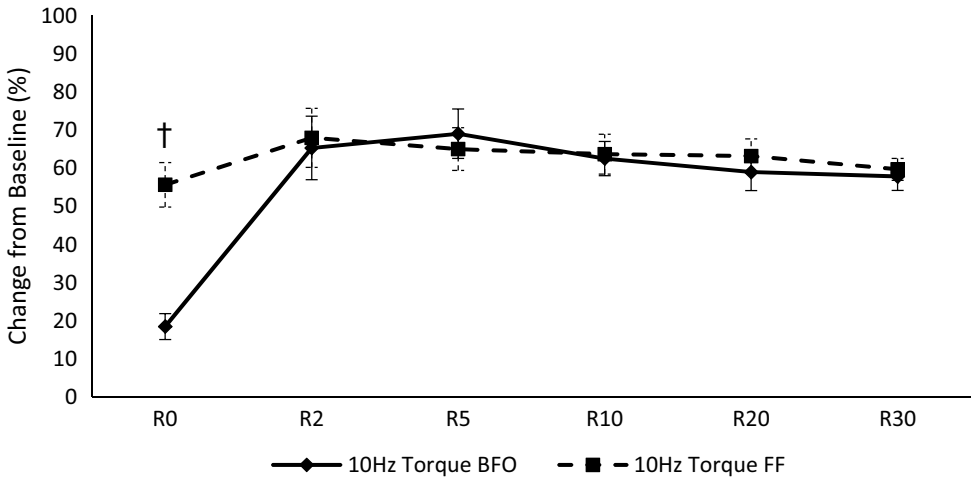

Fig. 4 Represents values of 10-Hz torque percent changes during R0–R30 minutes of recovery compared to baseline. *Dagger* denotes significant difference between BFO and FF conditions. Values displayed as mean ± SE

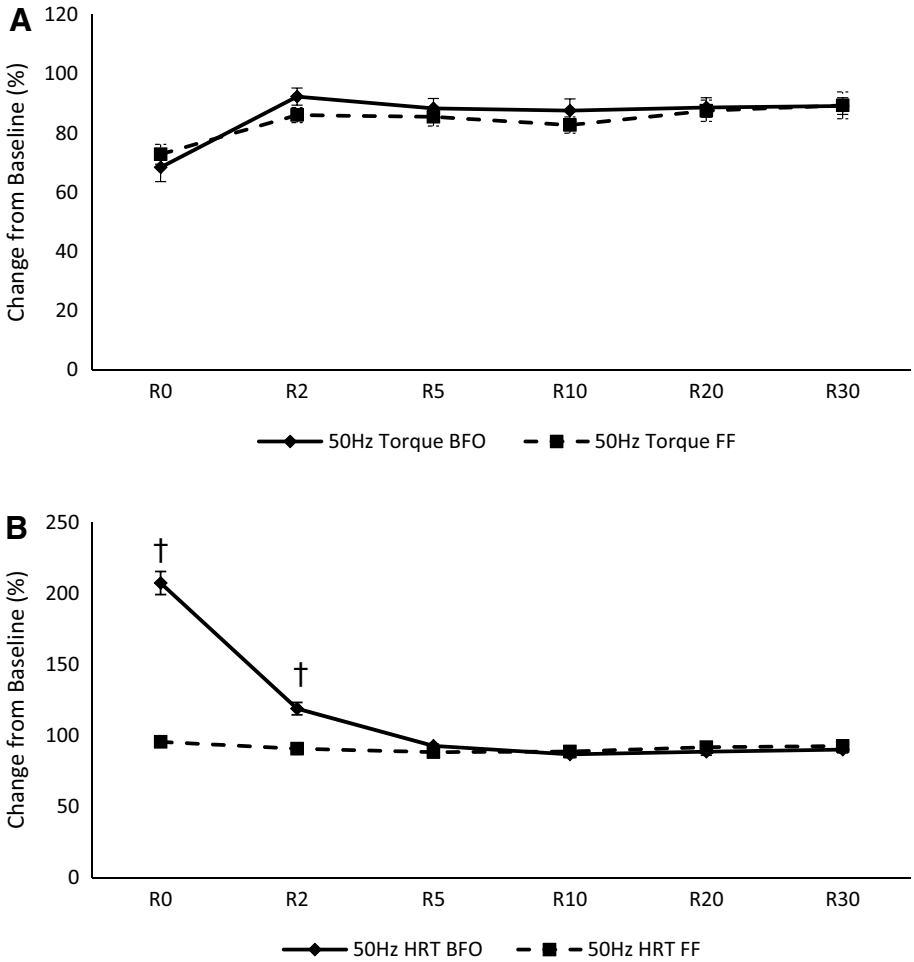

Fig. 5 **a** Represents values of 50-Hz torque percent changes during R0–R30 minutes of recovery compared to baseline. **b** Represents values of 50-Hz half-relaxation-time percent change during R0–R30 minutes of recovery compared to baseline. Dagger denotes significant difference between BFO and FF conditions. Values displayed as mean ± SE

exhibiting an increase in muscle activation before declining, while the FF protocol did not increase before declining. Maximal elbow flexor EMG following both protocols showed similar declines between protocols that were not significant at FP but were at R0 (11–30%). However, by R5 maximal EMG had recovered to baseline values in both protocols. Submaximal flexor EMG following each protocol showed a strong trend ($P = 0.06$) with BFO increasing from ~ 27% at 25% of normalized time to 65% at task failure, and FF increasing from ~ 25–40% at task failure (data not displayed). Submaximal EMG was only recorded

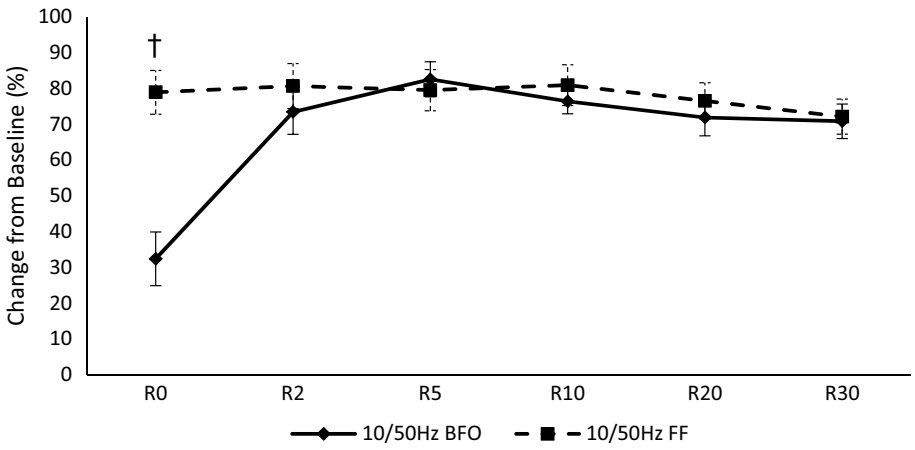

Fig. 6 Represents values of the 10/50 Hz torque ratio as percent changes during R0–R30 minutes of recovery compared to baseline. Dagger denotes significant difference between BFO and FF conditions. Values displayed as mean ± SE

during the fatiguing task and was not assessed during the recovery period.

Discussion

The current study assessed the effects of a single bout of blood flow occlusion during low-intensity elbow flexor contraction compared with the same protocol performed with an unrestricted blood flow. This BFO paradigm was used to explore relevant factors responsible for results found from established chronic training BFR protocols known to cause hypertrophy and strength improvements (Kumar et al. 2009; Burd et al. 2010). The main findings of the current study were: (1) time to task failure (FP) was shorter during blood flow occlusion compared with the unrestricted bout (234 ± 44 vs. 1026 ± 752 s, respectively); (2) MVC strength (voluntary fatigue) was reduced to a similar degree in both protocols with no differences between protocols in voluntary activation; (3) although the 50-Hz torque responses were similar to the changes in MVC, in contrast the intrinsic contractile torque measures at 1 Hz and 10 Hz were depressed to a greater amount following BFO than those during the FF protocol (see Figs. 3, 4), and 50-Hz HRT was longer following BFO. During the 30-min recovery period, MVC and VA recovered at a similar rate between protocols, but MVC remained significantly depressed. The more attenuated 1-Hz and 10-Hz torques following BFO compared to FF were no longer different between protocols by R2, but remained lower than baseline values. The 50-Hz torque responses were however recovered in both protocols by 30 min. These changes are indicative of a greater amount of peripheral fatigue induced during BFO than FF.

When normalized for time, the rates of decline and recovery in the MVC were similar between the two protocols as previously observed when comparing low-intensity bouts of contraction with and without BFR (Cook et al. 2013), however, voluntary activation was impaired more in the FF protocol in the current study. This was presumably due to the greater time in the FF protocol and therefore more contractile work required to reach task failure of ~ 60% of MVC. The response from the 50-Hz stimulation likely paralleled the MVC decline in both protocols during the fatiguing task, but was not assessed until R0 at which time the loss was significant and similar to the MVC loss. Recovery of voluntary activation was more impaired following the longer task (FF) for the first 10 min of recovery, which is likely due to greater time under tension (80% longer) during FF. These results are in agreement with Behm and St. Pierre (1997) who found similar results in VA following a long-term fatiguing task when compared with a short task. The accumulation of metabolic by-products would be countered upon release of the restriction (Cady et al. 1989), and a return to "normal" blood flow shown by the rapid recovery of half-relaxation-time observed within 5 min of rest following the fatiguing task. Therefore, the similar decrease in MVC between protocols indicates that blood flow occlusion caused the same amount of voluntary fatigue but in a significantly shorter amount of time than with FF (see Fig. 1). However, the MVC recovered at similar rates between the two protocols, but neither returned to baseline (~ 90% of baseline) following 30 min (see Fig. 1). The similar change in MVC and the 50-Hz responses have been observed previously in other fatiguing protocols (Edwards et al. 1977) and supports that voluntary activation was maximal and that these recover rapidly (within 30 min) as compared with force responses induced by lower frequencies of activation.

The depression of torques at lower frequencies of stimulation compared with high frequencies is reflected in the 10/50 Hz ratio (see Fig. 6). For the BFO protocol this ratio was depressed by ~ 79% from baseline compared with ~ 20% for FF at R0, and did not recover by the end of 30 min in either protocol. The inability of the muscle to produce force at low-frequencies (10 Hz), compared to the relative small reduction in forces elicited from high-frequencies (50 Hz) is indicative of fatigue-induced muscle impairment referred to

as low-frequency fatigue (Edwards et al. 1977; Jones et al. 1979). In this experiment immediately following the BFO protocol we observed greater reductions in both 1-Hz and 10-Hz torques compared with the FF protocol (see Figs. 3, 4). The reduction of the 10/50 Hz ratio is indicative of failure to produce force due to mechanisms beyond the point of the neuromuscular junction (Jones 1996). This low-frequency fatigue has been observed extensively and used as an indicator of peripheral fatigue following bouts of exercise or fatiguing contractions (Edwards et al. 1977; Hill et al. 2001; Verges et al. 2009). Low-frequency fatigue features a more severely affected loss of force that may take days to recover fully (Jones 1996). In our study, 2 min after the task, BFO had recovered to the same amount as FF (~60% of baseline), however, both protocols remained depressed from baseline for the remainder of recovery. This low-frequency fatigue may be indicative of structural damage to the muscle fiber or impairments in excitation–contraction coupling mechanisms caused by Ca^{2+} disruption following prolonged exercise (Jones 1981; Booth et al. 1997). Perhaps the greater amount of peripheral fatigue induced by BFO is indicative of an alteration in the preferred metabolic energy production of the type I oxidative fibers, caused by the ischemic environment. The early (0–2 mins) rapid recovery may be due to reperfusion and a hyperemic response following restriction providing a renewed supply of oxygen for type I fiber metabolism.

It has been reported that contractile output during low-intensity contractions (20% MVC) with complete occlusion of blood flow results in greater neural activation (EMG) to maintain task force (Moritani et al. 1992). Yasuda et al. (2009) also noted greater increases in EMG following BFO compared to moderate blood flow restriction, and attributed this to increased neural compression and blood flow impairment resulting in greater muscle activation and greater energetic demand at the same external load. Although the current study does not have a direct measure of arterial perfusion, we believe that the pressure applied (250 mmHg) likely created near complete occlusion, and therefore the observed increase in submaximal RMS EMG during each protocol indicates greater neural drive was required to maintain force during the fatiguing task. However, the BFO and FF protocols did not differ in the increased amount of submaximal RMS EMG at task failure likely because both protocols ended with each participants volitional failure point (FP), when task force (20% MVC) was no longer able to be maintained. However, importantly the BFO protocol was ~80% shorter in absolute time than the FF protocol. Therefore, the change in submaximal RMS EMG indicates a relatively greater modulation in muscle activation pattern and supports what has been shown in other similar studies (Moritani et al. 1992; Scott et al. 2015; Yasuda et al. 2008). Compared to the Karabulut et al. (2010) study that observed a greater reduction in maximal EMG amplitude (at pre- and post-MVCs) following blood flow restriction, the current study observed no difference in maximal RMS EMG values between protocols at task termination. The difference is likely due to the current study terminating the intervention at failure, compared with completion of a set number of repetitions in the former study.

Therefore we propose, that although both protocols reached a similar level of voluntary fatigue (MVC), low-intensity exercise in combination with blood flow occlusion causes a greater amount of peripheral fatigue than low-intensity exercise alone, and that BFO therefore recovers at an accelerated rate compared to FF despite remaining lower than baseline after 30 min. Based on other studies (Moritani et al. 1992; Scott et al. 2015; Yasuda et al. 2008), in an ischemic environment fatigue will be greater and result in an earlier or greater amount of activation of type II muscle fibers compared with an environment without BFO. Fatigue following BFO recovers at a faster rate when unrestricted blood flow is restored, causing a reperfusion of oxygenated blood flow. This is likely due to anaerobically (due to the ischemic environment caused by BFO) fatigued type I muscle fibers reverting to aerobic energy production, which allows voluntary activation of the muscle to be restored despite a greater amount of peripheral impairments.

This study provides important insights about how BFO may alter the neuromuscular system to enhance strength and hypertrophy gains with chronic training as used in previous studies (Ozaki et al. 2011; Abe et al. 2010; Manimmanakorn et al. 2013a). Although training regimes at near complete blood flow occlusion are not likely to be used chronically, the results of this study provide evidence that blood flow occlusion results in a greater level of peripheral fatigue, observed through intrinsic changes of the muscle properties at low-frequency.

Author contributions DB Copithorne: experimental design, data collection and analysis, all manuscript revisions and edits. CL Rice: all manuscript revisions and edits.

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Compliance with ethical standards

Conflict of interest DB Copithorne and CL Rice have no conflicts of interest that are directly relevant to the content of this article.

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