BLOOD FLOW RESTRICTION INCREASES METABOLIC STRESS BUT DECREASES MUSCLE ACTIVATION DURING HIGH-LOAD RESISTANCE EXERCISE

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ABSTRACT: *Introduction*: We investigated differences in metabolic stress (lactate) and muscle activation (electromyography; EMG) when high-load resistance exercise (HL) is compared with a condition in which blood flow restriction (BFR) is applied during the exercise or during the rest interval. *Methods*: Twelve participants performed HL with BFR during the intervals (BFR-I), during the set (BFR-S), and without BFR. Each condition consisted of 3 sets of 8 repetitions with knee extension at 70% of 1-repetition maximum. Lactate and root mean square (RMS) from the surface EMG of the vastus lateralis were calculated. *Results*: Lactate increased in all protocols but was higher with BFR-I than with BFR-S and HL. RMS decreased under all conditions, with a larger effect size in BFR-I (1.47) than in BFR-S (0.66) and HL (0.59). *Discussion*: BFR-I increases lactate, possibly as a result of reduced restoration of ATP. Muscle activation seems to be impacted by mechanical stress but may be reduced by metabolic stress.

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The American College of Sports Medicine recommends that participants perform resistance exercise with a load of at least 70% of their 1-repetition maximum (1RM) to stimulate substantial gains in muscle mass and strength.[1] However, over the past decade, several studies have found that low-load resistance exercise (e.g., 20%–40% of 1RM) in combination with blood flow restriction (BFR) results in muscle mass gain similar to that obtained with traditional high-load resistance exercise (HL; ~70% 1RM).[2–7]

The benefits of low-load resistance exercise combined with BFR are thought to be driven by acute metabolic stress, whereas HL is driven predominantly by mechanical factors.[8] It is conceivable that the combination of high levels of both mechanical and metabolic stress could work together to augment muscle growth. Therefore, it seems reasonable to speculate on the importance of keeping both mechanical and metabolic stress elevated.

Given that restricting blood flow increases metabolic stress during low-load resistance training[9,10] and that muscle contraction against high loads restricts blood flow while performing the set,[11–13] one possible strategy to increase metabolic stress is to restrict blood flow during rest intervals. However, maintenance of BFR after completion of exercise does not offer benefits to muscle growth,[14] suggesting that metabolites, *per se*, do not appear to have anabolic properties in the absence of mechanical stress.

Taken together, these results suggest that maintenance of BFR only during the rest intervals may offer a novel strategy for potentially augmenting the effects of high-load contractions in resistance exercise by increasing both metabolic stress and muscle activation. Therefore, this study compares metabolic stress (by blood lactate) and muscle activation (by electromyography–root mean square [EMG–RMS] amplitude) during HL under 3 conditions: blood flow restricted during the sets (BFR-S), blood flow restricted during the rest intervals (BFR-I), and without BFR.

MATERIALS AND METHODS

Participants. Twelve male participants (29 ± 2 years of age, 177 ± 4 cm, and 81 ± 9 kg) volunteered to participate in the study. Participants had not engaged in any kind of regular resistance training within the prior 6 months and did not participate in any parallel program of physical training during the study period. In addition, they were instructed not to perform any kind of physical activity 24 h prior to their visits to the laboratory. All participants were free from cardiovascular and/or neuromuscular disorders; were informed about the benefits, discomforts, and possible risks of the study; and provided written, informed consent before participation. The study was conducted according to the Declaration of Helsinki, and the research ethics

Abbreviations: 1RM, 1-repetition maximum; BFR, blood flow restriction; BFR-I, blood flow restriction applied during the intervals between sets; BFR-S, blood flow restriction applied during the set; EMG, electromyography; ES, effect size; HL, high-load resistance exercise; RMS, root mean square

Key words: blood flow restricted exercise; high load; metabolites; motor unit recruitment; strength training

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committee of the University of São Paulo approved the experimental protocol.

Experimental Procedure. Each participant visited the laboratory 6 times over the course of 4 weeks. During the first visit, lower body arterial occlusion pressure (determined with a portable vascular Doppler probe) and 1RM (tested with the leg extension machine) were assessed. During the second and third visits, participants repeated the 1RM test and were considered familiar with the test procedures when the interday strength value variation was $\leq 5\%$, having the highest value as 1RM. The 1RM values for all participants were obtained within 3 visits. In the last testing session, participants were familiarized with HL and BFR in the unilateral knee extension exercise. They completed 1 set of 8 repetitions at 70% 1RM of unilateral knee extension of the dominant leg, immediately followed by 60 s of BFR (80% arterial occlusion pressure). During the fourth through the sixth visits, the participants randomly performed 1 of 3 experimental protocols: BFR-I, in which BFR was applied during the rest intervals between sets and removed during the sets of muscle contractions; BFR-S, in which BFR was applied during the sets of muscle contractions and removed during the rest intervals between sets; and HL, during which the participants performed HL without BFR. Experimental sessions were conducted with an interval of at least 72 h between sessions, and measurements of EMG amplitude from the vastus lateralis muscle and blood lactate levels were measured for each experimental session.

Determination of BFR Pressure. Arterial occlusion was performed to determine the pressure used for the BFR protocols. After 10 min of supine rest, a vascular Doppler probe (DV-600; Marted, Ribeirão Preto, São Paulo, Brazil) was placed on the tibial artery to capture its auscultatory pulse. A nylon cuff (17.5 × 90 cm; JPJ, São Paulo, Brazil) was placed at the top of the thigh and inflated to the lowest point at which an auscultatory pulse was no longer detected. This value was defined as the *arterial occlusion pressure*. For the experimental protocols using BFR, a pressure equivalent to 80% of the resting arterial occlusion pressure was used, which has been previously shown to induce favorable increases in muscle size and strength.[3,15] Visual details of procedures used to determine the arterial occlusion pressure and the administration of the resistance exercise associated with BFR are provided elsewhere.[16]

Exercise Protocols. During the experimental sessions, participants performed unilateral knee extension exercise with 3 different protocols (BFR-I, BFR-S, and HL). Individuals performed 3 sets of 8 repetitions at 70% of their 1RM with 60-s rest periods between sets. The duration of each movement cycle was set at 2 s (1 s for the concentric phase and 1 s for the eccentric phase of the exercise) and controlled by a metronome. The positioning of the participant and the range of motion were the same as those used in the 1RM test. For the BFR conditions, a pressure cuff (17.5 × 90 cm) was placed at the top of the thigh and inflated to a value corresponding to 80% of the participant's resting arterial occlusion pressure.[3,15–17]

1RM Test. The test for 1RM in the knee extension exercise was conducted according to the guidelines of the American Society of Exercise Physiologists.[18] The exercise used was unilateral knee extension of the dominant leg, with dominance being defined as the leg that the participant used to kick a ball. The test was performed with a leg extension machine (SL 1030; Righetto, Campinas, Brazil). The participants performed a general 5-min warm up by running on a treadmill at 9 $km.h^{-1}$, followed by 3 min of light stretching of the lower limbs. They then performed a specific warm up composed of 1 set of 8 repetitions at approximately 50% 1RM and, after a 2-min rest, 1 set of 3 repetitions at approximately 70% 1RM. Both loads were estimated based on the participant's familiarization sessions. Three minutes after the specific warm up, the 1RM test was performed; each participant performed a complete cycle of the exercise with the greatest load that they could lift. A complete cycle consisted of the participant performing a full knee extension (0°) starting from the initial position of 90° of flexion and returning to the starting position while maintaining control during the entire movement range. The load was progressively increased from the last series of the specific warm up until the participant could fully perform the exercise 1 time. Rest periods of 3 min between attempts were used, with a maximum of 5 attempts. The greatest load lifted during the trials was considered to be the 1RM. Test–retest reliability of 1RM measurements was estimated by typical error (i.e., SD of the difference between the second and third test session / $\sqrt{2}$) and the typical error expressed as the coefficient of variation of the measurement (coefficient of variation percentage = [typical error of the difference between second and third test session means of the second and third test session] × 100).[19] The typical error and the coefficient of variation of 1RM assessments were 1.0 kg and 1.3%, respectively.

Blood Lactate Concentration. Measurements of blood lactate concentration were taken at rest (pre), at the end of the interval between the first and second sets; between the second and third sets; and at 0, 3, 5, and 10 min after the training protocol. After the area had been cleaned with alcohol, a small arterial blood sample (25 μl) was collected from the ear lobe and transferred to a heparinized capillary tube. Subsequently, the sample was transferred to an Eppendorf tube containing 50 μl of 1% sodium fluoride and stored in ice. The blood lactate concentration was measured by an electroenzymatic lactate analyzer (1500 Sport; Yellow Springs Instruments, Yellow Springs, OH) that had been previously calibrated with a known concentration of lactate of 5 $mmol{\cdot}L^{-1}$.

Surface EMG. The muscle electrical activity (surface EMG) was recorded with an 8-channel EMG system (EMG System, São José dos Campos, Brazil) with an acquisition frequency of 1,000 HZ and band-pass filter of 20–500 HZ. A single differential electrode was used in which two 36-mm-diameter electrodes (Ag-Ag/CI; Kendal, São Paulo, Brazil) were placed over the belly of the vastus lateralis muscle and aligned in parallel with the expected muscle fiber orientation.[20] Before electrode placement, the skin was shaved with a hand razor and carefully cleaned with ethanol. In addition, a ground electrode was placed on the medial portion of the patella.

During the first experimental session, the anchor point of each electrode was marked on the skin of each participant with semipermanent ink. This tag was used in the following sessions to maintain the same positioning of the electrodes in all experimental protocols. The EMG was recorded during each set of unilateral knee extensions.

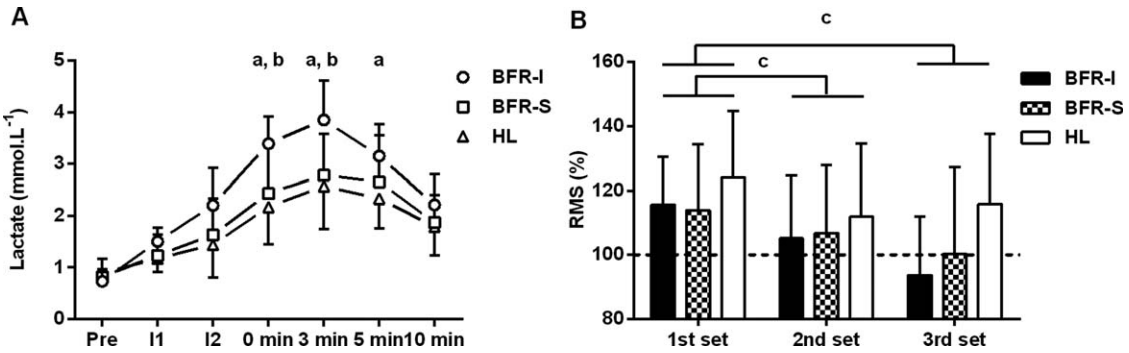

FIGURE 1. Lactate concentration (mmol.L^{-1}; **A**) pre; at the end of the interval between the first and second sets (I1); between the second and third sets (I2); and at 0, 3, 5, and 10 min after the protocols. Root mean square (RMS; %) of surface electromyography (**B**) during each experimental protocol: BFR-I, in which BFR was applied during the intervals between sets; BFR-S, in which BFR was applied during muscle contractions; and HL, traditional resistance training without BFR. $^{a}P < 0.05$, BFR-I compared with HL; $^{b}P < 0.05$, BFR-I compared with BFR-S; $^{c}P < 0.05$, compared with the first set.

The raw electromyographic signals were digitally filtered (fourth order Butterworth, band-pass 20–500 HZ) and converted to RMS for each concentric phase. The EMG signal obtained during the concentric muscle action of the last 3 repetitions of each unilateral knee extension set was averaged and normalized as percentage of the average of the first 3 repetitions. To identify the concentric phase of movement during exercise, an electrogoniometer (EMG System) was fixed on the side of the participant's knee, and the electrogoniometer signal was synchronized with the EMG. The axis of the electrogoniometer was aligned with the center of rotation of the knee, and its rods were fixed with Velcro strips along the longitudinal axis of the leg and thigh so that the 0° position of the goniometer corresponded to the full knee extension (0°).

Statistical Analysis. Data are presented as mean and SD. Data normality was tested by the Shapiro–Wilk test and visual inspection (box plots) to observe the presence of outliers; however, no outliers were observed. After data normality had been checked, a mixed model for repeated measures was applied, with experimental protocols (BFR-I, BFR-S, and HL) and times (pre; at the end of the interval between the first and second sets; between the second and third sets; at 0, 3, 5, and 10 min after the protocols for blood lactate concentration; and first, second, and third sets of the protocols for EMG) as fixed factors and participantss as a random factor. The number of repetitions was compared by one-way ANOVA. When a significant F value was found, the Tukey post hoc test was used for multiple comparisons. Additionally, Cohen's effect sizes (ES) were conducted to evaluate the magnitude of the change in EMG amplitude (first to third set changes) following experimental protocols with the criteria of $\leq$0.49, small; 0.50–0.79 medium, and $\geq$0.80, large.[21] $P \leq 0.05$ was considered significant. Data were analyzed in SAS 9.3 (SAS Institute, Cary, NC).

RESULTS

Number of Repetitions. All participants were able to perform the total number of repetitions in HL (24 ± 0 repetitions) but not in BFR-S (23.8 ± 0.4 repetitions) or in BFR-I (23.1 ± 1.0 repetitions). The number of repetitions performed in BFR-I was less than in HL ($P = 0.006$) and BFR-S ($P = 0.027$) protocols.

Blood Lactate. There was a significant protocol × time interaction with lactate ($P = 0.0089$). Compared with rest (pre), blood lactate increased at 0, 3, 5, and 10 min in all protocols. However, lactate was significantly higher for BFR-I at 0 and 3 min compared with both BFR-S and HL and was greater than HL only at 5 min ($P = 0.018$). There were no significant differences between BFR-S and HL protocols (Fig. 1A).

Muscle Activation. There was a decrease in EMG amplitude (RMS) in the second (8.5% ± 7.7%) and third (12.5% ± 11.0%) sets compared with the first set (main effect of time, $P < 0.05$), with no differences between the experimental protocols (Fig. 1B). BFR-I produced a large ES (1.47) in decreasing RMS from the first to third sets, whereas BFR-S and HL produced medium ESs of 0.66 and 0.59, respectively.

DISCUSSION

The main finding of this study is that HL with BFR applied during the rest interval results in the greatest amount of metabolic stress (accounted for by blood lactate) and a large ES on decreased muscle activation (represented by EMG amplitude).

Higher levels of blood lactate may partially explain the lower number of repetitions performed in BFR-I. Applying BFR during rest intervals may have compromised the restoration of blood flow and oxygen supply,[22] leading to a reduction in ATP resynthesis.[23] This is supported by the findings of studies that used low-load resistance exercise with BFR, demonstrating that phosphocreatine concentration decreases and intramuscular inorganic phosphate accumulates when BFR is maintained during rest intervals compared with the protocol that removed BFR during this period.[9,10] The results of the present study

indicate that restricting blood flow during the rest intervals of HL seems to induce similar results. On the other hand, when we applied BFR only during the sets, the high-load contraction itself probably restricted blood flow, reducing any beneficial effect of BFR. This would explain the similar increase in lactate between BFR-S and HL protocols. In addition, removing BFR interval restored blood flow, allowing the recovery of muscle substrates during the rest interval.[9] Given that that high mechanical stress was held constant but metabolic stress was augmented by applying BFR during rest interval, this novel strategy may offer a potential benefit for long term muscle hypertrophy.

Regarding muscle activation, previous studies have shown an increase when resistance exercise at low load (20%–30% 1RM) is performed in combination with BFR[6,11,24,25]; our results, however, show that BFR does not increase muscle activation when the load is already relatively high (>70% 1RM) and, in fact, may reduce it. Muscle activation increased similarly throughout the sets but was not different between conditions for any of the sets and was reduced during subsequent sets of exercise. The decrease in muscle activation during multiple sets of resistance exercise has also been observed in other studies. For instance, EMG amplitude decreased ~12% after performing 4 sets of dynamic knee extensions to failure.[26] Similarly, 20 sets of 1RM decreases muscle activation by 27.8% in men.[27] The mean differences between the first and the third set, although not significant, yielded ESs that were larger in BFR-I (1.47) than in both BFR-S (0.66) and HL (0.59), suggesting that BFR during the rest interval may have impacted muscular function more than traditional HL. Nevertheless, our results support the notion that HL induces acute fatigue not only in the contractile apparatus but also in the central nervous system. One possible explanation for the reduction in muscle activation in our study is that type III and type IV muscle afferents sense metabolite accumulation, particularly H^+, lactate, and ATP,[28] and also may inhibit α-motor neurons and/or the descending neural drive, attenuating motor unit recruitment and reducing muscle activation.[29,30] Thus, it is possible to suggest that, regardless of BFR, HL induced increased metabolite accumulation and that this may have decreased muscle activation across the sets. However, it is not possible to distinguish the contribution to the decrease in the discharge rate of the motor units due to actual reduction in neural drive from that because of afferent feedback from the muscle.

This study has limitations. First, the amount of BFR was determined from a single resting arterial occlusion measurement, and it is possible that the amount of restriction decreased during exercise.[31] Second, the lactate levels were estimated from ear lobe blood samples. Although this allowed us to quantify systemic lactate accumulation, it did not allow us to detect differences in lactate accumulation in or around the active muscle tissue. Finally, although we assessed surface EMG, this is not a direct measure of muscle activation and may be impacted by motor unit cycling.[32]

In conclusion, the results of this study suggest that HL in combination with BFR during rest intervals increases blood lactate concentration and decreases EMG amplitude. This indicates that muscle activation seems to be impacted by mechanical stress but may be reduced by metabolic stress.

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Ethical Publication Statement: We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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