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Acute cardiovascular responses to unilateral bicep curls with blood flow restriction

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ABSTRACT

A consensus on the acute cardiovascular responses to low intensity (LI) resistance exercise (RE) combined with blood flow restriction (BFR) has not yet been reached. This study was designed to compare acute cardiovascular responses to a single bout of LIRE, high intensity (HI) RE, and LIRE with BFR in physically active young males. Participants completed 3 RE sessions in random order, where each session consists of 4 sets of unilateral dumbbell bicep curls. Cardiovascular hemodynamics were measured at baseline and right after each set of RE. Aortic augmentation index (AIx) was significantly higher after set 2,3,4 of RE in LI + BFR session compared to LI session ($P < 0.05$). Brachial systolic blood pressure (SBP), heart rate (HR), brachial rate pressure product (RPP), and central RPP responses did not differ between LI and LI + BFR sessions ($P > 0.05$). HI session had a higher central SBP, brachial RPP, central RPP, and aortic AIx compared to LI session after each set of RE ($P < 0.05$), but not brachial SBP ($P > 0.05$). Taken together, this study showed that LIRE combined with BFR acutely augmented aortic stiffness, as also observed in HI session, but myocardial oxygen consumption was only higher in HI session when compared to LI session. Thus, although BFR did not exaggerate cardiovascular responses nor cause extra myocardial oxygen consumption, it should be prescribed with caution when control of acute aortic stiffening is necessary during RE.

1. Introduction

Resistance training has well documented health benefits on the muscle gains and maintenance of muscle strength and mass.[1] The American College of Sports Medicine (ACSM) recommends performing resistance training at intensities of at least 70% of one repetition maximum (1RM) to promote significant gains of muscle strength and mass.[2] However, resistance exercise (RE) performed at such high intensity (HI) are often not advised for patients recovering from the orthopedic injuries because of the elevated risk of re-injury.[3] To address this specific concern, others have utilized an innovative training method called blood flow restriction (BFR) training.[4]

BFR training involves placing a pneumatic cuff, similar to a blood pressure (BP) cuff, as a tourniquet around a working limb proximally to maintain arterial inflow to the muscles while preventing venous return during exercise.[4]When combined with RE, BFR training has been shown to promote considerable gains in muscle strength and mass only using low intensity (LI) as low as 20–30% 1RM,[5–7] which is hypothesized to be caused by the restriction of blood flow to the working muscles during LIRE that mimics the local muscular physiological environment induced by HIRE (e.g., high level of metabolic stress), thus, it is translated to have comparative hypertrophic benefits of HIRE while performing at lower intensity.[6]

Arterial stiffness has been shown to acutely increase during RE,[8] which can last for approximately 1 h during the recovery following exercise,[9] and the magnitude of increase vary by the intensity of exercise.[10] However, very few studies on how RE with BFR alters this acute arterial stiffening response to RE showed conflicting findings, where acute LIRE with BFR demonstrated both positive and negative effects on arterial stiffness in healthy young people.[11] In addition, original data indicated possible safety concerns over BFR that restriction of blood flow to the working muscles may exaggerate cardiovascular stress during exercise.[12–14] Moreover, a

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consensus on the cardiovascular changes promoted by LIRE with BFR when compared to LIRE or HIRE without BFR has not yet been reached, where divergent results were found when comparing the LIRE with or without BFR versus HIRE on BP, heart rate (HR) and rate pressure produce (RPP).[15,16] Lastly, there is paucity of studies that investigated the acute responses of LIRE with BFR on central BP, which has been shown to be a better indicator for the pathogenesis of cardiovascular disease (CVD) than peripheral BP.[17–19] When central BP is combined with measurements of arterial stiffness, we can generate a full picture of how cardiovascular hemodynamics respond to BFR with RE.[20–22]

Thus, the purpose of this study was to compare the acute cardiovascular responses to a single bout of LIRE, HIRE, and LIRE with BFR in physically active young males. It was hypothesized that HIRE and LIRE with BFR would lead to an increase in both peripheral and central cardiovascular responses as well as aortic stiffness when compared to LIRE alone in physically active young males.

2. Method

2.1. Participant

This study (#3761920) was approved by the Institutional Review Board at Springfield College, MA, USA. Seventeen physically active young males aged between the ages of 21–35 were recruited for this study. Fifteen was the sample size required to detect a medium effect size with 80% power at a significance level of 0.05, based on a power analysis using G*Power version 3.1.[23] Inclusionary criteria included: no prior history of a deep vein thrombosis, the absence of any diagnosed CVD, varicose vein, hypertension, diabetes, lymphedema, blood anticoagulant medications in use, or any other contraindication to exercise as outlined in the ACSM's guidelines for exercise testing and prescription.[24]

2.2. Procedure

Prior to the first visit, all the participants signed an informed consent form and completed a medical history questionnaire. Participants who consented and met the inclusionary criteria went through a familiarization session and three exercise sessions in random order: LIRE session, HIRE session, and LIRE + BFR session, with one week between each visit. All the visits took place in the Human Performance Laboratory of Springfield College at the same time of the day for each participant. Participants were required to maintain their normal exercise routines and diet throughout the entire course of participation. Self-reported daily sodium consumption three days prior to each visit was recorded for each participant. No caffeine, performance enhancing supplements, or alcohol was allowed to be consumed 24 h prior to each visit.

Familiarization session. In the first visit, the participants went through a familiarization session. The researchers recorded the age of each participant, measured the height of each participant via stadiometer (Seca GmbH, Hamburg, Germany), and measured the other anthropometric variables including weight, body mass index, and body composition of each participant via the Tanita digital scale (Tanita Corporation of America Inc., Chicago, IL, USA). Limb occlusion pressure (LOP) of the dominant arm was tested under resting condition via the Edan SD3 vascular doppler (Edan USA, San Diego, CA, USA) and the Smart Tools BFR cuff (Smart Tools Plus, Strongsville, OH, USA). Next, the researchers tested the 1RM of the unilateral dumbbell bicep curl on the dominant arm of the participant. To ensure a proper execution of unilateral dumbbell bicep curls, the participants needed to stabilize their upper torso by holding onto a stationary structure with the nondominant arm while being in a sitting position. In this study, the participants were also required to execute the unilateral dumbbell bicep curls at a constant cadence (2 s for the concentric phase and 2 s for the eccentric phase) over the whole range of motion. The timing was paced by a metronome, and the participants were instructed to avoid the Valsava maneuver while executing unilateral dumbbell bicep curls because of its augmenting effect on BP.[25]

Exercise sessions. In the second, third, and fourth visits, participants went through three exercise sessions in random order: LI, HI, and LI + BFR session. At the beginning of each session, participants were required to rest in the sitting position for at least 10 min. Then participants were fitted with a Polar H10 HR sensor (Polar Electro Inc., Bethpage, NY, USA), which measures the HR continuously during the whole session. Baseline HR was recorded; brachial SBP, brachial DBP, central SBP, central DBP, and aortic augmentation index (AIx) at baseline were also measured with the SphygmoCor device (AtCor Medical, Sydney, Australia) on the non-dominant arm. Aortic AIx is an indirect measurement of aortic stiffness,[26] which is an independent predictor of future CVD.[27] For the RE, participants first warmed up with one set of five repetitions of unilateral dumbbell bicep curls at 60% of 1RM and then performed three different protocols of RE respectively in three exercise sessions. For the LI session, participants completed four sets of 15 repetitions of unilateral dumbbell bicep curls at 20% of 1RM. For the HI session, participants completed four sets of eight repetitions of unilateral dumbbell bicep curls at 80% of 1RM. For the LI + BFR session, participants completed the same repetitions, sets, and intensity of unilateral dumbbell bicep curls as the LI session while wearing a BFR cuff around the upper portion of the dominant arm. There was 180 s of rest between each set of RE for all three exercise sessions. The protocols of RE with BFR were modified from prior research, in which LI + BFR session was shown to promote a greater hypotensive effect compared to LI session.[28] For all three exercise sessions, HR, brachial SBP, brachial DBP, central SBP, central DBP, and aortic AIx were measured immediately after each set of RE on the non-dominant arm. RPP, which is an indirect measurement of myocardial oxygen consumption, was calculated by multiplying HR (bpm) with brachial SBP (mmHg) or central SBP (mmHg) respectively at each data collection time point.[29]

Determination of blood flow restriction. In this study, the Smart Tools BFR cuff (width 10.2 cm) was placed around the deltoid tuberosity of the dominant arm to restrict blood flow immediately before the start of each set of RE. The cuff pressure used to restrict blood flow during the exercise session was 80% of LOP in a resting condition for each participant.[7] The cuff pressure was maintained throughout the RE bout except for 180 s of deflation during the rest time between each set.

2.3. Statistical analysis

A within-subject design was used. Statistical tests were performed with GraphPad Prism (GraphPad Software Inc, San Diego, CA). Two-way repeated measures ANOVA (RE session X RE set) were used to evaluate differences between groups, and Tukey's post hoc test were used to further identify values that were significantly different. Statistical significance was set at $P < 0.05$ for all analyses.

3. Results

3.1. Characteristics of participant

Weight, height, BMI, body composition, age, LOP, 80% of LOP as occlusion pressure in BFR, and daily sodium consumption of the participants before each session are presented in Table 1. There was no significant difference in daily sodium consumption before each visit over four lab visits ($P > 0.05$).

Table 1
Anthropometric measurement of participants was conducted in familiarization (F) session. Average daily sodium consumption was recorded by self-reported food log over three days prior to low intensity (LI), high intensity (HI), and LI with blood flow restriction (LI + BRF) sessions respectively. One-way repeated measures ANOVA was used to examine whether sodium consumption before each visit changes for 17 physically active young males over four lab visits (F, LI, HI, LI + BFR). There was no significant difference in daily sodium consumption over four lab visits (P > 0.05). n = 17.

Characteristics	Minimum	Maximum	Mean	SD
Age (years)	21	35	25	3.8
Height (cm)	168	186	177	5.9
Weight (kg)	57	93	80	9.7
BMI	18.1	30.4	25.5	3.0
Body Fat (%)	5.4	25.6	17.3	5.4
LOP (mmHg)	110	160	130	16.0
80% LOP (mmHg)	88	128	104	12.8
Average Daily Sodium Consumption (mg /day)				
F session	196	3768	2005	1187.7
LI session	241	5000	2281	1381.6
HI session	225	4303	2127	1108.0
LI + BFR session	196	5000	2194	1268.4

3.2. Brachial and central blood pressure

At baseline (i.e., set 0), brachial SBP, brachial DBP, central SBP, and central DBP were similar between sessions (P > 0.05; Fig. 1). During RE, HI session had higher brachial SBP than baseline after each set of RE (P < 0.05; Fig. 1A), but LI and LI + BFR sessions were similar to baseline (P > 0.05). There was no significant difference in brachial DBP between sessions after each set of RE (P > 0.05). Unlike brachial SBP, all three sessions had higher central SBP than baseline during RE (P > 0.05; Fig. 1B). HI session had higher central SBP than LI session after each set of RE and had higher central SBP than LI + BFR session after set 3,4 of RE, respectively (P > 0.05). There was a trend for higher central SBP in LI + BFR session compared to LI session after set 2 of RE (P = 0.085). For brachial DBP, LI session had lower brachial DBP than baseline after set 1,2 of RE (P < 0.05; Fig. 1C). HI and LI + BFR sessions had no difference in brachial DBP than baseline after each set of RE (P > 0.05). There was no significant difference in brachial DBP between LI and LI + BFR sessions after each set of RE (P > 0.05). The LI session had lower central DBP than baseline after set 1,2,3 of RE (P < 0.05; Fig. 1D). HI and LI + BFR sessions had no significant difference in central DBP than baseline after each set of RE (P > 0.05). There was no significant difference in central DBP between LI and LI + BFR sessions after each set of RE (P > 0.05).

3.3. Heart rate and rate pressure product

At baseline (i.e., set 0), HR, brachial RPP, central RPP were similar between sessions (P > 0.05; Fig. 2). During RE, HI, LI, and LI + BFR sessions all had higher HR than baseline after each set of RE (P < 0.05; Fig. 2A). HI session had higher HR than LI and LI + BFR sessions after each set of RE (P < 0.05). There was no significant difference in HR between LI and LI + BFR sessions after each set of RE (P > 0.05). During RE, HI, LI, and LI + BFR sessions all had higher brachial RPP than baseline after each set of RE (P < 0.05; Fig. 2B). HI session had higher brachial RPP than LI and LI + BFR sessions after each set of RE (P < 0.05). There was no significant difference in brachial RPP between LI and LI + BFR sessions after each set of RE (P > 0.05). During RE, HI, LI, and LI + BFR sessions all had higher central RPP than baseline after each set of RE (P < 0.05; Fig. 2C). HI session had higher central RPP than LI and LI + BFR sessions after each set of RE (P < 0.05). There was no significant difference in central RPP between LI and LI + BFR sessions after each set of RE (P > 0.05).

3.4. Aortic stiffness

At baseline (i.e., set 0), aortic AIx were similar between sessions (P > 0.05; Fig. 3). During RE, HI, LI, and LI + BFR sessions all had higher aortic AIx than baseline after each set of RE (P < 0.05). Interestingly, LI session had lower aortic AIx after set 4 compared to

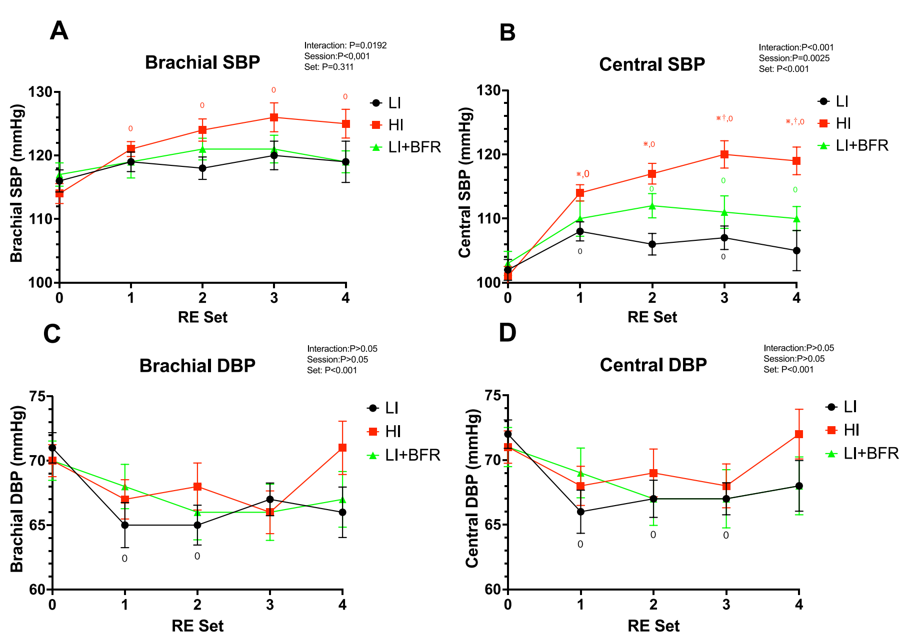

Fig. 1. Group-by-set and session comparisons in low intensity (LI) session, high intensity (HI) session, and LI with blood flow restriction (LI + BFR) session. Data were analyzed using a two-way repeated measures ANOVA with Tukey's post hoc test to identify differences in brachial systolic blood pressure (SBP; **A**), central SBP (**B**), brachial diastolic blood pressure (DBP; **C**), and central DBP (**D**) at each time point. *P < 0.05 vs. LI. †P < 0.05 vs. LI + BRF. ‡P < 0.05 vs. HI. Data are presented as mean ± SEM. n = 17.

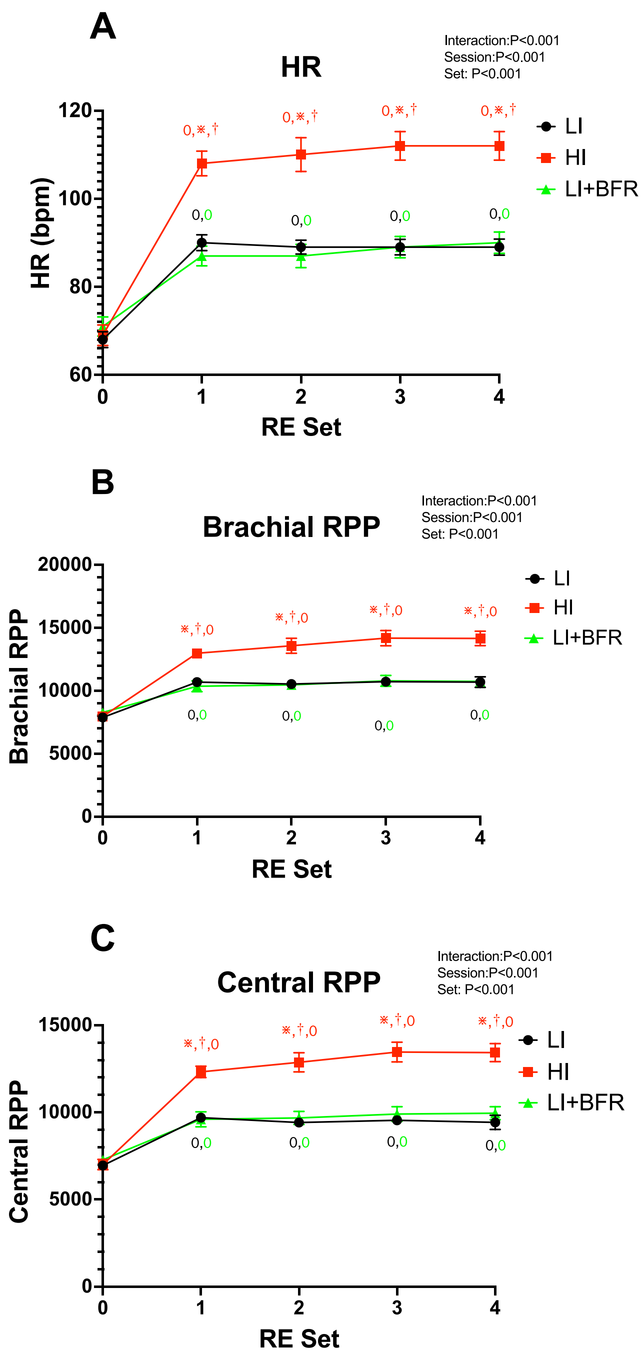

Fig. 2. Group-by-set and session comparisons in low intensity (LI) session, high intensity (HI) session, and LI with blood flow restriction (LI + BFR) session. Data were analyzed using a two-way repeated measures ANOVA with Tukey's post hoc test to identify differences in heart rate (HR; **A**), brachial rate pressure product (RPP; **B**), and central RPP (**C**). *P < 0.05 vs. LI. †P < 0.05 vs. LI + BRF. ‡P < 0.05 vs. HI. Data are presented as mean ± SEM. n = 17.

after set 1 of RE (P < 0.05). HI session had higher aortic AIx than LI and LI + BFR sessions after each set of RE (P < 0.05). LI + BFR session had higher aortic AIx than LI session after set 2,3,4 of RE (P < 0.05).

4. Discussion

In the present study, we investigated the acute cardiovascular responses to a single bout of LIRE, HIRE, and LIRE with BFR in

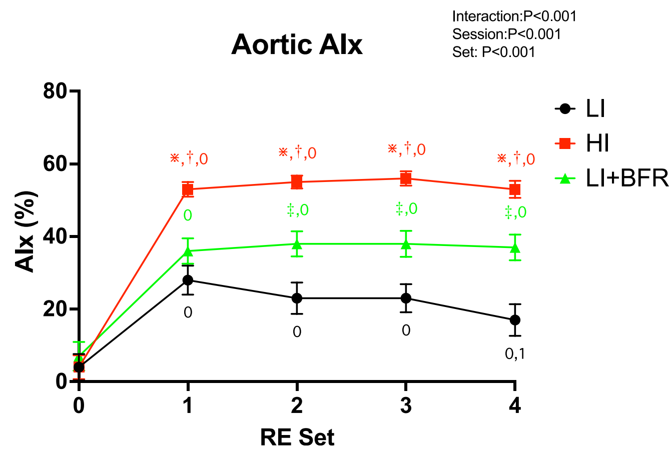

Fig. 3. Group-by-set and session comparisons in low intensity (LI) session, high intensity (HI) session, and LI with blood flow restriction (LI + BFR) session. Data were analyzed using a two-way repeated measures ANOVA with Tukey's post hoc test to identify differences in aortuc augmentation index (AIx). *P < 0.05 vs. LI. †P < 0.05 vs. LI + BRF. ‡P < 0.05 vs. HI. Data are presented as mean ± SEM. n = 17.

physically active young males. In support of our hypothesis, during HIRE, there were higher HR, brachial RPP, and central RPP, which indicate higher myocardial consumption, compared to LIRE with or without BFR. Interestingly, central SBP, but not brachial SBP increased during HIRE compared to LIRE with and without BFR. Unlike HIRE, LIRE with BFR had similar HR, brachial SBP, central SBP, brachial RPP, and central RPP compared to LIRE, which indicates that myocardial oxygen consumption was not exaggerated by BFR during LIRE. However, similar to HIRE, LIRE with BFR induced a higher aortic AIx compared to LIRE, indicating a higher aortic stiffness. Taken together, these findings provide unique evidence that not only higher intensity but also other stressors like BFR can cause an acute arterial stiffening during RE. Although BFR does not exaggerate cardiovascular responses nor cause extra myocardial oxygen consumption during LIRE, it should be prescribed with caution when control of acute aortic stiffening is necessary during RE.

4.1. HI but not LI + BFR exaggerates central SBP

In this study, brachial SBP increased from baseline to during RE in HI session but did not change in LI and LI + BFR sessions. This finding is inconsistent with similar study which showed that brachial SBP increased from baseline to during unilateral bicep curls in LI (20% 1RM), HI (80% 1RM), and LI + BFR sessions.[30] The inconsistency could be explained by the less rest time (30s) allowed between each set of RE in that study compared to the present study (180s). A rapid reduction in BP has been seen in the recovery period after acute HIRE, which was assumed to be caused by reactive hyperemia. Reactive hyperemia is characterized by the sudden increase in the perfusion of vasodilated muscle which was occluded during contraction of RE.[31] When compared between different sessions in this study, there was no difference in brachial SBP at each measurement time points during RE. This finding is inconsistent with another similar study which showed that HI session (80% 1RM) and LIRE (20% 1RM) with BFR had a higher brachial SBP during bilateral leg press exercises compared to LIRE.[32] The inconsistency could be explained by the use of continuous BFR during RE in that study, thus, time under tension was longer during the session with BFR, compared to the use of an intermittent BFR protocol in the current study. It has been shown that exercise volume (i.e., time under tension), but not exercise intensity, contributed more to the increased BP during acute RE.[33]

Central BP is the BP in the ascending aorta. Despite having a relatively constant DBP, SBP may increase by up to 40 mmHg greater in the brachial artery than in the aorta, which is due to the increased arterial stiffness as blood travels from central to peripheral arteries.[19] In this study, we found that, unlike brachial SBP, LI and LI + BFR session had an increased central SBP during RE compared to the baseline. Furthermore, HI session had a higher central SBP than LI and LI + BFR sessions during RE. Very few studies investigated the central BP response to LIRE combined with BFR and reported inconsistent findings. There was a study in which central SBP increased from baseline to during 20% 1RM knee extension exercise with BFR,[34] but another study using moderate intensity (60% 1RM) unilateral handgrip exercise with BFR showed no increase in central SBP compared to without BFR.[35] A plausible explanation for the inconsistency is the use of different levels of LOP during BFR (i.e., occlude the arterial blood inflow to the working muscles to different extents) in these studies. BFR during RE may cause a greater venous pooling of metabolites locally in the working muscle and, consequently, activates the metabolic-based exercise pressor reflex which exaggerate cardiovascular responses.[13] Indeed, it has been well documented that accumulation of metabolites like protons and lactate in the working muscles during LIRE with BFR mimics the physiological environment of working muscles during HIRE and, therefore,stimulate muscle growth.[36] Nonetheless, the underlying mechanism of how BFR facilitates the exercise pressor reflex is unclear. Furthermore, the LI + BFR session had a trend for higher central SBP compared to LI session which did not happen in brachial SBP. A potential explanation for this discrepancy is that changes in central hemodynamics (e.g., central SBP) is more cardiac driven, rather than controlled by peripheral vascular resistance, thus, the activation of exercise pressor reflex will have a larger effect on central SBP compared to brachial SBP.[35] In this study, only LI session had a decrease in brachial and central DBP from baseline to during RE exercise. It could be explained by a preserved arterial endothelial function (e.g., vasodilation) with LI session compared to HI and LI + BFR sessions which cannot be determined in this study.[37]

4.2. BFR did not cause increased HR or myocardial oxygen consumption

In this study, all three RE sessions had an increase in HR from baseline to during RE.

The HI session had the highest HR during RE, and the LI session and LI + BFR sessions were not different from each other. This finding is inconsistent with similar studies that showed that the HI session (80% 1RM) and LI + BFR session (20% 1RM) had a higher HR during unilateral bicep curls compared to the LI session,[30] and the LI + BFR session (30% 1RM) had higher HR during single-arm bicep curls compared to the LI session.[38] These discrepancies can be attributed to the different width of BFR cuffs used in these studies and it has been shown that a wide BFR cuff caused a greater elevation in HR compared to a narrow BFR cuff during RE.[34] In this study, brachial and central RPP showed identical findings to HR in pairwise comparisons between RE sessions during RE despites the discrepancy in the responses of brachial and central SBP during RE, indicating that HR is the dominating factor over SBP in terms of the myocardial oxygen consumption. Therefore, the finding of RPP response in this study is also inconsistent to the previously mentioned studies which had different HR responses compared to this study.[30,38] More importantly, this study showed that LIRE with BFR did not cause additional myocardial oxygen consumption, which was indicated by the unchanged brachial and central RPP compared to LIRE without BFR in physically active young males, rendering this method safe and feasible for this population.[15]

4.3. BFR and HIRE induced comparative aortic stiffening

In this study, all three RE sessions had an increase in aortic AIx from baseline to during RE, indicating an acute increase in aortic stiffness. The increase in aortic stiffness was maintained at the similar level in HI and LI + BFR sessions but dropped down slowly in the LI session. The HI session had the highest aortic AIx during RE, and the LI + BFR session had a higher aortic AIx compared to LI session, indicating that both higher intensity and BFR had an augmenting effect on the acute response of arterial stiffness to RE. This finding is inconsistent with a similar study that showed there was no difference in aortic AIx during handgrip exercise between three RE protocols: LIRE (40% 1RM), moderate intensity (60% 1RM) with BFR or without BFR.[35] In addition, one study showed that LI (40% 1RM) knee extension and flexion exercise with BFR decreased AIx,[39] but another study showed LI (30% 1RM) bench press exercise with BFR increased AIx.[40] The discrepancy is likely due to the inaccurate methods of prescribing occlusion pressure during BFR in these two studies, where one study used rating of perceived exertion and the other used same occlusion pressure for all participants, instead of using individual LOP for each participants.

Arterial stiffening is constantly observed with the progression of age and CVD.[41] Arterial stiffness has been shown to acutely increase during conventional RE,[8] which can last for approximately 1 h during the recovery following RE.[9] In a long term, RE training increases arterial stiffness in the resting condition.[42–44] Paradoxically, RE-trained individuals have a decreased CVD mortality and morbidity compared to physically inactive controls.[45] It is plausible that there may be some unique vasoprotective adaptations on vasculature to the repetitive acute arterial stiffening after a long-term RE training. Indeed, studies have demonstrated that arteries taken from RE-trained individuals are protected from high pressure-induced endothelial dysfunction.[46] It is unclear why the arterial stiffness acutely increases during RE or whether it is pathological, to determine that is beyond the scope of this study. Nevertheless, this study provides a new angle to investigate the paradox between RE and arterial stiffness by showing that not only higher intensity but also restriction of blood flow to the working muscle can cause an acute increase in arterial stiffness during RE.

There are several strengths of this study that can be emphasized. Very few studies have examined the acute response of central BP to LIRE with BFR compared to both LIRE and HIRE. This study was conducted with a more reliable BFR device (i.e., the Smart Tools BFR cuff is listed by the U.S. Food and Drug Administration) compared to most of the similar studies where unstandardized cuffs were widely used.[15] To reach a consensus on this topic, more controlled and randomized studies with a standard operating procedure and reliable BFR device focusing on various populations are strongly recommended to examine both peripheral and central cardiovascular responses to acute RE with BFR. Studies on optimizing the intensities of RE with BFR are also warranted to develop the individualized exercise prescription for RE with BFR in other populations beyond healthy adults.

4.4. Limitations

This study is not without limitations. This study only measured the cardiovascular responses during RE with BFR, but not during the recovery following RE. Future studies are warranted to continuously monitor the changes in cardiovascular hemodynamics to RE with BFR from the baseline, throughout the duration of exercise, and till it recovers back to baseline, as it may provide unique insight into the patterns how cardiovascular system responds and adapts to RE with BFR. Another limitation is that we only chose one RE movement (i.e., unilateral bicep curl) to compare and potentially reach a consensus with previous studies on this topic. However, it limits its translation to conventional whole-body RE movement. Thus, future studies are warranted to determine the cardiovascular responses to whole-body RE movement when combined with BFR as it may better simulate the traditional RE training.

5. Conclusions

This study provides unique evidence that not only higher intensity but also other stressors like BFR can cause acute increase in arterial stiffness, thus, it should be prescribed with caution when control of acute aortic stiffening is necessary during RE. In summary, this study found that HIRE induced greater cardiovascular responses, myocardial oxygen consumption, and aortic stiffness compared to LIRE with or without BFR during a single bout of unilateral dumbbell bicep curls. BFR did not exaggerate cardiovascular responses nor cause extra myocardial oxygen consumption. However, our study suggests that BFR can cause acute aortic stiffening during a single bout of unilateral dumbbell bicep curls. With respect to the myocardial oxygen consumption, BFR remains safe and feasible for physically active young males when combined with LIRE.

Authorship statement

Category 1.

Conception and design of study: Xiangyu Zheng, Samuel AE Headley, Stephen A Maris, Daniel M Smith; acquisition of data: Xiangyu Zheng; analysis and/or interpretation of data: Xiangyu Zheng, Daniel M Smith.

Category 2.

Drafting the manuscript: Xiangyu Zheng; revising the manuscript critically for important intellectual content: Samuel AE Headley, Stephen A Maris, Daniel M Smith.

Category 3.

Approval of the version of the manuscript to be published (the names of all authors must be listed): Xiangyu Zheng, Stephen A Maris, Samuel AE Headley, Daniel M Smith.

Declaration of competing interest

The authors declare no conflicts of interest.

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References

1. Krzysztofik M, Wilk M, Wojdala G, Golas A. Maximizing muscle hypertrophy: a systematic review of advanced resistance training techniques and methods. *Int J Environ Res Publ Health*. 2019;16.
2. American College of Sports M. American College of Sports Medicine position stand. Progression models in resistance training for healthy adults. *Med Sci Sports Exerc*. 2009;41:687–708.
3. Lambert BS, Hedt C, Moreno M, Harris JD, McCulloch P. Blood flow restriction therapy for stimulating skeletal muscle growth: practical considerations for maximizing recovery in clinical rehabilitation settings. *Tech Orthop*. 2018;33: 89–97.
4. Patterson SD, Hughes L, Head P, Warmington S, Brandner C. Blood flow restriction training: a novel approach to augment clinical rehabilitation: how to do it. *Br J Sports Med*. 2017;51:1648–1649.
5. Slysz J, Stultz J, Burr JF. The efficacy of blood flow restricted exercise: a systematic review & meta-analysis. *J Sci Med Sport*. 2016;19:669–675.
6. Hughes L, Paton B, Rosenblatt B, Gissane C, Patterson SD. Blood flow restriction training in clinical musculoskeletal rehabilitation: a systematic review and meta-analysis. *Br J Sports Med*. 2017;51:1003–1011.
7. Laurentino GC, Ugrinowitsch C, Roschel H, et al. Strength training with blood flow restriction diminishes myostatin gene expression. *Med Sci Sports Exerc*. 2012;44:406–412.
8. Sardeli AV, Gaspari AF, Chacon-Mikahil MP. Acute, short-, and long-term effects of different types of exercise in central arterial stiffness: a systematic review and meta-analysis. *J Sports Med Phys Fit*. 2018;58:923–932.
9. DeVan AE, Anton MM, Cook JN, Neidre DB, Cortez-Cooper MY, Tanaka H. Acute effects of resistance exercise on arterial compliance. *J Appl Physiol*. 2005;98: 2287–2291.
10. Tabaie EA, Reddy AJ, Mamidi D, et al. The relationship between arterial stiffness and resistance training. *Cureus*. 2021;13, e20213.
11. Amorim S, Rolnick N, Schoenfeld BJ, Aagaard P. Low-intensity resistance exercise with blood flow restriction and arterial stiffness in humans: a systematic review. *Scand J Med Sci Sports*. 2021;31:498–509.
12. Loenneke JP, Wilson JM, Wilson GJ, Pujol TJ, Bemben MG. Potential safety issues with blood flow restriction training. *Scand J Med Sci Sports*. 2011;21:510–518.
13. Spranger MD, Krishnan AC, Levy PD, O'Leary DS, Smith SA. Blood flow restriction training and the exercise pressor reflex: a call for concern. *Am J Physiol Heart Circ Physiol*. 2015;309:H1440–H1452.
14. Cristina-Oliveira M, Meireles K, Spranger MD, O'Leary DS, Roschel H, Pecanha T. Clinical safety of blood flow-restricted training? A comprehensive review of altered muscle metaboreflex in cardiovascular disease during ischemic exercise. *Am J Physiol Heart Circ Physiol*. 2020;318:H90–H109.
15. Neto GR, Novaes JS, Dias I, Brown A, Vianna J, Cirilo-Sousa MS. Effects of resistance training with blood flow restriction on haemodynamics: a systematic review. *Clin Physiol Funct Imag*. 2017;37:567–574.
16. Domingos E, Polito MD. Blood pressure response between resistance exercise with and without blood flow restriction: a systematic review and meta-analysis. *Life Sci*. 2018;209:122–131.
17. Agabiti-Rosei E, Mancia G, O'Rourke MF, et al. Central blood pressure measurements and antihypertensive therapy: a consensus document. *Hypertension*. 2007;50:154–160.
18. Terentes-Printzios D, Gardikioti V, Vlachopoulos C. Central over peripheral blood pressure: an emerging issue in hypertension research. *Heart Lung Circ*. 2021;30:1667–1674.
19. McEniery CM, Cockcroft JR, Roman MJ, Franklin SS, Wilkinson IB. Central blood pressure: current evidence and clinical importance. *Eur Heart J*. 2014;35: 1719–1725.
20. Suleman R, Padwal R, Hamilton P, Senthilselvan A, Alagiakrishnan K. Association between central blood pressure, arterial stiffness, and mild cognitive impairment. *Clin Hypertens*. 2017;23:2.
21. Kohara K. Central blood pressure, arterial stiffness and the heart in hypertensive patients. *Hypertens Res*. 2009;32:1056–1058.
22. Oliveras A, Segura J, Suarez C, et al. Modification over time of pulse wave velocity parallel to changes in aortic BP, as well as in 24-h ambulatory brachial BP. *J Hum Hypertens*. 2016;30:186–190.
23. Faul F, Erdfelder E, Buchner A, Lang AG. Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. *Behav Res Methods*. 2009;41:1149–1160.
24. Thompson PD, Arena R, Riebe D, Pescatello LS, American College of Sports M. ACSM's new preparticipation health screening recommendations from ACSM's guidelines for exercise testing and prescription, ninth edition. *Curr Sports Med Rep*. 2013;12:215–217.
25. Hackett DA, Chow CM. The Valsalva maneuver: its effect on intra-abdominal pressure and safety issues during resistance exercise. *J Strength Condit Res*. 2013;27:2338–2345.
26. Catalano M, Scandale G, Carzaniga G, et al. Aortic augmentation index in patients with peripheral arterial disease. *J Clin Hypertens*. 2014;16:782–787.
27. Vlachopoulos C, Aznaouridis K, O'Rourke MF, Safar ME, Baou K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with central haemodynamics: a systematic review and meta-analysis. *Eur Heart J*. 2010;31: 1865–1871.
28. Neto GR, Sousa MS, Costa PB, Salles BF, Novaes GS, Novaes JS. Hypotensive effects of resistance exercises with blood flow restriction. *J Strength Condit Res*. 2015;29:1064–1070.
29. White WB. Heart rate and the rate-pressure product as determinants of cardiovascular risk in patients with hypertension. *Am J Hypertens*. 1999;12: 50S–55S.
30. Brandner CR, Kidgell DJ, Warmington SA. Unilateral bicep curl hemodynamics: low-pressure continuous vs high-pressure intermittent blood flow restriction. *Scand J Med Sci Sports*. 2015;25:770–777.
31. MacDougall JD, Tuxen D, Sale DG, Moroz JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol*. 1985;58:785–790.
32. Poton R, Polito MD. Hemodynamic responses during lower-limb resistance

exercise with blood flow restriction in healthy subjects. *J Sports Med Phys Fit*. 2015;55:1571–1577.
33. Gjovaag TF, Mirtaheri P, Simon K, et al. Hemodynamic responses to resistance exercise in patients with coronary artery disease. *Med Sci Sports Exerc*. 2016;48: 581–588.
34. Rossow LM, Fahs CA, Loenneke JP, et al. Cardiovascular and perceptual responses to blood-flow-restricted resistance exercise with differing restrictive cuffs. *Clin Physiol Funct Imag*. 2012;32:331–337.
35. Credeur DP, Jones R, Stanford D, Stoner L, McCoy S, Jessee M. Central cardiovascular hemodynamic response to unilateral handgrip exercise with blood flow restriction. *Eur J Appl Physiol*. 2019;119:2255–2263.
36. Pearson SJ, Hussain SR. A review on the mechanisms of blood-flow restriction resistance training-induced muscle hypertrophy. *Sports Med*. 2015;45: 187–200.
37. Horiuchi M, Okita K. Blood flow restricted exercise and vascular function. *Int J Vasc Med*. 2012;2012, 543218.
38. Vieira PJ, Chiappa GR, Umpierre D, Stein R, Ribeiro JP. Hemodynamic responses to resistance exercise with restricted blood flow in young and older men. *J Strength Condit Res*. 2013;27:2288–2294.
39. Figueroa A, Vicil F. Post-exercise aortic hemodynamic responses to low-intensity resistance exercise with and without vascular occlusion. *Scand J Med Sci Sports*. 2011;21:431–436.
40. Tai YL, Marshall EM, Glasgow A, Parks JC, Sensibello L, Kingsley JD. Pulse wave reflection responses to bench press with and without practical blood flow restriction. *Appl Physiol Nutr Metabol*. 2019;44:341–347.
41. Donato AJ, Machin DR, Lesniewski LA. Mechanisms of dysfunction in the aging vasculature and role in age-related disease. *Circ Res*. 2018;123:825–848.
42. Collier SR, Kanaley JA, Carhart Jr R, et al. Effect of 4 weeks of aerobic or resistance exercise training on arterial stiffness, blood flow and blood pressure in pre- and stage-1 hypertensives. *J Hum Hypertens*. 2008;22:678–686.
43. Miyachi M, Donato AJ, Yamamoto K, et al. Greater age-related reductions in central arterial compliance in resistance-trained men. *Hypertension*. 2003;41: 130–135.
44. Miyachi M, Kawano H, Sugawara J, et al. Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. *Circulation*. 2004;110:2858–2863.
45. Liu Y, Lee DC, Li Y, et al. Associations of resistance exercise with cardiovascular disease morbidity and mortality. *Med Sci Sports Exerc*. 2019;51:499–508.
46. Durand MJ, Dharmashankar K, Bian JT, et al. Acute exertion elicits a H2O2-dependent vasodilator mechanism in the microvasculature of exercise-trained but not sedentary adults. *Hypertension*. 2015;65:140–145.