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Effects of Leg Blood Flow Restriction during Walking on Cardiovascular Function

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Abstract

Introduction—Exercise with limb blood flow restriction (BFR) is a very popular exercise modality in Japan and is spreading widely to the rest of the world. The underlying principle of this training modality is that under the conditions of restricted blood flow, even low-intensity exercise can provide significant muscle strength and hypertrophy. One concern, however, is that BFR during exercise may place unnecessary burden on those with compromised cardiac function.

Methods—We determined the impact of leg BFR during walking on cardiovascular function in 17 young (26 ± 1 years) healthy volunteers. Each subject underwent five bouts of 2-minute treadmill walking at 2 miles/hour with 1-min interval either with or without tourniquet cuffs inflated on both thighs.

Results—Heart rate increased more during the BFR session, whereas stroke volume decreased greater during the BFR session. Blood pressure increased significantly and substantially during the BFR session. Consequently, an increase in double product, an index of myocardial oxygen demand, was >3-fold higher in the BFR condition. Systemic arterial compliance evaluated by stroke volume/pulse pressure ratio significantly increased during the control session by 14% but reduced during the BFR condition by 19%. Popliteal artery flow-mediated vasodilation decreased significantly after the exercise with BFR but not after the control session.

Conclusions—Even at low intensity, the aerobic exercise with BFR requires a greater cardiac work and decreases endothelial function. Limb BFR during exercise may need to be more cautiously prescribed to those with compromised cardiac conditions.

Keywords

blood pressure; rate-pressure product; tourniquet; endothelial function

Introduction

Exercise and fitness industries are a rapidly evolving field with a number of new exercise modalities emerging frequently. One such exercise method involves short-term low-intensity exercise with tourniquet ischemia or vascular occlusion (1,2,9–11,13,17,21,22). This training is a very popular and widely-practiced mode of exercise in Japan and is rapidly gaining popularity in other countries (11). The primary attraction of this novel exercise

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Conflict of interest

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technique is that with limb blood flow restriction (BFR), even low-intensity exercise training can provide a significant gain of muscle mass and strength (13,17,22). The application of a low intensity exercise to increase muscle mass and function has considerable clinical implications as high intensity exercise is often difficult to achieve in the elderly as well as in patients with musculoskeletal or neurological disorders.

Recently, the concept of the exercise with BFR has been incorporated into a more common activity of daily exercise (ie, walking). Previous studies in humans (2) and animals (1) demonstrated that the combination of leg muscle blood flow restriction with slow walk training induces significant muscle hypertrophy and strength gains despite the fact that walking training was performed at very low intensity. Despite mounting evidence for the efficacy of low-intensity BFR exercise, the relative safety of BFR exercise has not been established. It is possible that ischemia induced by BFR may increase blood pressure and myocardial oxygen demand by augmenting regional and systemic vascular resistance as well as through the accumulation of metabolites and the subsequent stimulation of chemoreflex (21,22). Additionally, limb BFR for an extended period of time could induce the ischemia-reperfusion injury upon the release of BFR (3,4). One of the hallmark features of the ischemia-reperfusion injury is endothelial damage (16). Reperfusion after a period of BFR causes local injury secondary to an acute inflammatory response. Neutrophils and platelets are activated as a result of the insult, producing reactive oxygen species and adhesion molecule, which impair endothelial function (16). These hemodynamic changes induced by the BFR exercise may not be favorable or even detrimental, particularly in those with compromised cardiovascular conditions. However, these issues have not been addressed.

Accordingly, the purpose of this study is to determine effects of the combination of leg blood flow restriction with slow walking exercise on cardiovascular function in healthy young people. We hypothesize that a low-intensity aerobic exercise with BFR induces exaggerated elevations in blood pressure and myocardial oxygen demand as well as post-exercise decreases in endothelial function.

Methods

Subjects

A total of 17 apparently healthy sedentary or recreationally active adults (11 males and 6 females) between the ages of 19 and 34 years (mean age: 26 ± 1 years) were recruited. Participants were healthy, normotensive, non-obese (BMI: 23.0 ± 0.5 kg/m²), non-medicated, non-smokers, and free of overt cardiovascular disease (as assessed by medical health questionnaire). Prior to the exercise protocol, any subjects who had a ankle-brachial pressure index of less than 0.9, an indicative of peripheral arterial disease, were excluded (6). All subjects gave their written informed consent to participate. The study was reviewed and approved by the Institutional Review Board at The University of Texas at Austin.

Experimental protocol

The study protocol consisted of measurements of hemodynamic responses during the walking test and endothelial function before and after the walking test. Each subject walked with or without blood pressure cuffs inflated on both legs (BFR and control condition, respectively) on 2 different days. The 2 testing days were separated by ~7 days. The order of these experimental conditions was randomized.

All measurements were performed in a quiet, temperature-controlled room (24–26°C) after at least 4 hour fasting and an abstinence of caffeine. After 20 minutes of supine rest, each subject underwent baseline measurements of heart rate, blood pressure, popliteal arterial structure, and endothelial function (via flow-mediated vasodilation: FMD) followed by the

walking test. Immediately after the walking test, each subject had 20 minutes of supine rest. To determine the hypothesized ischemia-reperfusion injury induced by BFR, FMD test was repeated 20 minutes after the end of each exercise.

Heart rate, blood pressure, and ankle-brachial pressure index at rest

Heart rate and blood pressure were measured in supine position with vascular testing device equipped with electrocardiograms and four extremities-oscillometric pressure sensor cuffs (VP-2000, Colin Medical, San Antonio, TX). Ankle-brachial pressure index was calculated as a ratio of ankle systolic blood pressure and brachial systolic blood pressure.

Popliteal arterial structure and flow-mediated vasodilation

Subjects rested in the prone position with the right knee at an angle of $\sim 20^\circ$. Popliteal artery properties were evaluated with a duplex ultrasound machine equipped with a high-resolution multi-frequency linear-array transducer (iE33, Philips, Bothell, USA). Using a customized transducer holding device, a linear array transducer was positioned on the popliteal fossa. The rapid inflation/deflation pneumatic cuff (Hokanson) was placed on immediately proximal to the transducer. In order to measure baseline diameter and blood flow, the longitudinal two-dimensional and Doppler ultrasound images of the popliteal artery were acquired consecutively. After the baseline measurement, pneumatic cuff was rapidly inflated at 300 mmHg for 5 minutes, as previously described (14). To quantify shear rate during the reperfusion, blood flow velocity was acquired from 10 second before the cuff-deflation until 20 seconds after the deflation. The longitudinal two-dimensional images were taken from 30 to 90 second after the cuff-deflation. Arterial diameter and blood flow velocity were analyzed using commercially available software (Brachial Analyzer, Medical Imaging Applications, Coralville, Iowa) by a blinded investigator. The average of at least 10 end-diastolic artery diameters before blood flow occlusion was used for the baseline diameter and the average of three peak end-diastolic diameters during reperfusion phase was used for maximum diameter. FMD was expressed as the percent change in popliteal artery diameters using the equation: $(\text{maximum diameter} - \text{baseline diameter}) / \text{baseline diameter} \times 100$. The area under the curve of shear rate (4 times velocity divided by baseline diameter) for the first 20 seconds of reperfusion phase was computed to normalize FMD. The popliteal artery, rather than the brachial artery, was chosen in order to assess the impact of blood occlusion placed on the lower limbs on endothelial function. The data on the FMD were available on a subset of 12 subjects who completed the tests.

Blood pressure and systemic hemodynamics during exercise

Throughout the walking exercise protocols, beat-to-beat arterial blood pressure waveforms were continuously recorded via photoplethysmography (Portapres Model 2, TNO TPD Biomedical Instruments, The Netherlands) placed on the middle finger of the left hand of each subject. Subjects were instructed to keep the hand at heart level during the entire testing session. Brachial blood pressure was estimated from the finger blood pressure waveform with a transfer function (BeatScope 1.0 software; TNO TPD; Biomedical Instrumentation; Amsterdam, The Netherlands), and then calibrated with successive-recorded brachial blood pressure by the oscillometric device (HEM-907XL, Omron Health Care Co., Kyoto, Japan). Heart rate, stroke volume, and cardiac output were calculated from the finger blood pressure waveform using the validated Model flow method incorporating age, sex, height, and weight (BeatScope 1.0 software, TNO TPD Biomedical Instrumentation, Amsterdam, The Netherlands) (18,19,24). Total peripheral resistance was determined as the quotient of mean arterial pressure/cardiac output. Double product, an index of myocardial oxygen demand, was calculated by systolic blood pressure $\times$ heart rate, and stroke volume/pulse pressure ratio (SV/PP) was used as an index of systemic arterial

compliance. Stroke volume, cardiac output, and total peripheral resistance were reported as relative changes (%) from the baseline.

Exercise test

Exercise test consisted in five bouts of 2-minute treadmill walking at 2 mile/hour with 1-min rest between each bout as previously described by Abe et al. (2). This particular protocol practiced for 3 weeks was demonstrated to be highly effective in inducing significant increases in muscle mass and maximal strength (2). During the BFR protocol, leg blood flow was restricted by applying a blood pressure cuff to the most proximal portion of both legs (2). In order to familiarize the subject with the cuff pressure, the cuff was first inflated to 120 mmHg for 30 seconds. Pressure was released for 10 seconds, and then the cuffs were re-inflated. The pressure was increased by 20 mmHg and held for 30 seconds, and then released for 10 seconds between occlusive stimulations. This process was repeated until a final occlusion pressure of 160 mmHg was reached. Baseline data (standing on treadmill before exercise) were recorded for 3 minutes after reaching occlusion pressure of 160 mmHg. Immediately following the termination of exercise, the blood pressure cuffs were deflated. In the control session, subjects performed the same exercise as mentioned above, without the application of a pressure cuff.

Statistical Analyses

Analyses of variance with repeated measures were used to compare hemodynamic responses during exercise. In the case of a significant F value, a post hoc test using the Newman-Keuls test identified significant differences among mean values. Pearson correlational analyses were performed to determine relations of interests. All data are presented as mean $\pm$ SEM. Differences were considered significant at a level of $P<0.05$.

Results

There were no baseline (standing on treadmill before exercise) differences in heart rate and blood pressure between both sessions. In the control session, systolic blood pressure increased significantly through the exercise bouts whereas no significant changes were observed in mean and diastolic blood pressure (Figure 1). In the BFR session, systolic, mean, and diastolic blood pressure all rose significantly during the exercise, and the magnitude of increase was significantly and markedly greater than those observed in the control session.

Increases in mean arterial pressure during the BFR exercise session were due to sustained elevations (or more precisely, less reductions) in total peripheral resistance as cardiac output increased similarly in both sessions (Figure 2). Increases in mean arterial pressure were associated significantly with the corresponding increases in total peripheral resistance ($r=0.74$). Although there were no differences in cardiac output responses between the two sessions, stroke volume responses were lower and heart rate responses were greater in the BFR session than in the control session.

Double product increased mildly ($\sim 30\%$) in the control session from baseline whereas it demonstrated a gradual marked increase through the entire BFR exercise session reaching $\sim 90\%$ increase at the end of the exercise session (Figure 3). The ratio of SV/PP, a surrogate marker of systemic arterial compliance, increased slightly during the control session but decreased during the BFR session. The changes in SV/PP ratio were associated with the corresponding changes in mean arterial pressure ($r=-0.85$) and total peripheral resistance ($r=-0.77$).

As shown in Table 1, heart rate, blood pressure, and popliteal artery blood flow returned to pre-exercise levels at the time of the FMD measurements (20 minutes after exercise). There were no differences in post-ischemic hyperemic responses, as assessed by the area under the curve of shear rate, before and after each exercise session. FMD did not change in the control session but significantly reduced in the BFR session. (Figure 4) The area under the curve of shear rate during the reperfusion phase were comparable between the 2 sessions. As such, results of FMD were not different even when FMD was normalized for the shear rate.

Discussion

The primary findings of this study are as follows. First, the exercise-induced elevation in blood pressure was significantly higher in the BFR session compared with the control session. This was primarily due to the sustained elevations in total peripheral resistance exerted by the blood flow restriction. Second, although cardiac output increased similarly between the two sessions, stroke volume increased less and heart rate increased more during exercise with BFR. As a result, the increase in double product, an index of myocardial oxygen demand, was significantly greater in the BFR session than in the control session. Similarly, systemic arterial compliance, as estimated by the stroke volume/pulse pressure ratio, increased significantly during the control session but reduced during the BFR condition, suggesting that left ventricular afterload is augmented by the BFR during the exercise. Third, FMD of the popliteal artery decreased significantly after the exercise with BFR, suggesting that endothelial function is diminished presumably through the ischemia-reperfusion injury. These series of hemodynamic events may not be favorable and might provide a circulatory challenge, particularly for those with compromised cardiovascular function.

Although there are numerous studies demonstrating the muscular benefits of BFR with exercise, studies investigating the cardiovascular effects of blood flow constraint are sparse (20). This is the first study to measure systemic hemodynamics and blood pressure responses during a low-intensity aerobic exercise combined with BFR. Cardiac output increased similarly in both exercise sessions, suggesting that metabolic/perfusion demand was homologous. However, there were marked increases in heart rate and blood pressure when lower intensity exercise was combined with the BFR. Consequently, the elevation of double product (i.e., myocardial oxygen demand) was greater during low-intensity exercise with the leg tourniquet. The exercise-induced increase in stroke volume is blunted probably due to the leg tourniquet-induced decrease in venous return and/or an elevation in regional vascular resistance and the concomitant increase in afterload. In order to maintain cardiac output, heart rate was increased to offset the drop in stroke volume. Alternatively, plasma concentrations of lactic acid are known to be higher during low intensity exercise with BFR compared with normal condition (21,22), and lactic acid is a potent stimulator of chemoreceptors. Activation of these pressor reflexes stimulates sympathetic outflow, subsequently increasing heart rate and cardiac contractility (5). These results obtained during an aerobic exercise combined with BFR were consistent with a previous study indicating that stroke volume at supine rest was reduced with BFR due to the decreased venous return (7).

In the present study, blood flow restriction resulted in a 3-fold elevation in myocardial work, as assessed by the double product, although the absolute values of double product attained during exercise were rather mild (23). However, the use of young healthy subjects in the present study may have underestimated the overall magnitude of the myocardial oxygen demand. In contrast to normotensive individuals, hypertensive patients demonstrate exaggerated blood pressure responses to dynamic exercise (12). Additionally, aging is associated with the augmented vasoconstrictor responsiveness of exercising muscle to

sympathetic stimulation (8,15). Therefore, limb BFR during exercise may need to be more cautiously prescribed, particularly to populations at high risk of developing cardiac events during exercise (8,15). Interestingly, an increase in rate-pressure product was also accompanied by an increase in diastolic blood pressure in the present study. Because higher diastolic blood pressure favors better coronary perfusion, exercise with BFR may cause less angina despite higher rate-pressure product.

The rationale for measuring FMD in the present study is associated with the ischemia-reperfusion injury, which refers to myocardial, vascular, or electrophysiological dysfunction that is induced by the restoration of blood flow to previously ischemic tissue (3,4). Myocardial infarction is the most deleterious form of ischemia-reperfusion injury. Although prolonged blockage of blood flow and the resultant ischemia itself could cause decreases in cellular function and cellular apoptosis, more organ damage can be done during reperfusion or reintroduction of blood flow to the previously ischemic tissues. Reperfusion of ischemic tissue is associated with microvascular dysfunction that is often manifested as impaired endothelium-dependent dilation. The mechanism of the ischemia-reperfusion injury is complex, but is associated with endothelial dysfunction (16). We hypothesized that similar form of injury may occur to vascular tissues on lower limbs when the blood pressure cuffs are released after BFR exercise. A salient observation is a significant decrease in popliteal artery FMD after a lower-intensity aerobic exercise combined with leg BFR. It should be emphasized that there were no significant differences in popliteal artery blood flow at rest and hyperemic responses before and after each exercise intervention. Hence, results of FMD were the same even when FMD was normalized for shear rate. Collectively, these results suggest that endothelial function is reduced after BFR walking and that the reduction in endothelial function observed after the combination of the BFR with slow walking may be attributed to the ischemic-reperfusion injury on vascular endothelium.

There are some limitations in the present study that should be emphasized. First, we used young healthy subjects for the present investigation. Hemodynamic responses to aerobic exercise with limb BFR may be different in older or diseased populations (8,15). It would have been more clinically relevant if the experiments had been conducted in these populations. However, we were not able to obtain an IRB approval on applying the BFR to patients with cardiovascular disease because of a perceived concern for deep vein thrombosis and pulmonary embolism. Further studies in these populations are warranted. Second, there is a disconnection regarding the potential short-term risks versus long-term benefits of blood flow restricted exercise. Currently, it is not known whether the long-term benefits of the BFR exercise (i.e., gains of muscle mass and strength) could outweigh the acute cardiovascular risks. This question needs to be explored in the future. Third, we indirectly estimated stroke volume and cardiac output from the finger blood pressure waveform. Although the model flow methods for estimating change in cardiac output have been validated during various conditions including cycling exercise (18,19,24), it may not provide absolute levels of cardiac output. Therefore, only the relative changes from the baseline were reported in the present study.

In conclusion, leg blood flow restriction during low-intensity aerobic exercise are associated with exaggerated increases in blood pressure and cardiac work, reductions in systemic arterial compliance, as well as post-exercise decreases in lower limb endothelial function. These hemodynamic changes may place unnecessary circulatory burden, particularly for those populations with compromised cardiac function. As such, exercise with blood flow restriction should be prescribed carefully.

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References

1. Abe T, Kearns CF, Manso Filho HC, Sato Y, McKeever KH. Muscle, tendon, and somatotropin responses to the restriction of muscle blood flow induced by KAATSU-walk training. *Equine Vet J Suppl* 2006;345–348. [PubMed: 17402445]
2. Abe T, Kearns CF, Sato Y. Muscle size and strength are increased following walk training with restricted venous blood flow from the leg muscle, Kaatsu-walk training. *J Appl Physiol* 2006;100:1460–1466. [PubMed: 16339340]
3. Faust KB, Chiantella V, Vinten-Johansen J, Meredith JH. Oxygen-derived free radical scavengers and skeletal muscle ischemic/reperfusion injury. *Am Surg* 1988;54:709–719. [PubMed: 3143290]
4. Hammond B, Hess ML. The oxygen free radical system: potential mediator of myocardial injury. *J Am Coll Cardiol* 1985;6:215–220. [PubMed: 2989350]
5. Hayes SG, McCord JL, Koba S, Kaufman MP. Gadolinium inhibits group III but not group IV muscle afferent responses to dynamic exercise. *J Physiol* 2009;587:873–882. [PubMed: 19103679]
6. Hirsch AT, Haskal ZJ, Hertzner NR, Bakal CW, Creager MA, Halperin JL, Hiratzka LF, Murphy WR, Olin JW, Puschett JB, Rosenfield KA, Sacks D, Stanley JC, Taylor LM Jr, White CJ, White J, White RA, Antman EM, Smith SC Jr, Adams CD, Anderson JL, Faxon DP, Fuster V, Gibbons RJ, Halperin JL, Hiratzka LF, Hunt SA, Jacobs AK, Nishimura R, Ornato JP, Page RL, Riegel B. ACC/AHA 2005 guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): executive summary a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease) endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation. *J Am Coll Cardiol* 2006;47:1239–1312. [PubMed: 16545667]
7. Iida H, Kurano M, Takano H, Kubota N, Morita T, Meguro K, Sato Y, Abe T, Yamazaki Y, Uno K, Takenaka K, Hirose K, Nakajima T. Hemodynamic and neurohumoral responses to the restriction of femoral blood flow by KAATSU in healthy subjects. *Eur J Appl Physiol* 2007;100:275–285. [PubMed: 17342543]
8. Koch DW, Leuenberger UA, Proctor DN. Augmented leg vasoconstriction in dynamically exercising older men during acute sympathetic stimulation. *J Physiol* 2003;551:337–344. [PubMed: 12824451]
9. Kubota A, Sakuraba K, Sawaki K, Sumide T, Tamura Y. Prevention of disuse muscular weakness by restriction of blood flow. *Med Sci Sports Exerc* 2008;40:529–534. [PubMed: 18379217]
10. Madarama H, Neya M, Ochi E, Nakazato K, Sato Y, Ishii N. Cross-transfer effects of resistance training with blood flow restriction. *Med Sci Sports Exerc* 2008;40:258–263. [PubMed: 18202577]
11. Manini TM, Clark BC. Blood flow restricted exercise and skeletal muscle health. *Exerc Sport Sci Rev* 2009;37:78–85. [PubMed: 19305199]
12. Montain SJ, Jilka SM, Ehsani AA, Hagberg JM. Altered hemodynamics during exercise in older essential hypertensive subjects. *Hypertension* 1988;12:479–484. [PubMed: 3192293]
13. Moore DR, Burgomaster KA, Schofield LM, Gibala MJ, Sale DG, Phillips SM. Neuromuscular adaptations in human muscle following low intensity resistance training with vascular occlusion. *Eur J Appl Physiol* 2004;92:399–406. [PubMed: 15205956]

14. Parker BA, Ridout SJ, Proctor DN. Age and flow-mediated dilation: a comparison of dilatory responsiveness in the brachial and popliteal arteries. *Am J Physiol Heart Circ Physiol* 2006;291:H3043–3049. [PubMed: 16861699]
15. Pickering TG. Pathophysiology of exercise hypertension. *Herz* 1987;12:119–124. [PubMed: 2953661]
16. Seal JB, Gewertz BL. Vascular dysfunction in ischemia-reperfusion injury. *Ann Vasc Surg* 2005;19:572–584. [PubMed: 15981128]
17. Shinohara M, Kouzaki M, Yoshihisa T, Fukunaga T. Efficacy of tourniquet ischemia for strength training with low resistance. *Eur J Appl Physiol Occup Physiol* 1998;77:189–191. [PubMed: 9459541]
18. Stok WJ, Baisch F, Hillebrecht A, Schulz H, Meyer M, Karmaker JM. Noninvasive cardiac output measurement by arterial pulse analysis compared with inert gas rebreathing. *J Appl Physiol* 1993;74:2687–2693. [PubMed: 8396105]
19. Sugawara J, Tanabe T, Miyachi M, Yamamoto K, Takahashi K, Iemitsu M, Otsuki T, Homma S, Maeda S, Ajisaka R, Matsuda M. Non-invasive assessment of cardiac output during exercise in healthy young humans: comparison between Modelflow method and Doppler echocardiography method. *Acta Physiol Scand* 2003;179:361–366. [PubMed: 14656373]
20. Takano H, Morita T, Iida H, Asada K, Kato M, Uno K, Hirose K, Matsumoto A, Takenaka K, Hirata Y, Eto F, Nagai R, Sato Y, Nakajima T. Hemodynamic and hormonal responses to a short-term low-intensity resistance exercise with the reduction of muscle blood flow. *Eur J Appl Physiol* 2005;95:65–73. [PubMed: 15959798]
21. Takarada Y, Nakamura Y, Aruga S, Onda T, Miyazaki S, Ishii N. Rapid increase in plasma growth hormone after low-intensity resistance exercise with vascular occlusion. *J Appl Physiol* 2000;88:61–65. [PubMed: 10642363]
22. Takarada Y, Takazawa H, Sato Y, Takebayashi S, Tanaka Y, Ishii N. Effects of resistance exercise combined with moderate vascular occlusion on muscular function in humans. *J Appl Physiol* 2000;88:2097–2106. [PubMed: 10846023]
23. Tanaka H, Kiyonaga A, Terao Y, Ide K, Yamauchi M, Tanaka M, Shindo M. Double product response is accelerated above the blood lactate threshold. *Med Sci Sports Exerc* 1997;29:503–508. [PubMed: 9107633]
24. Wesseling KH, Jansen JR, Settels JJ, Schreuder JJ. Computation of aortic flow from pressure in humans using a nonlinear, three-element model. *J Appl Physiol* 1993;74:2566–2573. [PubMed: 8335593]

Figure 1.

Changes in blood pressure in response to the exercise sessions with (closed circles) or without (open circles) leg blood flow restriction (BFR). * $P < 0.05$ vs. baseline in the same condition. † $P < 0.05$ vs. control condition; Baseline refers to standing on the treadmill before exercise. Data are mean $\pm$ SEM.

Figure 2. Changes in systemic hemodynamic variables in response to the exercise sessions with (closed circles) or without (open circles) leg blood flow restriction (BFR). Data show relative changes from the baseline (standing on the treadmill before exercise). †P<0.05 vs. control condition. Data are mean±SEM.

Figure 3.

Changes in double product (an index of myocardial oxygen demand) and stroke volume/pulse pressure ratio (SV/PP; an index of systemic arterial compliance) to the exercise sessions with (closed circles) or without (open circles) leg blood flow restriction (BFR). Data show relative changes from the baseline (standing on the treadmill before exercise). †P<0.05 vs. control condition. Data are mean±SEM.

Figure 4. Popliteal artery flow-mediated vasodilation (FMD) (*top*) and normalized FMD with area under the curves of shear rate (*bottom*) before and after exercise session with or without leg blood flow restriction (BFR). Data are mean $\pm$ SEM.

Table 1

Hemodynamic variables and popliteal artery properties before and after walking test (post measurements were performed 20 minutes following the completion of the exercise test).

	Control		BFR	
	Before	After	Before	After
Heart Rate, bpm	54 ± 2	53 ± 1	56 ± 2	56 ± 1
Brachial SBP, mmHg	111 ± 2	112 ± 2	112 ± 2	114 ± 2
Brachial DBP, mmHg	61 ± 1	63 ± 1	62 ± 1	64 ± 1
Ankle SBP, mmHg	125 ± 2	125 ± 1	122 ± 2	125 ± 2
Ankle DBP, mmHg	64 ± 1	65 ± 1	65 ± 2	66 ± 1
Ankle-Brachial Index, ratio	1.13 ± 0.01	1.12 ± 0.01	1.08 ± 0.01	1.11 ± 0.01
Diastolic Baseline diameter, mm	6.0 ± 0.2	5.9 ± 0.3	5.9 ± 0.2	6.1 ± 0.2
Blood flow at rest, ml/min	85.7 ± 13.5	81.6 ± 10.4	93.1 ± 7.2	92.2 ± 12.1
AUC of shear rate, s	5208 ± 415	5199 ± 352	5353 ± 412	5307 ± 400

Data are mean±SEM. SBP=systolic blood pressure, DBP=diastolic blood pressure, AUC=area under the curve.