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Original Research

Acute cardiovascular response after maximal cycling exercise in endurance- and strength-trained men

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Abstract

Background: Post-exercise hypotension is an important regulator of ambulatory blood pressure—an independent risk factor for cardiovascular disease. Although post-exercise hypotension may be associated with aerobic exercise capacity in male athletes, it has not been explored whether muscular strength or strength training affects post-exercise hypotension. To elucidate whether the cardiovascular responses after exercise differ between endurance- and strength-trained men, this study investigated cardiovascular indices (e.g., blood pressure, cardiac output, total peripheral resistance) before and after maximal cycling exercise in male long-distance runners, weightlifters, and sedentary peers. **Methods**: Ten male intercollegiate long-distance runners, nine weightlifters, and 10 sedentary peers performed maximal incremental cycling. Cardiovascular indices were measured before and at 15, 30, 60, and 90 min after the exercise. **Results**: The runners had remodeled hearts and higher maximal oxygen uptake, and the weightlifters had a higher resting systolic blood pressure. Blood pressure decreased after exercise in all groups. Although the weightlifters showed higher systolic blood pressure than the sedentary men throughout the experiment, the changes from baseline showed no intergroup differences in blood pressure. Cardiac output increased and total peripheral resistance decreased after exercise relative to baseline in all groups; there were no intergroup differences in changes in these measures. **Conclusions**: The mode of habitual exercise training may affect post-exercise hypotension similarly in endurance- and strength-trained male athletes in spite of their different cardiovascular adaptations.

Keywords: Blood pressure; Endurance exercise; Post-exercise hypotension; Resistance exercise; Vascular resistance

1. Introduction

Hypertension is a well-recognized independent risk factor for coronary heart disease—one of the leading causes of mortality worldwide. A higher proportion of men have hypertension compared with age-matched women, especially in young and middle-aged individuals [1]. Not only office blood pressure (BP) but also ambulatory BP is important to consider for the prevention of coronary heart disease; individuals with normal office BP and elevated 24-hour ambulatory BP (i.e., masked hypertension) may have a larger hazard ratio for all-cause mortality than individuals with sustained hypertension [2]. An essential contributor to ambulatory BP is the BP response to exercise, since endurance (e.g., walking or cycling) and resistance (e.g., lifting objects and mopping) exercises are components daily living activities.

The BP responses to exercise consists of two components, a BP elevation during exercise and a BP reduction after exercise known as "post-exercise hypotension". In particular, post-exercise hypotension is important because it persists for 2 to 4 hours under laboratory conditions and can be sustained for 12 hours or longer under free-living conditions [3]. There are inter-individual differences in the levels of post-exercise hypotension, and it is affected by factors such as physical fitness or daily exercise habits. For example, Dujic *et al.* [4] reported that greater post-exercise hypotension was associated with lower aerobic exercise capacity in male soccer players. However, previous studies have focused mainly on aerobic capacity and endurance exercise training [4,5], even though cardiovascular adaptations depend on exercise mode as endurance-trained athletes have eccentric remodeled heart and lower arterial stiffness, while strength-trained athletes have concentric remodeled heart and higher arterial stiffness [6,7]. To understand the effects of physical fitness or exercise training on post-exercise hypotension, other types of athletes (i.e., other than endurance-trained athletes) also need to be investigated. The aim of this study was to examine whether changes in cardiovascular indices (e.g., BP, cardiac output (CO), and peripheral vascular resistance (TPR)) after maximal cycling exercise differ between male long-distance runners (i.e., endurance-trained athletes) and weightlifters (i.e., strength-trained athletes).

2. Material and methods

2.1 Subjects

Thirty-three male collegiate students participated in this study (age, 18–23 years). The exclusion criteria for

participation were as follows: coronary heart disease, hypertension, ischemic heart disease, cardiac valve disease, cardiac myopathy, diabetes, and tachycardia. They were divided into three groups based on their exercise experiences: long-distance runners (n = 10), weightlifters (n = 13), and sedentary peers (control, n = 10). Daily exercise habit was investigated by using a questionnaire (sedentary vs. weightlifters vs. runners (mean $\pm$ SD); time, (0.1 $\pm$ 0.3) vs. (2.4 $\pm$ 0.5) vs. (2.6 $\pm$ 0.5) h/day and frequency, (0.2 $\pm$ 0.6) vs. (6.0 $\pm$ 0.0) vs. (5.5 $\pm$ 1.2) days/week). We excluded data of four weightlifters who had >94 kg body weight from later analysis because of technical problems with the acquisition of clear echocardiographic images.

Written informed consent was obtained after a verbal explanation of the study and anticipated risks in accordance with the Declaration of Helsinki. All procedures were reviewed by the ethics committee of the Japan Institute of Sports Sciences.

2.2 Procedure

The subjects visited the laboratory twice. During the first visit, the study was explained in detail, and the subjects became accustomed to using the experimental instruments. The second visit on the next day was used for data collection. Subjects fasted for 12 h prior to the test and were instructed to avoid strenuous activity and caffeine for 24 h prior to each test.

The air temperature in the laboratory was maintained between 23 and 25 °C. Baseline measurements of cardiac morphology (e.g., left ventricular mass and wall thickness), BP, blood flow (e.g., CO, stroke volume (SV), and forearm blood flow (FBF)), and vascular resistance (TPR and forearm vascular resistance (FVR)) were performed after subjects rested for more than 15 min in the supine position. Subjects were instructed to rest in the supine position from 5 to 90 min after maximal cycling exercise, and BP, SV, heart rate (HR), CO, TPR, FBF, and FVR were measured at 15 (p15), 30 (p30), 60 (p60), and 90 (p90) min after cessation of exercise. The subjects were allowed to drink water ad libitum during the experimental period.

2.3 Exercise and measurement of maximal oxygen uptake

The exercise test consisted of cycling on an ergometer (Powermax VII, COMBI Wellness, Tokyo, Japan) to prevent accidents (e.g., knee injury) especially in sedentary men who were not familiar with intensive exercise and heavy weightlifters. The subjects warmed up with a load of 100 W for 4 min, while HR was monitored using an electrocardiographic signal transmitter (ZS-910P, Nihon Kohden, Tokyo, Japan). Next, the maximal incremental cycling test started with a load of 120 W and increased by 17 to 30 W every 2 min. The test continued until the subjects became exhausted.

Testing was terminated if three or more of the following five criteria were met: (1) rating of perceived exertion greater than 17 on the Borg Scale; (2) respiratory exchange ratio greater than 1.1; (3) no increase in HR with increasing cycling load; (4) plateaued oxygen uptake (VO_2) (increase of 150 mL or less) with increasing cycling load, and (5) unsustainable pedaling at 60 rpm due to fatigue.

Ventilatory parameters and oxygen (O_2) and carbon dioxide (CO_2) concentrations in expired air during the cycling test were analyzed breath-by-breath using an open-circuit spirometry gas analysis system (Vmax, SensorMedics, Yorba Linda, CA, United States). The system was calibrated using a calibration gas with a known concentration of O_2 and CO_2 and a constant volume prior to measurement. The mean VO_2 was calculated for every 30 s of exercise. The highest value was defined as maximal oxygen uptake (VO_2max).

2.4 Cardiovascular measurements

2.4.1 Arterial blood pressure

Systolic and diastolic BP of the left upper arm (SBP and DBP, respectively) were measured in triplicate using the oscillometric method (Jentow, Nihon Colin, Aichi, Japan). The average value was used in the analysis. Mean BP (MBP) was estimated using the following formula: MBP = 2/3 (DBP) + 1/3 (SBP).

2.4.2 Cardiac structure and function

Echocardiography was performed (SSD-6500, Aloka Company, Tokyo, Japan) according to the recommendations of the American Institute of Ultrasound in Medicine [8]. ECG was monitored throughout the experiment (SSD-6500, Aloka Company, Gunma, Japan), and HR was calculated from the R-R interval. Echocardiography was conducted by YS, a researcher with an experience of echocardiograms for a few years [9], in the same laboratory as the exercise test. B-mode long-axis views of the left ventricle from the left parasternal region were obtained with a 1.88-MHz sector probe. M-mode measurements were performed over 10 continuous heartbeats. The imaging was analyzed offline using the echocardiography software. The left ventricular internal dimension at end-diastole (LVIDd), left ventricular internal dimension at end-systole (LVIDs), interventricular septum wall thickness at end-diastole (IVSTd), and posterior wall thickness at end-diastole (PWTd) were measured, and the mean value over three heartbeats was calculated. The ratio of average wall thickness, the average of IVSTd and PWTd, to LVIDd was used as an index of left ventricular hypertrophy [10]. Left ventricular mass (LVmass) was calculated according to a previous study [11]. End-diastolic volume (EDV) and end-systolic volume (ESV) were calculated using the method described by Teichholz *et al*. [12]. The SV was obtained by subtracting the ESV from the EDV. The ratio of SV to EDV represented ejection fraction (EF). CO was calculated as the product of HR and SV. TPR was calculated using the following formula: TPR = MBP / CO.

2.4.3 Forearm blood flow

TPR is a crucial factor of BP. However, the adaptations of limb arteries to exercise training depend on habitual activity status of each arm and leg, as radial arterial distensibility was greater in dominant arm versus non-dominant arm of baseball players and hammer throwers in a previous study [13]. Since weightlifting requires more arm strength relative to running, not only systemic but also forearm blood flow and vascular resistance (i.e., FBF and FVR) were measured. FBF was measured by venous occlusion plethysmography [14] using electronically calibrated strain gages [15] (EC-4, D. E. Hokanson, Bellevue, WA, United States). The strain gage was surrounded 5 cm below the antecubital crease of the right forearm. A rapid cuff inflator (E-20, D. E. Hokanson) fixed on the right upper arm was inflated to 50 mmHg for 5 s within 10 s, for a total of 3 min. The output voltage was converted from analog to digital (Power Lab, AD instruments, New South Wales, Australia), exported, and stored in a general-purpose computer for analysis (LabChart 5.25, AD instruments, New South Wales, Australia). FVR was calculated using the following formula: FVR = MBP / FBF.

2.5 Statistical analysis

Data are presented as mean ± standard deviation. The significance level was set at $p < 0.05$. SPSS version 25 (IBM, Armonk, NY, United States) was used for the statistical analysis. One-way (group) analysis of variance (ANOVA) was used to analyze the physical characteristics and baseline parameters. Two-way (group × time) repeated-measures ANOVA was used to analyze time-dependent changes in cardiovascular parameters. For post-hoc analysis, the Bonferroni method was used where significant F values were found.

3. Results

3.1 Physical characteristics

Age was higher among runners than weightlifters (Table 1, $p = 0.007$). The weightlifters had a higher body weight than the sedentary controls ($p = 0.019$) or runners ($p = 0.003$). The training period (range; runners, 4–15 years and weightlifters, 2–4 years), exercise time, and VO_2max, an indicator of aerobic exercise capacity, were greater for the runners than for the weightlifters ($p = 0.000$, $p = 0.003$, and $p = 0.000$, respectively) or the controls ($p = 0.000$, $p = 0.014$, and $p = 0.000$, respectively). Age, height, weight, and training period were lower in the weightlifter group (n = 9, Table 1) relative to weightlifters excluded from the statistical analysis (n = 4; 20.5 ± 0.6 years ($p = 0.011$), 173.9 ± 1.4 cm ($p = 0.018$), 103.3 ± 5.9 kg ($p = 0.000$), and 4.8 ± 0.5 years ($p = 0.003$), respectively). VO_2max was higher in the weightlifter group (Table 1) than in the excluded weightlifters (38.2 ± 5.4 mL/kg/min, $p = 0.031$).

3.2 Cardiovascular measurements

3.2.1 Blood pressure

The weightlifters had a higher pre-exercise SBP than the sedentary controls ($p = 0.040$, Table 2). There were no intergroup differences in pre-exercise DBP or MBP.

There were no interactions between time and group for SBP, DBP, or MBP (Fig. 1). The BP after exercise was lower than before exercise in all three groups ($p = 0.000$–0.003). The weightlifters had a higher pre- and post-exercise SBP than the sedentary controls ($p = 0.003$). While the weightlifters had a higher MBP than the sedentary controls the difference was not statistically significant (ANOVA, $p = 0.081$).

3.2.2 Cardiac structure and function

The runners had a greater IVSTd and PWTd than the sedentary controls ($p = 0.043$ and $p = 0.001$, respectively; Table 2), a greater LVmass and EDV than the weightlifters ($p = 0.001$ and $p = 0.016$, respectively) or sedentary controls ($p = 0.000$ and $p = 0.008$, respectively), and a greater ESV than the weightlifters ($p = 0.032$).

SV decreased from baseline to 15 min after exercise in all three groups ($p = 0.048$, Fig. 2A). On the other hand, HR in all three groups increased during the recovery period versus before exercise ($p = 0.000$, Fig. 2B). The runners showed higher SV and lower HR than the sedentary controls ($p = 0.001$ and $p = 0.005$, respectively) and weightlifters ($p = 0.002$ and $p = 0.013$, respectively) throughout the experiment. CO increased from baseline until 60 min after exercise ($p = 0.000$, Fig. 2C) without intergroup differences.

3.2.3 Vascular resistance

There was no interaction between time and group for TPR (Fig. 3A). TPR showed a lower value after versus before exercise ($p = 0.000$–0.003) without intergroup differences. Group-time interaction was detected in FVR (ANOVA, $p = 0.047$; Fig. 3B). FVR was lower 15 and 30 min after exercise in the sedentary control group ($p = 0.001$ and $p = 0.019$, respectively) and 15 to 60 min after exercise in the runner group ($p = 0.000$–0.035) relative to before exercise. For the weightlifters, post-exercise FVR did not differ from the baseline value.

4. Discussions

We investigated acute changes in cardiovascular indices before and after maximal cycling exercise in male long-distance runners, weightlifters, and sedentary peers. As cardiac morphology and VO_2max of the runners and SBP of the weightlifters suggest, runners and weightlifters had adapted to each mode of exercise training. However, there were no intergroup differences in BP changes after the exercise. According to Poiseuille's law, TPR and CO are crucial determinants of BP. In this study, there were no statistically significant intergroup differences in the changes

Table 1. Physical characteristics of subjects.

	Sedentary controls			Weightlifters			Runners			*p*-value
	(n = 10)			(n = 9)			(n = 10)			(ANOVA)
Age, years	19.7	±	0.9	19.2	±	0.7	20.7	±	1.2†	$p = 0.007$
Height, cm	170.8	±	4.3	167.7	±	6.1	173.5	±	7.6	$p = 0.147$
Weight, kg	64.9	±	6.0	75.5	±	11.9*	62.3	±	3.6†	$p = 0.003$
Training period, years	0.0	±	0.0	3.3	±	0.7*	8.1	±	3.1*†	$p = 0.000$
Exercise time, min	14.0	±	3.1	13.2	±	1.0	17.1	±	2.1*†	$p = 0.002$
VO_2max, mL/kg/min	48.3	±	3.1	47.9	±	7.8	69.0	±	4.4*†	$p = 0.000$

Data are presented as means ± standard deviations.

*$p < 0.05$, compared with sedentary controls and †$p < 0.05$, compared with weightlifters.

VO_2max, maximal oxygen uptake and ANOVA, analysis of variance.

Table 2. Baseline cardiovascular structure and function.

	Sedentary controls			Weightlifters			Runners			*p*-value
	(n = 10)			(n = 9)			(n = 10)			(ANOVA)
SBP, mmHg	113	±	5	120	±	7*	117	±	2	$p = 0.044$
DBP, mmHg	61	±	4	63	±	5	62	±	7	$p = 0.603$
MBP, mmHg	78	±	4	82	±	4	80	±	6	$p = 0.187$
IVSTd, cm	0.84	±	0.11	0.88	±	0.09	0.95	±	0.09*	$p = 0.044$
PWTd, cm	0.81	±	0.03	0.87	±	0.07	0.94	±	0.10*	$p = 0.001$
Average wall thickness/LVIDd	0.17	±	0.01	0.18	±	0.02	0.18	±	0.01	$p = 0.238$
LVmass, g	136	±	18	148	±	22	186	±	22*†	$p = 0.000$
EDV, mL	108	±	15	110	±	16	130	±	12*†	$p = 0.004$
ESV, mL	47	±	8	45	±	8	55	±	8†	$p = 0.019$
EF, %	57	±	3	59	±	4	57	±	5	$p = 0.619$

Data are presented as means ± standard deviations.

*$p < 0.05$, compared with sedentary controls and †$p < 0.05$, compared with weightlifters.

SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure; IVSTd, interventricular septum wall thickness at end-diastole; PWTd, posterior wall thickness at end-diastole; LVIDd, left ventricular internal dimension at end-diastole; LVmass, left ventricular mass; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction; and ANOVA, analysis of variance.

in TPR and CO after maximal cycling exercise. This is the first study to demonstrate that the mode of habitual exercise training may affect post-exercise hypotension similarly in endurance- and strength-trained male athletes in spite of their different cardiovascular adaptations.

Although athletes habitually engage in intensive exercise training, most of previous studies on post-exercise hypotension used low- to moderate-intensity aerobic exercises as an exercise test. Regarding high-intensity exercise, only three studies investigated temporary hypotension after exercise in soccer players [4], normotensive young men [16], and runners [9]. Even though these previous studies [4,9,16] agree that post-exercise hypotension occurs after maximal exercise in young healthy men, further studies have been needed to understand hypotension after intensive exercise in athletes. Therefore, we chose maximal exercise as an exercise test. It would be novel to clarify whether changes in cardiovascular indices after maximal exercise differ between endurance- and strength-trained athletes.

VO_2max and EDV in the runner group and SBP before exercise in the weightlifter group were higher than in the sedentary control group. These results coincided with our previous investigations [6,7] and suggest that runners and weightlifters had adapted to each exercise training mode. Arterial stiffness, an essential determinant of BP, increases with intensive resistance exercise training [17], and strength-trained men have increased arterial stiffness [6]. The higher SBP of the weightlifters in this study may therefore be due to a higher arterial stiffness. In conflict with our previous study [7], there were no differences in left ventricular morphology between the weightlifters and sedentary controls. Generally, morphological adaptations (e.g., cardiac remodeling) appear later than functional adaptations (e.g., changes in blood pressure and arterial stiff-

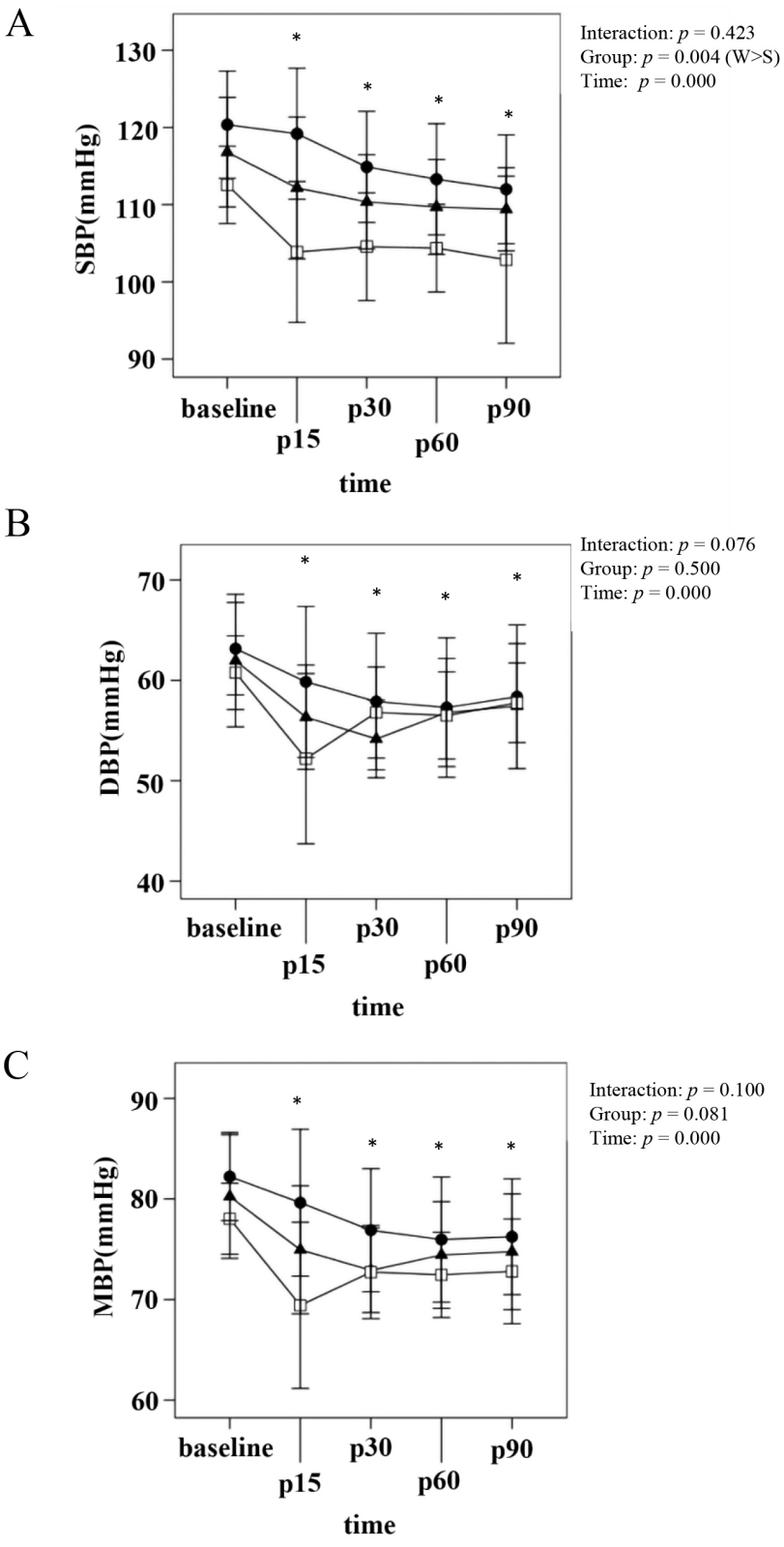

Fig. 1. Blood pressure before and after exercise. Data are presented as means ± standard deviations. □, Sedentary controls (S); ●, Weightlifters (W); and ▲, Runners. *$p < 0.05$ compared with baseline. p15, p30, p60, and p90; 15, 30, 60, and 90 min after exercise, respectively. SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure.

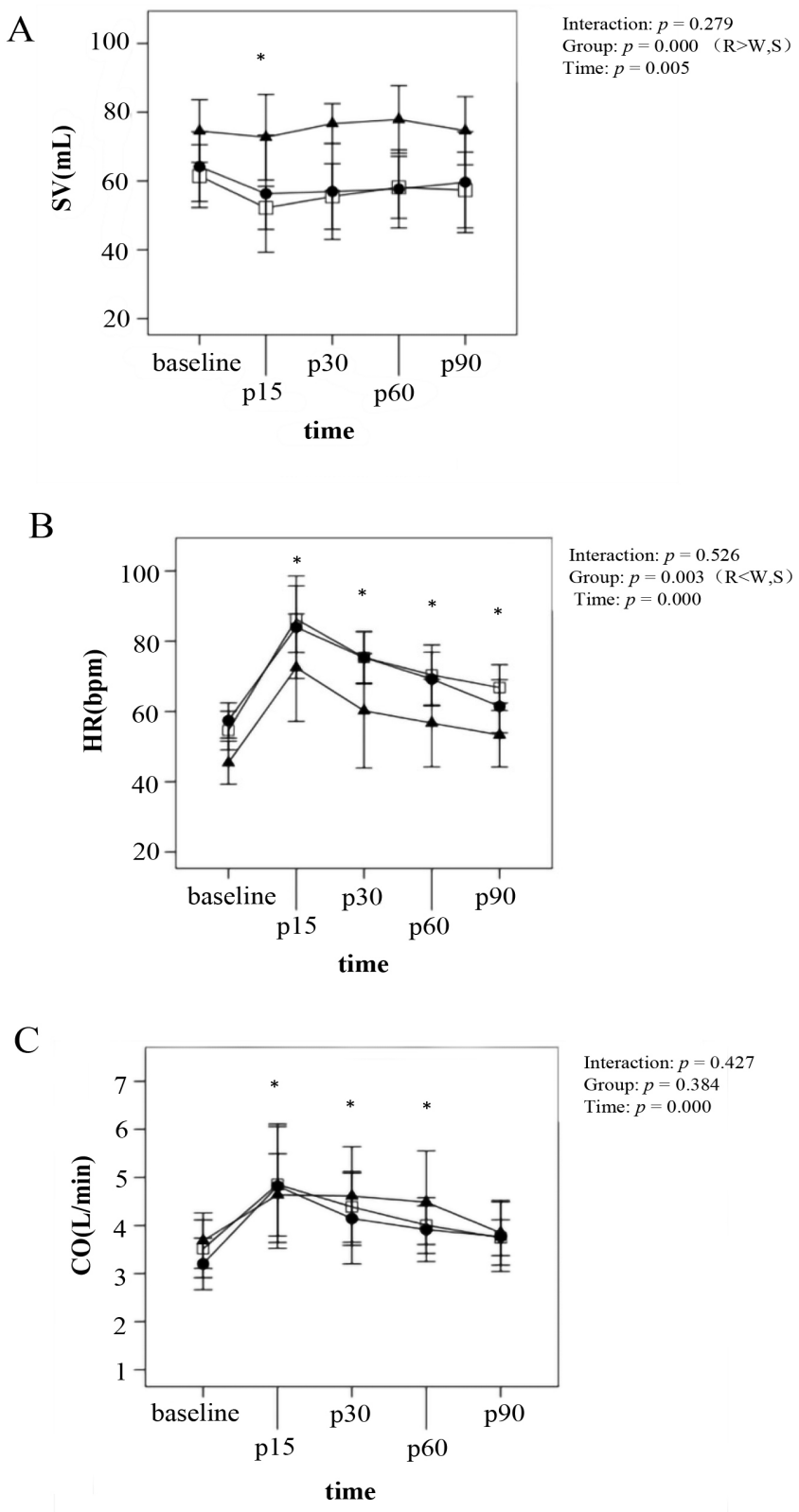

Fig. 2. Stroke volume (SV), heart rate (HR), and cardiac output (CO) before and after exercise. Data are presented as means ± standard deviations. □, Sedentary controls (S); ●, Weightlifters (W); and ▲, Runners (R). *$p < 0.05$ compared with baseline. p15, p30, p60, and p90; 15, 30, 60, and 90 min after exercise, respectively.

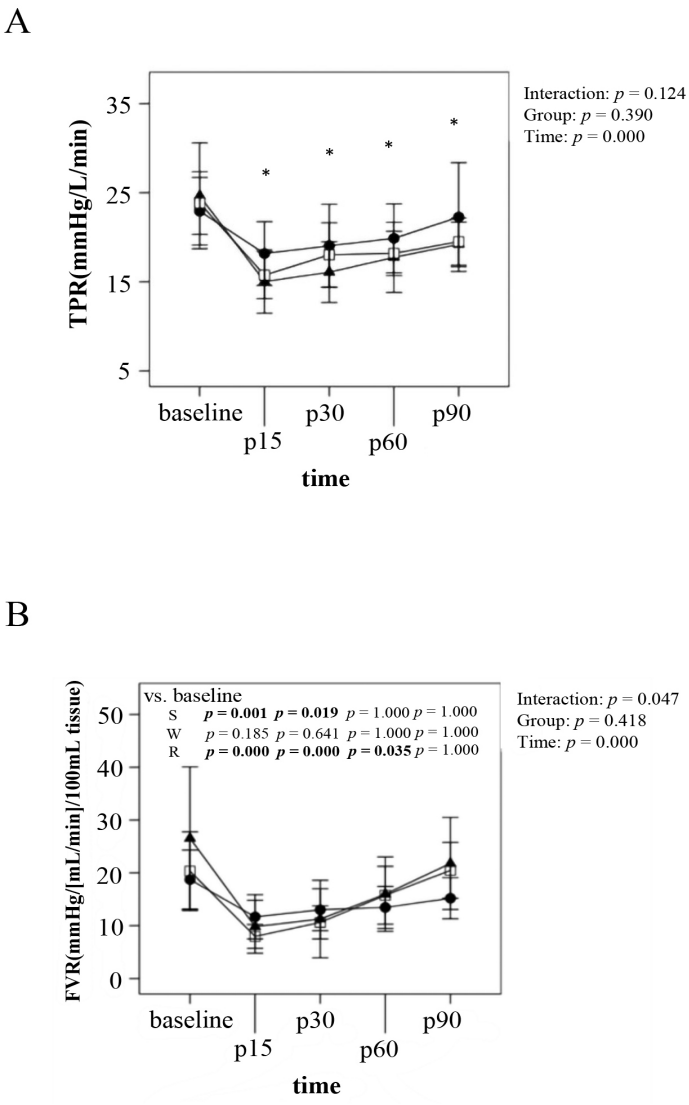

Fig. 3. Total peripheral resistance (TPR) and forearm vascular resistance (FVR) before and after exercise. Data are presented as means ± standard deviations. □, Sedentary controls (S); ●, Weightlifters (W); and ▲, Runners (R). p15, p30, p60, and p90; 15, 30, 60, and 90 min after exercise, respectively.

ness). Shorter training career of the weightlifters (3.3 ± 0.7 years) versus the runners (8.1 ± 3.1 years) may be associated with that cardiac remodeling was not detected in the weightlifters. Additionally, exclusion of weightlifters with body weight over 94 kg and longer career (4.8 ± 0.5 years) may be related to no cardiac morphology in the weightlifters.

Although there were differences in cardiovascular adaptations between the runners and weightlifters, post-exercise BP reductions were similar in both groups. Additionally, there were no intergroup differences in post-exercise reduction for TPR, an essential determinant of arterial load [18,19]. These results suggest that post-exercise hypotension is induced by a decrease in TPR and is not significantly affected by exercise training. As there was no difference between endurance- and strength-trained men in HR recovery during 30 s after exercise in our previous study [7], cardiovascular regulation after exercise may be similar between endurance- and strength-trained young men. However, we should note that the present results are inconsistent with a previous study of male soccer players [4] where baseline SBP and VO_2max were correlated with post-exercise reduction of SBP and DBP, respectively. This discrepancy may be due to the characteristics of subjects; the previous study [4] included a hypertensive man (resting SBP >150 mmHg) and participant age was higher (maximal age of subjects, 31 years) versus subjects in this study who were normotensive university students.

There was no detectable interaction between group and time in TPR, but the interaction in FVR was statistically significant. FVR was lower 15 to 60 or 15 to 30 min after versus before exercise in the runners and sedentary controls,

respectively. These results are generally in agreement with those of a previous study [20]. However, the FVR did not change with exercise for weightlifters. Local vasodilation is implicated in the vascular endothelial function. As suggested by a previous study investigating plasma concentrations of endothelin-1, a vasoconstrictor peptide, there are differences in endothelial function among strength-trained men versus endurance-trained and sedentary men [6]. Further studies are needed to elucidate the effects of exercise training on post-exercise hypotension in more detail.

Generally, an increase in CO elevates the BP. In this study, CO was higher after exercise than before exercise in all groups. This result is in accordance with a previous study [20] and suggests that CO did not participate in post-exercise hypotension. Senitko *et al*. [5] also demonstrated that post-exercise hypotension was independent of CO in sedentary men and women and endurance-trained women. However, they reported that in endurance-trained men, post-exercise hypotension was induced by decreases in CO and SV [5]. In addition, Dujic *et al*. [4] showed that post-exercise hypotension was due to reductions in CO and SV in male soccer players. The discrepancies in post-exercise changes in CO among studies may be due to differences in exercise intensity, time, and mode. Subjects performed maximal cycling exercise in this study (approximately 13–17 min) and the study by Coats *et al*. [20] (no information on exercise time), but Senitko *et al*. [5] used cycling exercise of lower intensity (60 % VO_2max) and longer time (60 min). In the study by Dujic *et al*. [4], participants conducted maximal running, which requires more arm action than cycling.

The present study had some limitations. First, the training period was not the same between the groups. The runners had a longer training period than the weightlifters because the population who participated in weightlifting during junior high school is very limited in Japan. Strength-trained athletes with longer careers should be investigated. Second, sample size was relatively small, and a power analysis was not performed. It is possible that intergroup differences in some parameters including BP changes and cardiac remodeling were underestimated. Third, since we studied young male collegiate students, the present findings cannot be generalized to the entire population. Forth, VO_2max may be underestimated in the runners due to exercise mode of this study (i.e., cycling). Fifth, BP did not return to the baseline level by the end of the recovery time. Experiments with longer recovery time is needed. Finally, we did not evaluate BP during exercise, which may influence the post-exercise BP changes.

5. Conclusions

The present study suggests that changes in blood pressure after maximal cycling exercise are similar between endurance-trained, strength-trained, and sedentary young men although their cardiovascular adaptations are different.

Abbreviations

BP, blood pressure; HR, heart rate; VO_2max, maximal oxygen uptake; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure; LVIDd, left ventricular internal dimension at end-diastole; LVIDs, left ventricular internal dimension at end-systole; IVSTd, interventricular septum wall thickness at end-diastole; PWTd, posterior wall thickness at end-diastole; LVmass, left ventricular mass; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction; SV, stroke volume; CO, cardiac output; TPR, total peripheral resistance; FVR, forearm vascular resistance.

Author contributions

YS contributed to the entire process of the work, including the design of the study, the acquisition, analysis, and interpretation of the data, and the manuscript. MN, TO and KE contributed to the acquisition and interpretation of data. MN, TO and KE contributed to the interpretation of the data and critical repeated revisions of the manuscript. All authors contributed to manuscript revision and read and approved the submitted version.

Ethics approval and consent to participate

All experiments were carried out in accordance with the declaration of Helsinki and approved by the ethical committee of Japan Institute of Sports Sciences. Written informed consent was obtained from all the subjects.

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Conflict of interest

The authors declare no conflict of interest.

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