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RESEARCH ARTICLE

High-intensity intermittent exercise and cardiovascular and autonomic function

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Abstract
Objective The effect of 12 weeks of high-intensity intermittent exercise (HIIE) on cardiac, vascular, and autonomic function of young males was examined.
Methods Thirty-eight young men with a BMI of 28.7 ± 3.1 kg m^{-2} and age 24.9 ± 4.3 years were randomly assigned to either an HIIE or control group. The exercise group underwent HIIE three times per week, 20 min per session, for 12 weeks. Aerobic power and a range of cardiac, vascular, and autonomic measures were recorded before and after the exercise intervention.
Results The exercise, compared to the control group, recorded a significant reduction in heart rate accompanied by an increase in stroke volume. For the exercise group forearm vasodilatory capacity was significantly enhanced, $P < 0.05$. Arterial stiffness, determined by pulse wave velocity and augmentation index, was also significantly improved, after the 12-week intervention. For the exercise group, heart period variability (low- and high-frequency power) and baroreceptor sensitivity were significantly increased.
Conclusion High-intensity intermittent exercise induced significant cardiac, vascular, and autonomic improvements after 12 weeks of training.

Keywords High-intensity intermittent exercise · Stroke volume · Heart period variability · Baroreceptor sensitivity · Arterial stiffness

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Introduction

The majority of studies examining resting cardiac, vascular, and autonomic change to chronic exercise have focused on steady-state aerobic training such as regular running and cycling. The major cardiac adaptations to these forms of aerobic exercises have included a lowering of resting heart rate and an increase in stroke volume [40]. Vascular adaptations to aerobic exercise have been examined by assessing variables such as limb blood flow, limb vasodilatory capacity, and arterial stiffness. Limb blood flow and vasodilatory capacity, indicants of the ability of the arterioles to dilate, has been enhanced by aerobic training in the exercising [19] and non-exercising muscles [31]. Boutcher and Boutcher [2] also found higher vasodilatory capacities in forearm and leg muscles in runners compared with the untrained. Regular aerobic training has also been shown to reduce arterial stiffness in healthy young, older, congestive heart failure, and diabetic adults.

Autonomic adaptations occurring with aerobic exercise include changes in vagal influence on the heart and arterial baroreflex sensitivity (BRS). Heart period variability (HPV) has typically been used to assess cardiac vagal influence. Vagal influence on the heart, as measured by HPV, has been shown to increase after steady-state endurance exercise training [39]. A significant effect of exercise training on resting high-frequency power was found in a meta analysis with the greatest effect shown in younger participants and longer interventions [29]. With regard to aerobic exercise training and BRS, some researchers have found an increase in arterial BRS following endurance training [21], whereas others have reported either no change or a decrease [4].

In contrast to aerobic exercise, examination of the resting cardiac, vascular, and autonomic function response

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to other forms of exercise has been scant. For example, little is known about the potential resting cardiovascular changes that may occur after exposure to regular high-intensity intermittent exercise (HIIE). HIIE typically consists of repeated short sprints on a cycle ergometer (6–30 s) followed by a brief period of low-intensity cycling [1]. HIIE has been shown to result in significant increases in both anaerobic and aerobic fitness [1]. In a series of studies, Gibala and colleagues [10] have examined skeletal muscle adaptations to HIIE and have demonstrated that HIIE consistently elevates maximal activity and protein content of a number of mitochondrial enzymes. Participation in HIIE also results in significant decreases in total and abdominal fat [1]. In addition, compared to steady-state aerobic exercise, HIIE has been shown to have a significantly greater acute impact on the autonomic nervous system, determined by heart rate and plasma catecholamine response to a single bout of exercise [37]. Although the effect of long-term HIIE training on autonomic function has not been examined, it has been shown that high-intensity endurance training was more effective for enhancing cardiac vagal control than a low-intensity exercise program [20]. Thus, carrying out more intensive exercise training may induce greater autonomic and cardiovascular adaptations. Therefore, the aim of this study was to examine the effect of 12 weeks of HIIE on resting heart rate, stroke volume, limb blood flow, limb vasodilatory capacity, arterial stiffness, HPV, and BRS in young males.

Methods

This study was an open-label, parallel-group with allocation concealment (sealed envelopes) individually randomized trial. The allocation ratio was 1:1 for two groups. The study took place in the exercise physiology laboratories, University of New South Wales, Sydney, Australia from March 2009 to November 2010. The trial has been registered in the Australian New Zealand Clinical Trials Registry (Registration Number ACTRN12612001003864).

Subjects

After obtaining approval from University Ethics Committee, males aged 18–35 years and a BMI of 23–35 kg m^{-2} were invited to participate in the intervention through advertisements and notice boards placed in public areas and student magazines. Study inclusion criteria included being a non-smoker, being physically inactive, not being diagnosed with any type of cardiovascular and pulmonary disease, and not being on any regular medication. Out of 38 participants recruited and consented, 20 participants were randomly allocated to an intervention group to receive a 12-week supervised HIIE training and 18 participants were assigned to a control group and were asked to maintain their normal daily routine (Fig. 1). For a variety reasons, five subjects withdrew from the exercise group and three from the control group. There was no significant difference for any variable between the drop-outs and subjects who completed the study. Allocation concealment was conducted by randomly picking a piece of paper labeled "exe" or "con" from the subject pool in an envelope. The trial was not blinded for participants, because of the nature of the exercise intervention, but outcome assessors and data analyzers were blinded. Subject number was based on results of prior research examining aerobic power increase to the same exercise protocol [38]. Aerobic power increase to HIIE produced a large effect size of 0.9. Thus, based on a moderately large effect size, sample sizes of 10–12 males per group provides a statistical power of 0.8 at an alpha of $P < 0.05$. Consequently, having greater than 16 males in each group provided a buffer for drop-out.

Pre and post testing

Participant recruitment, assignment, and drop-out are shown in Fig. 1. Pre testing involved participants coming to the laboratory after an overnight fast between 7 am and 11 am. The testing room was quiet and was maintained at a constant ambient air temperature of 22–23 °C. Participants

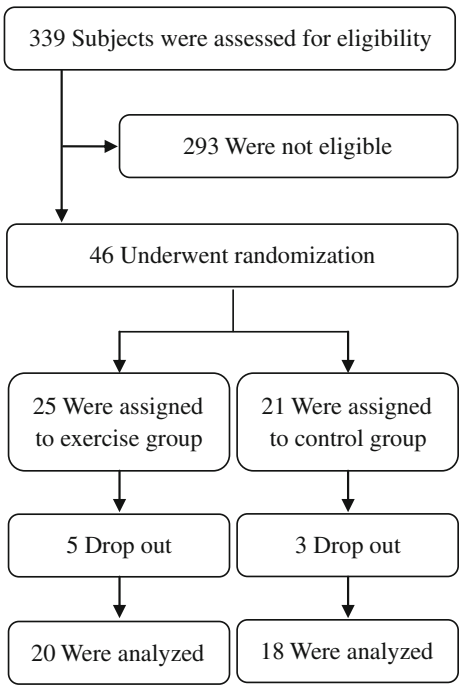

Fig. 1 Flow chart of participant recruitment, assignment and drop-out

were also instructed to abstain from caffeine, tobacco, and alcohol for at least 8 h before testing to avoid any confounding effects of stimulants or depressants on autonomic function. Participants were instructed not to exercise within 24 h of the test to eliminate the residual effects of exercise.

HIIE training program

The intervention group performed supervised HIIE training on a cycle ergometer (Monark 828E) at a frequency of three times a week for 12 weeks. Participants were instructed to cycle at a workload of 80–90 % of their maximum heart rate with a cadence between 120 and 130 rpm. During recovery, the cadence was reduced to 40 rpm with no change in resistance. Each exercise session consisted of a 5-min warm-up, 20 min of 8-s sprint and a 12-s recovery, and a 5-min cool-down. Workload was gradually increased during the trial depending on the participant's heart rate which was monitored by a Polar Heart Rate Monitor (Polar Electro Oy; Kempele, Finland).

HPV

Resting supine R–R intervals were recorded for 30 min, at a sampling rate of 1,000 Hz, using the Polar RS800CX Heart Rate Monitor (Polar Electro Oy; Kempele, Finland) and data were analyzed with the Polar ProTrainer 5^{TM} software. Total power (TP), parasympathetic (HF, RMSSD, and pNN50) and measures of cardiac sympathetic influence (VLF, LF, and TP) of HPV were evaluated.

Arterial baroreflex sensitivity

Arterial BRS was determined under spontaneous behavior using the sequence method (BaroCor^{TM} AFx, AtCor Medical, Australia). This technique has been shown to be a valid and reproducible method for determining arterial BRS in humans [26].

Augmentation index (AIx)

Blood pressure was recorded (BP Monitor; UNSW) with a wrist sensor placed on the left wrist radial pulse (Model 7000, Colin Medical; Japan). Central aortic waveforms were derived from the recorded radial arterial pressure waveform using SphygmoCor software (SphygmoCor, SCOR-2000, AtCor Medical; Australia). The use of a transfer function (SphygmoCor) has been validated against directly measured central aortic pressure [33]. An index of arterial stiffness (AIx) was defined as the ratio of the difference between the first (early systolic shoulder) and second peaks (late systolic shoulder) to pulse pressure and was expressed as a percentage. Because AIx is influenced by heart rate, it was normalized to a rate of 75 b min^{-1}.

Pulse wave velocity (PWV)

Central PWV was measured between the carotid and femoral arteries using a SphygmoCor® PWV system model SCOR-Vx and was analyzed with SphygmoCor Cardiovascular Management Suite (CvMS) software version 8. An ECG lead II was recorded during the test as well as pre systolic and diastolic blood pressure.

Impedance cardiography (ICG)

Impedance cardiography was measured using a Minnesota impedance cardiograph (Model 304B, Surcom; Minneapolis, MN, USA) and included the recording of heart rate, stroke volume, cardiac output, pre-ejection period (PEP), and left ventricle ejection time (LVET). Data were analyzed using cardiac output program software (COP, Microtronics Inc; Chapel Hill, NC).

Forearm blood flow and peak forearm blood flow

Forearm blood flow (FBF) was assessed using a plethysmograph (Model EC4, D.E. Hokansen, Inc; Bellvue, WA, USA) as has been previously described [3]. Peak FBF was used as a measure of vasodilatory capacity and was determined by a reactive hyperaemia condition using the venous occlusion technique. It has been shown that 5 min of blood flow occlusion induces a maximal vascular response. More details of peak FBF measurement can be found in a previously published article [3]. Forearm vascular resistance (FVR) was also calculated by dividing mean arterial pressure (MAP) by FBF.

Arterial blood pressure and R–R interval

Participants' pulse rates were obtained using the standard lead II of a surface electrocardiogram (LifePulse model LP10, HME), and beat-to-beat blood pressure was measured continuously using the Colin Jentow (Model 7000, Colin Medical; Japan).

Aerobic power

Maximal or peak oxygen consumption was measured using an electrically braked cycle ergometer (Ergomedic 839E, Monark) and open-circuit spirometry (True Max 2400, ParvoMedics). Participants cycled at a cadence of 70 rpm until volitional exhaustion. A ramp protocol was used, after a 3-min warm-up period at 30 W, the workload increased by 1 W every 2 s. Heart rate was monitored by the Polar

SC800 Heart Rate Monitor. The end point was achieved when the participant was unable to continue.

Statistical analysis

Data were analyzed using Statistical Package for Social Science for Windows software (SPSS 18, USA). To examine changes after the intervention, an analysis of covariance (ANCOVA) was used to evaluate differences between the two groups for variables that did not violate ANCOVA assumptions. Pre-intervention values were used as covariates. When assumptions were violated, an independent *t* test was conducted on the difference scores. No primary or secondary outcomes were defined. The statistical analysis was considered significant, when the probability level was <0.05. Effect sizes were calculated using partial eta squared (η^2) with values of 0.1, 0.3, and above 0.5 considered to be a small, medium, and large effect. Results are reported as mean and standard deviation of the mean.

Results

Exercise heart rate and power output

The average heart rate during the HIIE training sessions for the exercise group was 160 ± 9 b m^{-1} which corresponded to 88 % of HR peak. The average power output during the HIIE training sessions for the exercise group was 200 ± 10 W which corresponded to 81 % of maximal power output.

Body mass and aerobic power

Compared to controls, the exercise group's body mass was significantly decreased ($P = 0.001$, $\eta^2 = 0.20$) and aerobic power was significantly increased ($P = 0.001$, $\eta^2 = 0.39$) by 15 % for the exercise group (Table 1).

Cardiac response

Resting heart rate was significantly reduced in the exercise compared to the control group after 12 weeks of HIIE ($P = 0.001$, $\eta^2 = 0.37$), whereas stroke volume increased significantly ($P = 0.000$, $\eta^2 = 0.45$) after the intervention. PEP did not show a significant change ($P = 0.142$, $\eta^2 = 0.07$), but LVET significantly increased ($P = 0.003$, $\eta^2 = 0.26$) and PEP/LVET ratio was significantly decreased ($P = 0.014$, $\eta^2 = 0.19$; Table 2).

Blood pressure

Systolic blood pressure (SBP), $P = 0.005$ ($\eta^2 = 0.21$) diastolic blood pressure (DBP), $P = 0.003$ ($\eta^2 = 0.23$) and mean arterial pressure (MAP), $P = 0.001$ ($\eta^2 = 0.27$) were all significantly lower in the exercise group compared to control group after the intervention (Table 3).

FBF and vasodilatory capacity

Resting FBF did not show a significant change after the exercise intervention ($P = 0.092$, $\eta^2 = 0.08$) although it increased in the exercise group. Forearm vascular resistance was significantly lower in the exercise group

Table 1 Changes in body composition and aerobic power after the 12-week intervention (mean and standard deviation)

Variables	Exercise		Control	
	Pre	Post	Pre	Post
Weight (kg)	87.8 ± 11.7	86.3 ± 11.6*	89 ± 12.4	89.4 ± 12.9
BMI (kg m^{-2})	28.4 ± 2.4	27.9 ± 2.4*	29 ± 3.9	29.1 ± 3.8
Waist circumference (cm)	93.3 ± 6.1	89.8 ± 6.4*	93.7 ± 8.0	95.1 ± 8.2
$\dot{V}O_{2max}$ (l min^{-1})	3.0 ± 0.6	3.4 ± 0.6*	2.6 ± 0.5	2.7 ± 0.5
$\dot{V}O_{2max}$ (ml kg^{-1} min^{-1})	34.2 ± 4.4	39.4 ± 3.5*	29.0 ± 5.0	30.4 ± 5.5

BMI body mass index, $\dot{V}O_{2max}$ maximal oxygen uptake

* $P < 0.01$, change greater compared to that of control

Table 2 Changes in cardiac measures after the 12-week intervention (mean and standard deviation)

Variables	Exercise		Control	
	Pre	Post	Pre	Post
HR (b m^{-1})	67.4 ± 9.7	61.2 ± 8.9*	68.9 ± 7.7	70.4 ± 6.7
SV (ml)	77.2 ± 24.9	90.4 ± 26.3*	75.6 ± 18.9	70.2 ± 20.9
PEP (ms)	133.0 ± 12.6	129.2 ± 16.2	126.0 ± 16.3	130.1 ± 9.9
LVET (ms)	271.5 ± 26.5	286.8 ± 26.0*	281.1 ± 18.5	273.4 ± 16.6
PEP/LVET	0.49 ± 0.06	0.45 ± 0.07*	0.45 ± 0.07	0.48 ± 0.05

HR heart rate, *SV* stroke volume, *PEP* pre-ejection period, *LVET* left ventricular ejection time

* $P < 0.05$, change greater compared to that of control

Table 3 Changes in vascular measures after the 12-week intervention (mean and standard deviation)

Variables	Exercise		Control	
	Pre	Post	Pre	Post
AIx (%)	9.8 ± 12.4	6.2 ± 11.0*	7.1 ± 7.2	10.4 ± 6.8
FBF (ml 100 ml tissue^{-1} min^{-1})	1.9 ± 0.8	2.4 ± 0.7	2.1 ± 0.5	2.1 ± 0.7
FVR (mmHg ml^{-1} 100 ml tissue^{-1} min^{-1})	47.6 ± 15.3	36.7 ± 13.2*	39.8 ± 8.8	44.1 ± 13.8
FVR (mmHg ml^{-1} 100 ml tissue^{-1} min^{-1})[a]	4.2 ± 2.0	3.0 ± 0.8*	3.5 ± 1.1	3.6 ± 0.9
SBP (mmHg)	119.6 ± 9.9	115.5 ± 9.7*	117.4 ± 13.4	121.7 ± 12.8
DBP (mmHg)	63.7 ± 7.3	59.2 ± 7.5*	62.2 ± 7.0	65.8 ± 6.6
MAP (mmHg)	83.1 ± 8.2	78.7 ± 8.0*	80.7 ± 8.6	84.3 ± 7.0

AIx augmentation index, *FBF* forearm blood flow, *FVR* forearm vascular resistance, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *MAP* mean arterial pressure

[a] FVR during vasodilatory capacity measurement

* $P < 0.05$, change greater compared to that of control

compared to control group ($P = 0.008$, $\eta^2 = 0.18$; Table 3). Vasodilatory capacity (peak FBF), $P = 0.003$ ($\eta^2 = 0.31$) and FVR were significantly changed in the exercise group ($P = 0.001$, $\eta^2 = 0.34$; Fig. 2; Table 3).

PWV and augmentation index

Pulse wave velocity and AIx were used as indicators of arterial stiffness. PWV was significantly reduced in the exercise group compared to control group after 12 weeks of HIIE ($P = 0.013$, $\eta^2 = 0.21$; Fig. 3). AIx at a heart rate of 75 b m^{-1} was also significantly reduced in the exercise group compared to control group ($P = 0.024$, $\eta^2 = 0.13$; Table 3).

HPV

Participants in the exercise group showed a significant increase in low- ($P = 0.032$, $\eta^2 = 0.32$) and high-frequency power (ln) compared to the control group ($P = 0.014$, $\eta^2 = 0.32$). R–R interval, RMSSD, and pNN50 % were also significantly increased ($P = 0.001$, $\eta^2 = 0.35$; $P = 0.033$, $\eta^2 = 0.14$; $P = 0.019$, $\eta^2 = 0.17$, respectively; Table 4).

BRS

Baroreflex sensitivity was significantly increased for the exercise group after the 12-week intervention compared to the control group ($P = 0.011$, $\eta^2 = 0.17$; Fig. 4).

Discussion

Exercisers compared to controls experienced a significant reduction in heart rate and arterial stiffness, whereas stroke

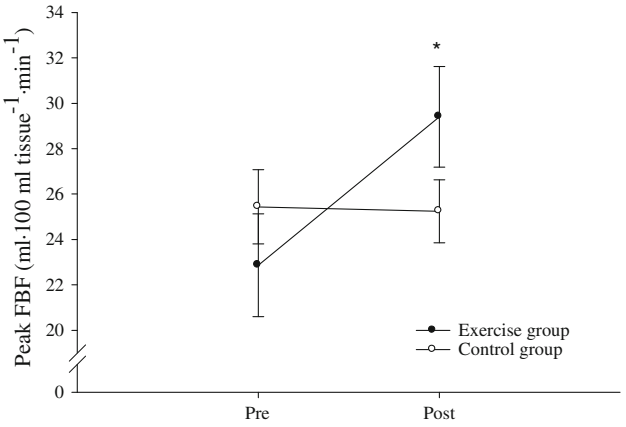

Fig. 2 Peak forearm blood flow change after 12 weeks of high intensity intermittent exercise. *Post relative to pre values in exercise group are significantly different from control group ($P < 0.05$)

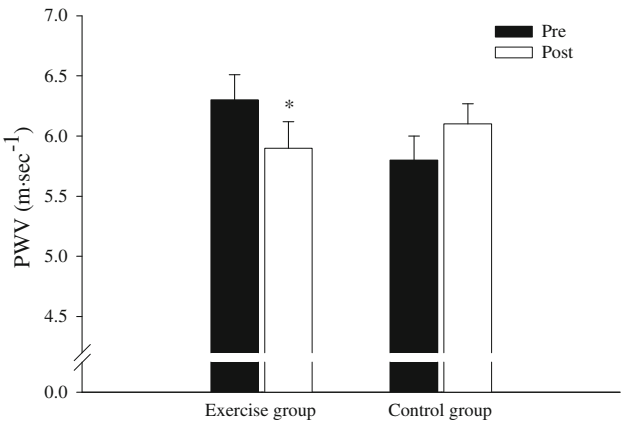

Fig. 3 Pulse wave velocity change after 12 weeks of high intensity intermittent exercise. *Post relative to pre values in exercise group are significantly different from control group ($P < 0.05$)

Table 4 Changes in resting heart period variability after the 12-week intervention (mean and standard deviation)

Variables	Exercise		Control	
	Pre	Post	Pre	Post
TP (au)	34,181 ± 45,536	63,035 ± 1,08,894	48,208 ± 60,290	47,226 ± 66,536
TP (ln)	9.8 ± 1.2	10.0 ± 1.5	10.4 ± 0.8	10.0 ± 1.4
VLF (au)	29,650 ± 39,944	57,958 ± 1,04,643	44,786 ± 59,352	44,238 ± 65,330
VLF (ln)	9.6 ± 1.4	9.7 ± 1.8	10.2 ± 1.0	9.7 ± 1.6
LF (au)	1,956 ± 1,728	2,812 ± 2,424	1,542 ± 941	1,598 ± 1,372
LF (ln)	7.3 ± 0.8	7.7 ± 0.8*	7.1 ± 0.7	7.0 ± 0.9
HF (au)	2,575 ± 5,175	2,265 ± 2,648	1,880 ± 1,887	1,391 ± 1,426
HF (ln)	6.9 ± 1.4	7.2 ± 1.1*	7.1 ± 1.0	6.8 ± 1.1
RMSSD (ms)	67.3 ± 62.0	74.4 ± 42.9*	65.9 ± 35.7	56.6 ± 29.7
PNN50 (%)	15.1 ± 12.4	19.3 ± 10.5*	17.7 ± 9.3	14.8 ± 9.0

TP total power (0.0–0.4 Hz), *VLF* very low frequency power (0.0–0.04 Hz), *LF* low frequency power (0.04–0.15 Hz), *HF* high frequency power (0.15–0.40 Hz), *ln* natural logarithm, *RMSSD* root mean square of successive differences, *pNN50* percentage of interval differences of successive N–N intervals greater than 50 ms

* $P < 0.05$, change greater compared to that of control

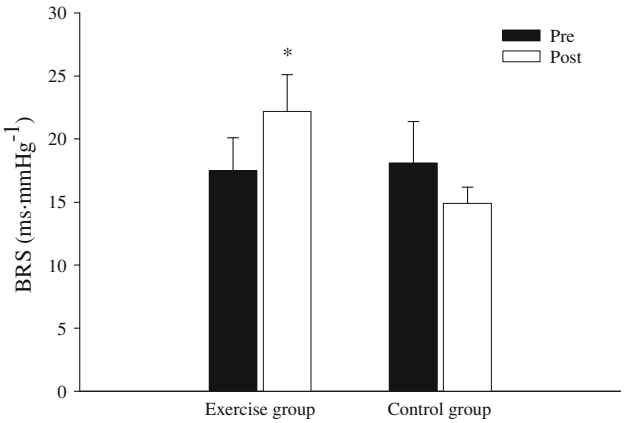

Fig. 4 Baroreflex sensitivity change after 12 weeks of high-intensity intermittent exercise. *Post relative to pre values in exercise group are significantly different from control group ($P < 0.05$)

volume, limb vasodilatory capacity, HPV, and baroreflex sensitivity were significantly increased. Aerobic power also improved significantly by 15 % for the exercise group. Thus, HIIE induced significant cardiac, vascular, and autonomic improvements after 12 weeks of training.

HIIE training resulted in a significant decrease in resting heart rate. These results are similar to the results of numerous longitudinal aerobic exercise studies that have also documented exercise-induced bradycardia [40]. Unfortunately, the mechanism that causes bradycardia has not been identified. It is considered to reflect a combination of reduced intrinsic heart rate, decreased sympathetic tone, and enhanced parasympathetic or vagal tone. Endurance training-induced decrease in intrinsic heart rate has been consistently found by several studies [13], although the increase in vagal influence on the heart found in the present study (discussed later) may have contributed to the decrease in resting heart rate found after HIIE training.

Schairer et al. [30] have demonstrated that the large exercise stroke volume of elite cyclists and runners was primarily influenced by increased preload and to a lesser extent by myocardial contractility. This indicates that, in younger subjects, endurance training may increase cardiac performance by inducing cardiac dilatation during exercise. Such dilatation is also present during rest, as young endurance-trained athletes typically possess significantly larger resting stroke volumes and lower resting heart rates compared to sedentary individuals [30]. These greater stroke volumes at rest could be achieved by increased myocardial contractility, although increased end-diastolic volume appears to be the more likely mechanism [30]. Enhanced end-diastolic volume has been shown to be increased as a result of aerobic exercise-induced blood volume expansion [6].

With regard to the 15 % increase in resting stroke volume found in the present study, the underlying mechanism is undetermined as blood volume change to HIIE was not assessed. However, the significant increase in resting cardiac contractility reflected by the systolic time intervals of LVET and PEP/LVET (Table 2) suggests that increased myocardial contractility may play a role in stroke volume enhancement after HIIE. For example, aerobic exercise training alters the contractile properties of cardiac muscle fibers of exercising rats. Changes include increased sensitivity of cardiac muscle fibers to Ca^{2+} activation and an enhanced cardiac fiber force–length relationship [8]. In addition, it has been shown that increased myocardial Ca^{2+}

sensitivity of steady-state tension accompanies aerobic interval training in mice [14]. As these studies used aerobic training to examine myocardial contractility change future research using interval sprinting as the exercise modality are needed.

The significant increases in vasodilatory capacity found after HIIE training supports the results of long-term aerobic exercise training that has shown enhanced vasodilatory capacity (arteriolar dilation) in exercising muscles. Cross-sectional studies have revealed that aerobically trained individuals possessed greater vasodilatory capacity compared to untrained individuals, whereas longitudinal research [19] found enhanced vasodilatory capacity in calf muscles after 6 months of cycling and jogging in older men and women. In terms of non-exercising muscles, Silber et al. [31] have reported augmentation of vasodilatory capacity in the forearm after 4 weeks of leg cycling exercise, whereas a number of studies have shown vascular function improvement of upper-limb following lower-limb exercise in healthy subjects in both conduit arteries such as brachial and radial [5, 28, 36] and resistance arteries [7, 11, 15]. Thus, the increase in forearm vasodilatory capacity found in the present study also shows that HIIE can increase limb capacity in non-exercising muscles. The mechanisms underlying this phenomenon have not been fully elucidated. The endothelial adaptation in conduit arteries may be attributed to increased shear rate, whereas its effect on resistance arteries is undetermined [12]. Other possible mechanisms influencing forearm vasodilatory adaptation include an increase in endothelial NO synthase (eNOS) and prostaglandin release and a decline in free-radical-mediated NO degradation and sympathetic vasoconstrictor tone [23].

Although longitudinal data examining arterial stiffness and exercise are limited Tanaka et al. [35] assessed arterial stiffness in middle-aged men before and after 3 months of aerobic exercise training and found a reduction in resting arterial stiffness. In addition, other studies have also found lowered arterial stiffness after training in patients with congestive heart failure and in type 2 diabetes. Sugawara and colleagues [34] assessed central arterial stiffness in postmenopausal women before and after 15 weeks of aerobic exercise training at a low and moderate intensity and found the same amount of reduction in arterial stiffness regardless of training intensity. The 37 % decrease in arterial stiffness found in the present study (Fig. 3) extends these results to HIIE.

The increase in HPV, reflecting increased vagal influence on the heart, found after 12 weeks of HIIE training supports the results of prior research with aerobic exercise [29]. In contrast, Sloan et al. [32] showed that autonomic control of the heart did not improve after resistance training. Madden et al. [18] also found similar results in comparing aerobic endurance and resistance training after 6 months in older women. Two studies have examined HPV using aerobic interval training in older physically active males and coronary artery disease patients. It was found that HPV increased after interval training [22, 27]. In contrast, to the aerobic interval training of the prior two studies, the interval exercise of the present study was anaerobic in nature. Thus, these results appear to be the first to show that high-intensity intermittent sprinting accompanied by brief low intensity exercise can significantly increase vagal influence on the heart. HRV was only assessed during spontaneous breathing at rest. That HPV was not also measured during paced breathing at rest is a limitation of this study.

The 12 % increase in arterial BRS (Fig. 4) found after HIIE training supports research examining aerobic exercise that found an increase in arterial BRS following training. Monahan et al. [21] determined arterial BRS before and after a 3 month walking program in sedentary middle aged and older men. They reported an average 25 % increase in arterial BRS. Other studies utilizing aerobic exercise, however, have not reported change.

An inverse relationship between arterial stiffness and BRS has been previously reported [17]. According to Lipman et al. [17] baroreflex sensitivity can be, in part, defined by vascular stiffness. Although the mechanism underlying this relationship is not fully understood, one possible explanation might be that the increased stiffness of the arteries, in which the arterial baroreceptors are located, decreases the engagement of the stretch-sensitive baroreceptors. This may be the reason why a reduction in arterial stiffness after HIIE training was accompanied with an increase in BRS. Future studies are required to test this hypothesis. Arterial baroreflex activity during HIIE, as opposed to rest, should also be examined as it is reset during aerobic exercise in an intensity-dependent manner which enables it to continuously control blood pressure [9].

The improvements in cardiovascular and autonomic indicators following HIIE training are likely to have health implications as low $\dot{V}O_{2max}$ has been shown to predict cardiac disease and high levels of arterial stiffness are associated with increased cardiovascular morbidity and mortality [16]. Furthermore, low vasodilatory capacity and BRS [25] have been linked to hypertension development and reduced HPV is a predictor of a number of cardiac complications [24].

In conclusion, HIIE brought about a significant reduction in heart rate and arterial stiffness, whereas aerobic fitness, stroke volume, limb vasodilatory capacity, HPV, and baroreflex sensitivity were significantly increased. Thus, regular HIIE induced significant cardiac, vascular, and autonomic improvements after 12 weeks.

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Conflict of interest None of the authors had a personal or financial conflict of interest.

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