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Effects of acute exercise programs on heart rate variability and vascular function in sedentary college students

A randomized controlled trial

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Abstract

Background: The specific effects of different exercises on cardiovascular health remain unclear. This study aimed to compare the effects of acute anaerobic threshold intensity continuous exercise (ATE), high-intensity interval training (HIIT), and stepwise incremental exercise (SIE) on heart rate variability (HRV) and vascular endothelial function in sedentary college students.

Methods: Thirty-five sedentary students were randomized to the ATE group (20.6 ± 2.4 years), the HIIT group (21.7 ± 4.2 years), or the SIE group (21.2 ± 2.8 years). Brachial artery flow-mediated dilation was measured at 10 and 30 minutes after exercise. HRV was measured within 35 minutes postexercise.

Results: It showed that the normalized low frequency postexercise was lower than baseline in the ATE group ($P < .05$). Flow-mediated dilation was decreased ($P = .006$) 30 minutes after exercise (6.4 ± 2.2%) compared to baseline (8.1 ± 1.4%) in the HIIT group and increased (+1.4%) in the ATE group with no statistical difference ($P > .05$). Significant correlations were detected between HRV frequency domain indices and brachial artery baseline diameter ($P < .05$).

Conclusion: Acute HIIT impairs vascular function 30 minutes after exercise, whereas acute ATE improves vascular function 10 minutes after exercise. Changes in vascular function may be related to changes in autonomic nervous system activity induced by acute exercise.

Abbreviations: ANS = autonomic nervous system, ATE = anaerobic threshold exercise, CVD = cardiovascular disease, FMD = flow-mediated dilation, HF n.u. = normalized high frequency, HIIT = high-intensity interval training, HR = heart rate, HRV = heart rate variability, LF n.u. = normalized low frequency, LF/HF = the ratio of low frequency and high frequency, MICT = moderate intensity continuous training, NO = nitric oxide, RMSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, SIE = stepwise incremental exercise, VO2max = maximal oxygen uptake.

Keywords: autonomic nervous system, exercise, vascular endothelium

1. Introduction

Sedentary behavior is a significant factor endangering public health. Many studies have shown a positive correlation between prolonged sitting and noncommunicable diseases, such as obesity, cardiovascular disease (CVD), diabetes, and cancer, in populations.[1,2] In China, data indicate that an increasing number of people are exposed to prolonged sitting, regardless of gender and age.[3] Nevertheless, existing research suggests that substituting sedentary behavior with physical activity has a beneficial protective effect on cardiovascular metabolism.[4,5] Considering that endothelial health is a fundamental prerequisite for the normal functioning of the cardiovascular system,[6] the basic process of CVD onset is associated with atherosclerosis.[7] Furthermore, endothelial dysfunction is linked primarily to vascular constriction, prethrombotic formation, and the impact of proinflammatory factors.[8] Therefore, it is essential to pay attention to the endothelial function of sedentary populations and conduct early interventions.

Flow-mediated dilation (FMD) is an endothelium-dependent arterial dilation response that is mediated mainly by nitric oxide

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The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of Beijing Sport University for Sports Science Experiments (2023100H).

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(NO). Peripheral vascular FMD has been shown to be associated with coronary endothelial function and independently predicts cardiovascular CVD outcomes.[9] Studies have confirmed the prognostic value of brachial artery FMD for cardiovascular events. Meta-analyses have indicated that in the long term, for every 1% decrease in brachial artery FMD, there is a 9% to 17% increase in cardiovascular event rates, whereas for every 1% increase in FMD, there is an 8% to 13% decrease in cardiovascular event rates.[10,11] Evidence regarding acute sedentary exposure shows a significant decrease in vascular function in healthy adults.[12] Therefore, strategies for interrupting sedentary behavior and implementing exercise interventions have been proposed internationally because both breaking sedentary behavior and exercise interventions have the potential to improve vascular function.[13–15] However, owing to methodological differences such as exercise protocols and diverse study populations, the findings across related studies are inconsistent.

The acute effects of single exercise sessions provide preliminary insights into the reasons for long-term adaptations. Currently, the correlation between acute changes in vascular function and long-term adaptations is unclear, and the results of FMD changes after single exercise sessions are inconsistent, even completely opposite.[16]

Moderate-intensity continuous training (MICT), high-intensity interval training (HIIT), and stepwise incremental exercise (SIE) are 3 common but different exercise types with essential differences in exercise intensity and mode. When acute effects on FMD are assessed, continuous aerobic exercise is the most common form of exercise, whereas interval exercise is relatively less utilized. SIE gradually approaches the physiological limits of the body and is commonly used in exercise testing but less so as an intervention. In clinical practice, considering different exercise levels is necessary to generate relevant clinical benefits (such as physical fitness, vascular health, etc.) in key clinical outcomes.[17] Although sedentary behavior has a more negative impact on vascular function in healthy young individuals than in adults and elderly individuals with metabolic disorders,[18] few studies have focused on sedentary young people. For sedentary young individuals, exercise interventions are highly important for preventing the decline in endothelial function and/or improving endothelial function.

Furthermore, the influence of the autonomic nervous system on the postexercise FMD response has not been well studied because of the difficulty in directly measuring autonomic nervous system (ANS) activity.[19] Moreover, there is currently a lack of research utilizing heart rate variability (HRV) to explore the correlation between the ANS and changes in vascular endothelial function.

Therefore, the purpose of this study was to compare the acute effects of different exercise protocols, including anaerobic threshold intensity continuous exercise (ATE), HIIT, and SIE, on HRV and vascular endothelial function in sedentary college students and to explore the correlation between acute exercise-induced HRV and changes in vascular endothelial function.

2. Materials and methods

2.1. Study design and participants

This study is a randomized controlled trial. Through simple randomization (drawing lots), and applying the sequentially numbered, opaque, sealed envelope method of allocation concealment, subjects who met the inclusion criteria and did not meet the exclusion criteria were randomly assigned to the ATE group, HIIT group, or SIE group after baseline testing. HRV and brachial artery FMD were measured at baseline and after each exercise. This study was approved by the Ethics Committee of Beijing Sport University for Sports Science Experiments (2023100H) and carried out in accordance with the Declaration of Helsinki. All the participants provided informed consent.

The study participants were students from Beijing Sport University who were recruited through online electronic media and offline poster advertisements. The inclusion criteria were as follows: aged 18 to 30 years and self-reported sedentary time of ≥ 7 hours per day.[20,21] The exclusion criteria were: smoking or cessation of smoking for ≤ 6 months; any existing cardiac or vascular diseases; intake of any medications (or growth hormones) affecting the cardiovascular system; intake of antioxidant vitamin supplements, anti-inflammatory drugs, or steroids; presence of exercise risk as determined by the Physical Activity Readiness Questionnaire (PAR-Q); any injuries affecting exercise; and concurrent participation in other trials. All included participants completed the International Physical Activity Questionnaire short form (IPAQ) to assess their physical activity status, and the data were analyzed and categorized into high and low activity levels according to the respective calculation methods.[22]

An a priori power calculation at the 5% significance level and 80% statistical power determined that a total of 36 participants would be sufficient to detect a small effect size of 0.25 (G-Power 3.1).[23] A participant flow diagram is shown in Figure 1.

2.2. Procedures

After signing the informed consent form, participants proceeded to the formal testing collectively. To ensure authentic test performance, all participants completed a one-week pretest familiarization program with experimental protocols and equipment operation under the supervision of a sports medicine specialist. Upon arrival at the laboratory, participants rested quietly for 5 minutes to stabilize heart rate (HR) before equipment placement and testing initiation. All exercise testing procedures were conducted in the same standardized laboratory environment. Researchers scheduled tests on the basis of participants' availability. Each participant needed to complete 4 tests on different days, including 3 baseline tests and one acute exercise intervention. There was a minimum of 3 days between each test. Before each test, the participants were required to abstain from alcohol and vigorous physical activity for 24 hours and avoid caffeine intake for 12 hours. During testing, the participants were instructed to maintain their current level of physical activity and dietary habits.

For the first test, a bioelectrical impedance testing system (Inbody 720, Seoul, Korea) was used to measure participants' body composition, mainly weight and body fat percentage. The participants fasted for at least 8 h before the measurement and arrived at the laboratory the next morning to complete the measurement. For the second test, an endothelial function testing system (UNEX, EF38G, Nagoya, Japan) was used to measure brachial artery FMD in the supine position at baseline. For the third test, the exercise cardiopulmonary metabolism testing system (Cosmed Quark, Rome, Italy) was used to conduct a maximal oxygen uptake (VO_{2max}) test on a power cycle ergometer, with the test results used as the basis for formulating exercise programs. Before the VO_{2max} test was conducted, baseline HRV was measured in participants at rest. For the fourth test, participants were asked to wear a HR monitor continuously until the examination was completed. They performed a preset exercise program on the power cycle ergometer, and HRV and brachial artery FMD were measured after exercise.

2.3. Brachial artery FMD

The brachial artery FMD was evaluated by endothelial function testing system (UNEX, EF38G) at baseline, 10 minutes after exercise (first cuff inflation), and 30 minutes after exercise (second cuff inflation). Existing literature reports discrepancies in the timing of post-exercise FMD measurements. While some studies measuring FMD have observed a reduction in FMD within 30 minutes post-exercise, significant methodological heterogeneity exists across these

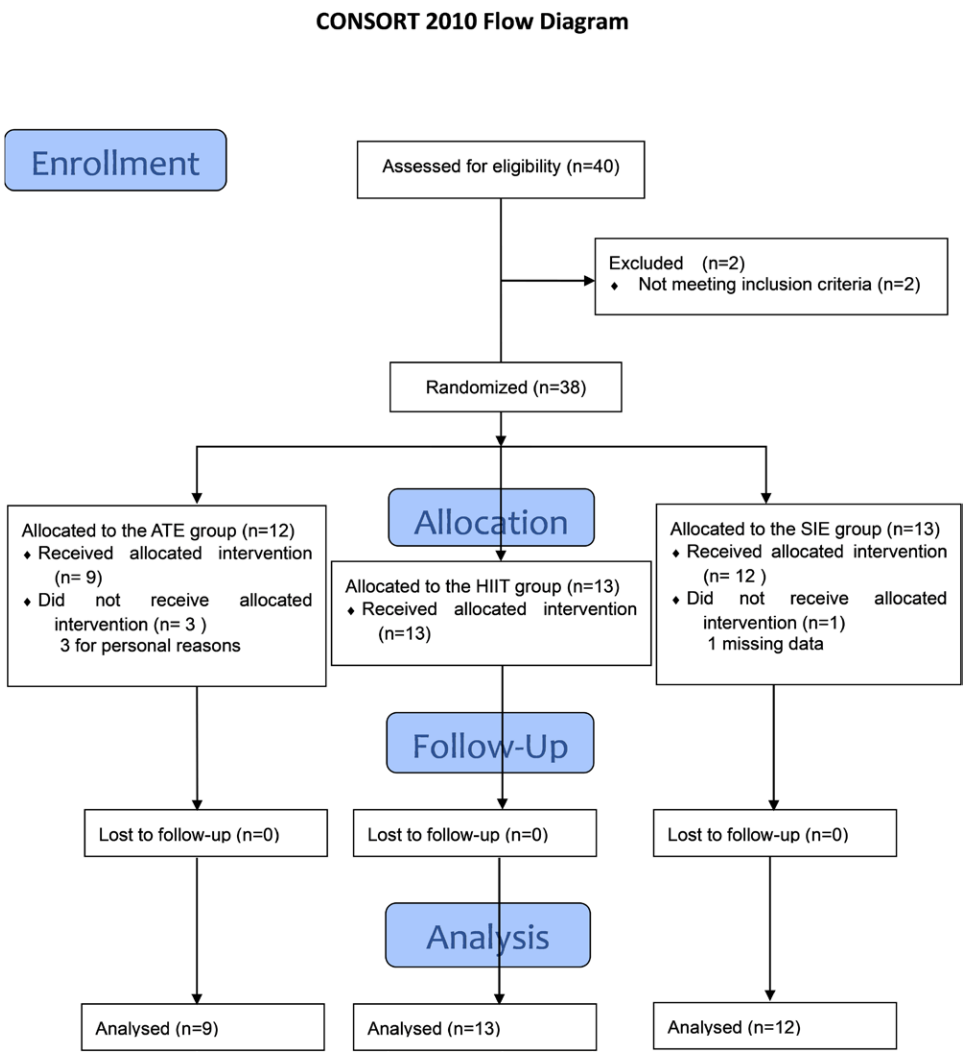

Figure 1. Flow diagram of participants.

investigations.[16] Therefore, the present study adopted the 30-minutes post-exercise time point for FMD assessment. The participants[23] first rested quietly in the supine position for 20 minutes. Initially, a 10 MHz multifrequency linear array probe was used to image the brachial artery at the distal one-third of the upper arm. The ultrasound probe is H-shaped, allowing simultaneous display of two short-axis cross-sectional and one long-axis longitudinal ultrasound images of the brachial artery. After the ultrasound image of the brachial artery was manually positioned, the fine adjustment system of the probe automatically adjusted the probe direction to obtain clearer images of the brachial artery. After the baseline diameter of the brachial artery was determined, the blood pressure of the noninflated side (left side) of the upper arm was measured via an electronic blood pressure cuff. Once the measurements were completed, the cuff on the inflated side (placed 2–3 cm distal to the elbow of the right arm) was rapidly inflated to exceed the resting systolic pressure by more than 50 mm Hg, and inflation was maintained for 5 minutes. After cuff deflation, the cuff pressure was released automatically, and the peak blood flow was measured within the first 60 seconds of reactive hyperemia. Then, digital images of the artery were captured every 5 seconds, from 30 to 120 seconds, to determine the peak dilation. FMD is defined as the percentage change in vessel diameter from baseline to peak dilation, calculated[24] as FMD = (peak diameter minus baseline diameter)/baseline diameter × 100%.

2.4. VO_{2max}

The ramp protocol[25] on a power cycle ergometer (Cosmed Quark) was used for the incremental exercise test. The participants warmed for 3 minutes at 0 W, and then the resistance was gradually increased until the participant reached exhaustion. The wattage increased by 20 W per minute for males and 15 W per minute for females. During exercise, participants must maintain a cycling cadence between 55 to 65 revolutions per minute. The maximum value of the average VO_2 over a continuous 30 seconds interval was recorded as the VO_{2max} result. Before each test, the system underwent gas calibration and metabolic calibration, and testing was conducted at a laboratory with a temperature of 20°C to 22°C and a relative humidity of 50%. The test was terminated when the participant met 3 or more of the following 4 conditions[25,26]: unable to maintain the required cadence range as the exercise load increased; HR reached 90% of the age-related maximum HR (220 - age); respiratory exchange ratio (RER) ≥ 1.15; and rating of perceived exertion (RPE) ≥ 18.

2.5. HRV

A HR monitor (Polar V800, Kempele, Finland) was used to measure the baseline and postexercise HRV. The participants[27] lay quietly in the supine position for 10 minutes to obtain the baseline HRV. The analysis of postexercise HRV included 5 time

periods: immediately after exercise (0–5 minutes), from immediately after exercise to the first cuff inflation (5–10 minutes), during the first cuff inflation (10–15 minutes), from the first to the second cuff inflation (15–30 minutes), and during the second cuff inflation (30–35 minutes). Five-minute segments were taken for HRV analysis at baseline and during post-exercise time period, with the two cuff inflations each lasting exactly 5 minutes. All data analysis was conducted via HRV analysis software (Kubios HRV 3.5.0, Kuopio, Finland). The selected HRV indices include time-domain indices: standard deviation of all NN intervals and the square root of the mean of the sum of the squares of differences between adjacent NN intervals (RMSSD); frequency-domain indices: normalized high frequency (HF n.u.), normalized low frequency (LF n.u.), and the ratio of low frequency to high frequency (LF/HF).

2.6. Exercise protocols

According to the results of the VO_{2max} test, the corresponding VO_2 values for each intensity were converted into power values on the power cycle ergometer to control exercise intensity. The AT intensity was determined using the V-Slope method with a cardiopulmonary exercise testing system. In the AT group, participants exercised continuously for 20 minutes. In the HIIT group, participants exercised for 1 minute at 90% of the VO_{2max} power, followed by 1 minute of active recovery at 50% of the VO_{2max} power, which was repeated for 10 sets, for a total of 20 minutes. In the SIE group, participants exercised for 3 minutes at each of the following power levels: 25 W, 50 W, 75 W, 100 W, and 125 W, for a total of 15 minutes. A 1-minute warm-up at 0 W was performed before each formal exercise session.

2.7. Statistical analysis

The Shapiro–Wilk test was used to assess the normality of all continuous variables. Normally distributed data were expressed as mean ± SD. Count data were presented as percentages, and differences between groups were compared using the chi-square test or Fisher's exact test. The Levene test was used to assess the homogeneity of the variance in the data. Two-way repeated measures analysis of variance (ANOVA) was used to compare differences in HRV and brachial artery FMD between different time points and intervention groups. post hoc multiple comparisons were conducted via LSD method. When no interaction effect was present, paired-samples *t* tests were used to compare differences in HRV and brachial artery FMD between different time points. Pearson correlation analysis was used to examine the correlation between brachial artery baseline diameter, HRV, and FMD at different time points. All analyses were performed using SPSS 26.0 (IBM Corp., New York), with the significance level set at $P < .05$.

3. Results

Of the 40 subjects recruited, 38 underwent randomization, with 4 excluded from data analyses because of personal reasons or missing data and 34 subjects were included in data analyses. The subjects were mostly female (70%, 69%, and 83%) with mean ages of 20.6 ± 2.4, 21.7 ± 4.2, and 21.2 ± 2.8 years, respectively. The baseline characteristics of the participants are presented in Table 1. Mean HRs during acute ATE, HIIT, and SIE bouts were 142 ± 17, 140 ± 14, and 128 ± 15 bpm, respectively, indicating that exercise intensities were maintained within the prescribed target zones. The parameter characteristics of the exercise sessions are displayed in Table 2.

3.1. HRV

HRV was impaired after acute HIIT and improved immediately after ATE, as indicated by the LF n.u ($P < .05$). Acute SIE did not affect HRV. The results are presented in Table 3, Figures 2 and 3.

Standard deviation of all NN intervals exhibited a significant time effect ($P = .000$), but no group effect ($P = .703$) or interaction effect ($P = .837$). The results of the time effect showed that there were statistically significant differences between each post-exercise time period and the baseline ($P < .05$).

RMSSD also showed a significant time effect ($P = .000$), but no group effect ($P = .853$) or interaction effect ($P = .095$). The results of the time effect showed that there were statistically significant differences between each post-exercise time period and the baseline ($P < .05$).

LF n.u. had significant effects on time ($P = .001$), group ($P = .033$), and interaction ($P = .049$). Simple effects analysis of time revealed that in the AT group, the LF n.u. was lower at 10 to 15 minutes (first cuff inflation) ($P = .025$), 15 to 30 minutes ($P = .038$), and 30 to 35 minutes (second cuff inflation) ($P = .010$) postexercise than that at baseline. In the HIIT group, LF n.u. was greater immediately after exercise ($P = .024$) and 5 to 10 minutes ($P = .033$) postexercise compared to baseline. In the SIE group, there were no differences between each post-exercise time point and baseline ($P > .05$). Simple effects analysis of the intervention showed that LF n.u. in the AT group was lower than the other 2 groups at each post-exercise time point ($P < .05$), whereas there were no differences between the other 2 groups ($P > .05$).

HF n.u. showed significant effects of time ($P = .000$) and group ($P = .032$), but no interaction effect ($P = .054$). The results of the time effect showed that there were no differences between

Table 1

Baseline characteristics.

	ATE (n = 10)	HIIT (n = 13)	SIE (n = 12)
Age (year)	20.6 ± 2.4	21.7 ± 4.2	21.2 ± 2.8
Female (%)	70	69	83
Height (cm)	173.0 ± 7.8	167.5 ± 7.0	165.5 ± 7.5
Weight (kg)	64.0 ± 14.7	64.1 ± 13.1	62.5 ± 12.9
BMI (kg/m^2)	21.2 ± 3.9	22.8 ± 4.3	22.7 ± 4.1
Body fat (%)	22.4 ± 8.9	27.8 ± 8.1	29.4 ± 8.7
VO_{2max} (mL/kg/min)	33.7 ± 5.2	29.5 ± 5.6	28.2 ± 5.5
Brachial artery baseline diameter (mm)	3.64 ± 0.52	3.49 ± 0.60	3.36 ± 0.51
Brachial artery FMD (%)	7.6 ± 1.4	8.1 ± 1.4	7.8 ± 1.5
Physically active (%)	70	46	58

Results are presented as mean ± SD.

ATE = anaerobic threshold intensity continuous exercise, BMI = body mass index, FMD = flow-mediated dilation, HIIT = high-intensity interval training, SIE = stepwise incremental exercise, VO_{2max} = maximal oxygen uptake.

Table 2

Exercise parameters of exercise sessions.

	ATE (n = 10)	HIIT (n = 13)	SIE (n = 12)
$\%VO_{2max}$ (%)	69 ± 8	N/A	93
VO_2 (mL/kg/min)	24.0 ± 5.4	N/A	26.3 ± 2.8
Power (w)	94 ± 41	N/A	N/A
HR (bpm)	142 ± 17	140 ± 14	128 ± 15
$50\%VO_{2max}$ (mL/kg/min)	N/A	14.7 ± 2.8	N/A
$90\%VO_{2max}$ (mL/kg/min)	N/A	26.5 ± 5.0	N/A
Power at $50\%VO_{2max}$ (w)	N/A	45 ± 19	N/A
Power at $90\%VO_{2max}$ (w)	N/A	113 ± 37	N/A

Results are presented as mean ± SD.

ATE = anaerobic threshold intensity continuous exercise, HIIT = high-intensity interval training, HR = heart rate, SIE = stepwise incremental exercise, VO_2 = oxygen uptake, VO_{2max} = maximal oxygen uptake.

Table 3

Effects of a single bout of exercise on HRV.

		Baseline	0–5′	5–10′	10–15′	15–30′	30–35′	Group	Time	Group × time
SDNN (ms)	ATE	80.17 ± 103.61	26.58 ± 16.35	32.44 ± 21.23	33.69 ± 19.24	36.20 ± 17.59	41.94 ± 21.68			
	HIIT	111.54 ± 134.20	19.92 ± 7.67	23.97 ± 12.13	43.22 ± 74.24	38.12 ± 20.40	66.47 ± 91.24	0.703	0.000	0.837
	SIE	114.26 ± 137.74	18.57 ± 14.52	19.82 ± 17.93	34.88 ± 62.54	31.33 ± 28.17	30.81 ± 18.70			
RMSSD (ms)	ATE	95.79 ± 151.48	30.41 ± 24.64	37.98 ± 32.58	40.93 ± 29.75	41.21 ± 24.75	49.14 ± 30.43			
	HIIT	135.28 ± 169.13	16.66 ± 7.84	21.99 ± 13.88	51.57 ± 106.21	36.15 ± 28.51	79.25 ± 130.92	0.853	0.001	0.905
	SIE	138.07 ± 173.07	19.61 ± 19.69	20.80 ± 25.28	43.26 ± 92.25	34.03 ± 41.04	34.25 ± 27.29			
LF n.u.	ATE	61.32 ± 20.65	53.73 ± 23.53	54.62 ± 27.42	43.70 ± 26.11	47.07 ± 24.57	42.38 ± 14.53			
	HIIT	55.49 ± 19.27	77.84 ± 10.01	72.49 ± 14.48	65.84 ± 17.65	65.56 ± 18.91	62.48 ± 18.35	0.033	0.001	0.049
	SIE	66.12 ± 15.59	75.28 ± 18.20	67.39 ± 17.21	68.64 ± 21.65	60.02 ± 18.22	56.76 ± 21.07			
HF n.u.	ATE	38.61 ± 20.57	46.04 ± 23.37	45.51 ± 27.35	56.22 ± 26.09	52.85 ± 24.53	57.57 ± 14.54			
	HIIT	44.27 ± 19.15	22.07 ± 9.98	27.41 ± 14.44	34.06 ± 17.64	34.42 ± 18.90	37.46 ± 18.31	0.032	0.000	0.054
	SIE	33.76 ± 15.51	24.64 ± 18.19	32.28 ± 17.26	31.29 ± 21.69	39.89 ± 18.28	43.08 ± 21.02			
LF/HF	ATE	2.72 ± 2.85	2.42 ± 2.67	2.42 ± 2.67	1.39 ± 1.65	1.62 ± 1.93	0.82 ± 0.38			
	HIIT	1.80 ± 1.49	3.79 ± 2.69	3.79 ± 2.69	4.39 ± 7.15	3.57 ± 3.94	2.64 ± 2.35	0.198	0.007	0.126
	SIE	2.91 ± 2.51	3.22 ± 2.74	3.22 ± 2.74	4.09 ± 3.91	1.92 ± 1.67	1.97 ± 1.67			

Results are presented as mean ± SD.

ATE = anaerobic threshold intensity continuous exercise, HF n.u. = normalized high frequency, HIIT = high-intensity interval training, HRV = heart rate variability, LF n.u. = normalized low frequency, LF/HF = the ratio of low frequency and high frequency, RMSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, SDNN = standard deviation of all NN intervals, SIE = stepwise incremental exercise.

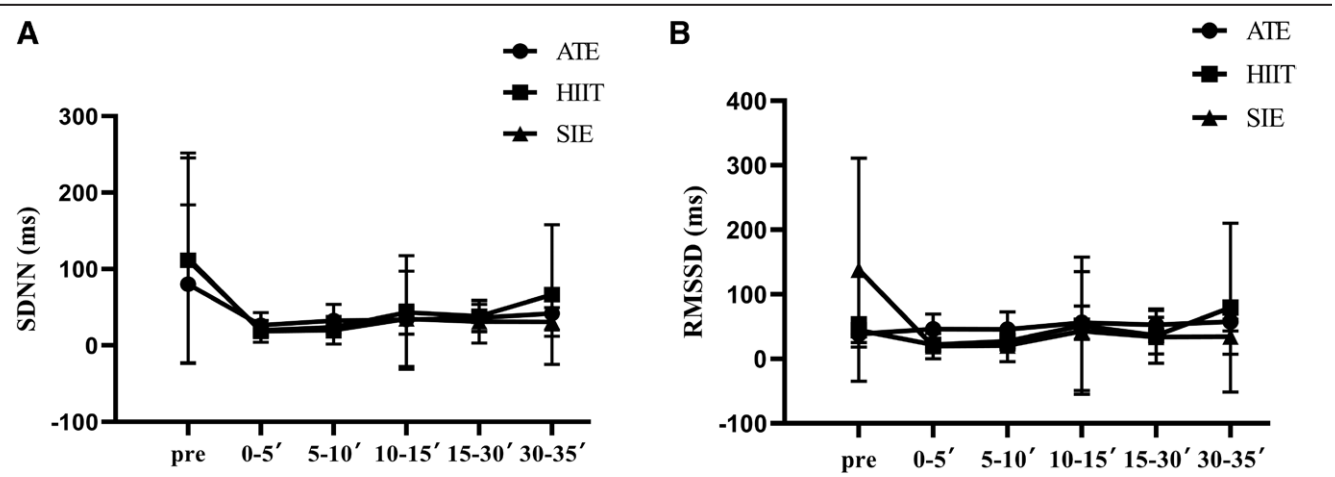

Figure 2. Time-domain indices of HRV before and after acute exercises. Mean ± SD. ATE = anaerobic threshold intensity continuous exercise, HIIT = high-intensity interval training, RMSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, SDNN = standard deviation of all NN intervals, SIE = stepwise incremental exercise.

each post-exercise time period and baseline ($P > .05$). The effect of the intervention showed that HF n.u. in the AT group was greater than that in the HIIT group and the SIE group ($P < .05$), whereas there was no significant difference between the HIIT group and the SIE group ($P = .884$).

LF/HF showed a significant time effect ($P = .007$) but no group effect ($P = .198$) or interaction effect ($P = .126$). The results of the time effect showed that LF/HF was higher only 0 to 5 minutes postexercise compared to baseline ($P = .050$).

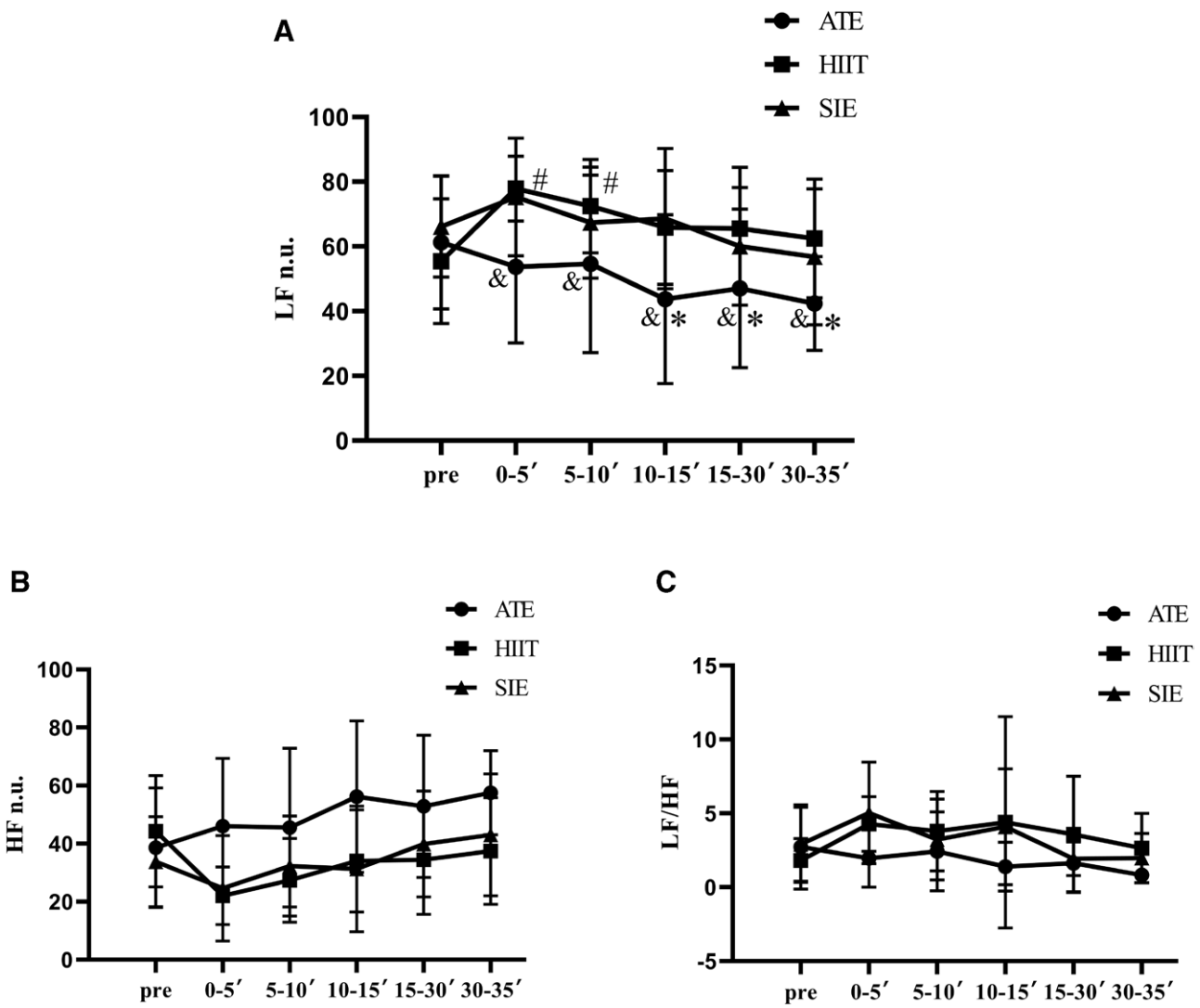

Figure 3. Frequency-domain indices of HRV before and after acute exercises. Mean ± SD. *Statistical difference to pre in the ATE group ($P < .05$); #Statistical difference to pre in the HIIT group ($P < .05$); &Statistical difference between the ATE group and the HIIT and SIE group ($P < .05$). ATE = anaerobic threshold intensity continuous exercise, HIIT = high-intensity interval training, HF n.u. = normalized high frequency, LF/HF = the ratio of low frequency and high frequency, LF n.u. = normalized low frequency, SIE = stepwise incremental exercise.

3.2. Brachial artery baseline diameter and FMD

FMD was improved 10 minutes after acute AT and decreased 30 minutes post-HIIT ($P < .05$) compared with baseline. SIE had no significant effect on FMD. The results of the brachial artery baseline diameter and FMD before and after exercise are presented in Table 4 and Figure 4.

Baseline diameter showed a significant time effect ($P = .000$), but no group effect ($P = .809$) or interaction effect ($P = .054$). The results of the time effect showed that the baseline diameter at 10 and 30 minutes after exercise was higher compared to baseline ($P = .001$).

FMD showed a significant time effect ($P = .048$), but no group effect ($P = .871$) or interaction effect ($P = .564$). The time effect showed that FMD at 30 minutes after exercise was lower than baseline ($P = .033$). The paired-sample t test indicated that FMD was lower only at 30 minutes after exercise in the HIIT group (6.4 ± 2.2%) compared to baseline (8.1 ± 1.4%, $P = .006$), whereas there were no significant differences between each time point and baseline in the other 2 groups ($P > .05$).

Individual variability in FMD response to exercise (Fig. 5): There were no significant differences in the rate of increase in FMD at 10 minutes after exercise among the 3 groups (ATE group: 55.6%, HIIT group: 38.5%, and SIE group: 41.7%, $P = .713$); similarly, there were no differences in the rate of increase in FMD at 30 minutes after exercise among the 3 groups (ATE group: 55.6%, HIIT group: 30.8%, and SIE group: 25%, $P = .346$).

3.3. Correlation analysis of HRV, brachial artery baseline diameter, and FMD

Pearson correlation analysis revealed that LF n.u. ($r_1 = 0.375$, 95% CI 0.04–0.63, $P = .022$; $r_2 = 0.048$, 95% CI −0.28 to 0.37, $P = .785$) and LF/HF ($r_1 = 0.492$, 95% CI 0.21–0.69, $P = .002$; $r_2 = 0.398$, 95% CI 0.07–0.64, $P = .016$) were positively correlated with the baseline diameter at the first and second inflations, whereas HF n.u. ($r_1 = -0.375$, 95% CI −0.63 to −0.04, $P = .022$; $r_2 = -0.407$, 95% CI −0.65 to −0.08, $P = .012$) was negatively correlated with the baseline diameter, indicating that as LF n.u. and LF/HF increased, the brachial artery baseline diameter also increased, and as HF n.u. increased, the brachial artery baseline diameter decreased (Table 5).

Table 4

Effects of a single bout of exercise on brachial artery baseline diameter and FMD.

		ATE	HIIT	SIE	Group	Time	Group × time
FMD (%)	Baseline	7.6 ± 1.4	8.1 ± 1.4	7.8 ± 1.5			
	10 min post-exercise	9.0 ± 3.4	8.5 ± 3.5	7.6 ± 2.7	0.871	0.048	0.564
	30 min post-exercise	7.1 ± 2.1	6.4 ± 2.2	7.2 ± 3.3			
Baseline diameter (mm)	Baseline	3.64 ± 0.52	3.58 ± 0.54	3.36 ± 0.51			
	10 min post-exercise	3.65 ± 0.70	3.88 ± 0.67	3.74 ± 0.53	0.809	0.000	0.054
	30 min post-exercise	3.71 ± 0.62	3.77 ± 0.59	3.69 ± 0.55			

Results are presented as mean ± SD.

ATE = anaerobic threshold intensity continuous exercise, FMD = flow-mediated dilation, HIIT = high-intensity interval training, SIE = stepwise incremental exercise.

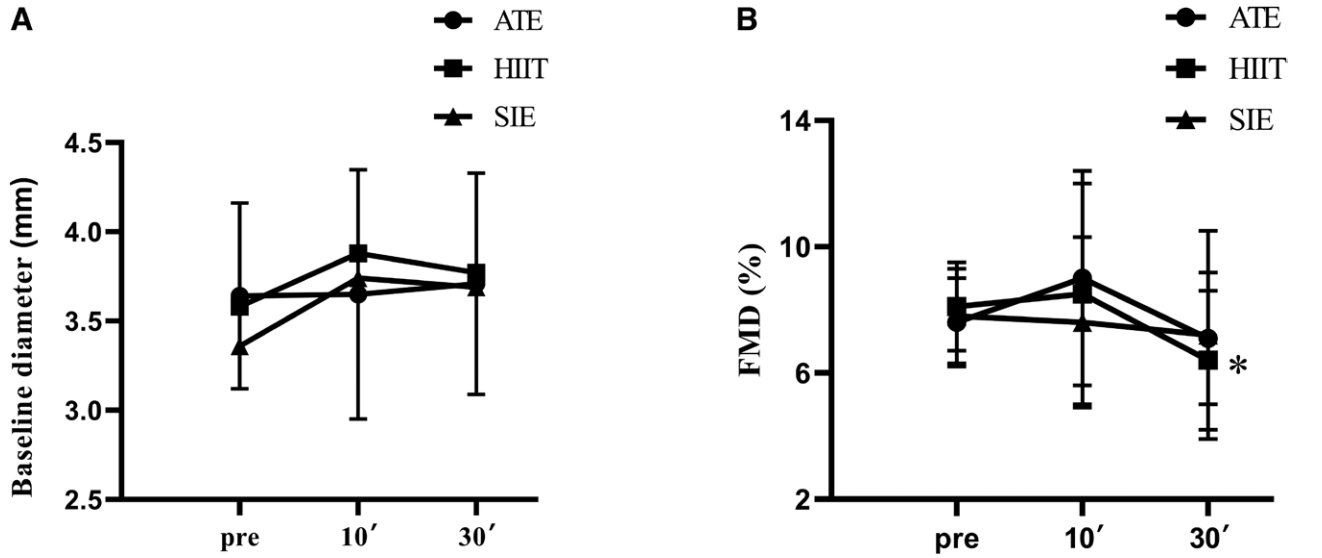

Figure 4. Brachial artery baseline diameter and FMD before and after acute exercises. Mean ± SD. *Statistical difference to pre in the HIIT group ($P < .05$). FMD = flow-mediated dilation.

4. Discussion

The rationale of the present work was to examine the effects of acute ATE, HIIT, and SIE sessions on HRV and brachial artery FMD in sedentary participants. Our major finding was that ATE improved HRV and FMD immediately after exercise, however, HIIT showed the opposite.

4.1. The effect of single exercise sessions on the HRV

A single session of HIIT impaired post-exercise HRV, whereas AT exercise improved post-exercise HRV, and SIE had no significant effect on HRV.

The trend of impaired HRV following acute exercise was consistent with the findings of the majority of previous studies, indicating a decline in function immediately after exercise and incomplete recovery in the short term. These findings suggest the activation of the sympathetic nervous system and the inhibition of the parasympathetic nervous system. Research has shown that the recovery rate after high-intensity exercise is lower than that after moderate or low-intensity exercise.[27] An impaired HRV after high-intensity exercise has also been reported in experienced runners.[28] In this study, HIIT acutely impaired HRV, suggesting immediate sympathetic activation postexercise. However, SIE did not affect the HRV. This may be related to the higher average exercise intensity of HIIT, even though the maximum exercise intensity reached during SIE was greater. Notably, AT exercise reduced post-exercise LF n.u. values, indicating a reduction in post-exercise sympathetic activity, and this trend persisted for up to 35 minutes after exercise. This may suggest a protective effect of low-intensity continuous exercise on the cardiovascular system. Increased sympathetic activity after exercise have been correlated with the risk of cardiac events and cardiovascular mortality (including sudden death).[29] A review evaluating stress-induced HRV responses found no studies reporting improvements in HRV after exercise compared with control groups or pre-exercise levels. However, these studies were methodologically inconsistent, with some analyzing frequency-domain indices and others using time-domain indices.[30] Therefore, further investigation into the phenomenon of improved HRV following low-intensity AT exercise is warranted.

Additionally, a study recruited untrained postmenopausal women and reported impaired HRV (mainly LF/HF) 60 minutes after a single session of HIIT at 90% of VO_{2max} on a treadmill.[31] However, this study was conducted in sedentary healthy students using a low-volume HIIT protocol, resulting in transient HRV impairment. These observations indicate attenuated cardiovascular responses to low-volume acute exercise in this population.

4.2. The effect of single exercise sessions on brachial artery FMD

This study revealed that, compared with baseline, AT exercise improved FMD at 10 minutes postexercise, HIIT decreased FMD at 30 minutes postexercise, and SIE had no significant effect on FMD.

Studies demonstrate a biphasic brachial artery FMD response to acute exercise: an initial decline followed by progressive recovery to baseline, with FMD remaining reduced within 1 hour post-exercise and may take 24 to 48 hours to

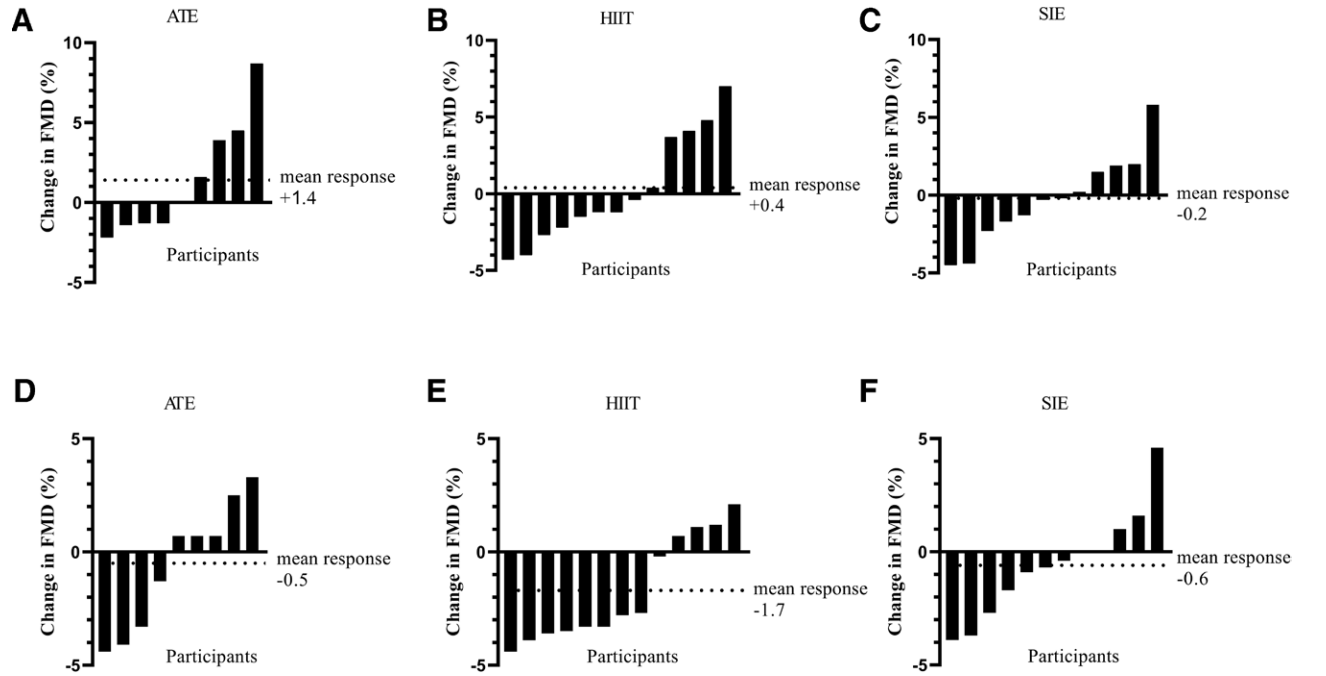

Figure 5. Individual FMD response after acute exercises. 10 min after exercises (A–C), 30 min after exercises (D–F). Dotted lines indicate the participants mean change in FMD with exercise. ATE = anaerobic threshold intensity continuous exercise, FMD = flow-mediated dilation, HIIT = high-intensity interval training, SIE = stepwise incremental exercise.

Table 5

Pearson's correlation coefficient between brachial artery diameter, HRV, and FMD.

	Baseline		First inflation		Second inflation	
	Baseline diameter	FMD	Baseline diameter	FMD	Baseline diameter	FMD
Baseline diameter	1	−0.049	1	−0.369*	1	−0.206
FMD	−0.049	1	−0.369*	1	−0.206	1
SDNN	−0.118	−0.021	−0.073	−0.217	−0.171	−0.211
RMSSD	−0.112	−0.007	−0.09	−0.191	−0.207	−0.182
LF n.u.	0.056	−0.022	0.375*	−0.099	0.048*	−0.037
HF n.u.	−0.054	0.027	−0.375*	0.098	−0.407*	0.033
LF/HF	0.3	−0.114	0.492**	0.019	0.398*	−0.11

Results are presented as mean ± SD.

FMD = flow-mediated dilation, HF n.u. = normalized high frequency, HRV = heart rate variability, LF n.u. = normalized low frequency, LF/HF = the ratio of low frequency and high frequency, RMSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, SDNN = standard deviation of all NN intervals.

*$P < .05$;

**$P < .01$.

fully recover.[16] The current literature exhibits inconsistencies in reported post-exercise FMD responses, primarily attributable to methodological heterogeneity across investigations. Critical confounders, including exercise intensity, duration, volume, mode, participant characteristics, timing of FMD measurements, accuracy of instruments and testers, may account for the observed discrepancies in the current literature.

Exercise intensity is an important influencing factor.[32] A study recruited 10 healthy males (mean age 22 years, self-reported weekly physical activity ≤ 2 hours) to perform 30 minutes of cycling exercise at different intensities of maximum HR, and assessed brachial artery FMD. The results showed that all intensities led to an immediate decrease in FMD, with the greatest decrease observed at 85% of the maximum HR and the smallest at 50% of the maximum HR. Even covariates were controlled, the data still indicated a negative correlation between exercise intensity and FMD. The authors discussed that while vascular adaptation to exercise training may rely on repeated increases in shear stress, oxidative stress induced by higher-intensity exercise may counteract the increase in shear stress.[33] Specifically, high-intensity exercise may impair endothelium-dependent vasodilation due to increased reactive oxygen species, leading to decreased bioavailability of NO.[34] It is worth noting that this study only measured FMD at a single time point immediately after exercise and included only male participants. A crossover trial recruited 20 healthy males (mean age 22.6 years) performing either MICT (exercise at 50% of VO_{2max}) or HIIT (30 sec of exercise at 100% of VO_{2max} followed by 30 seconds of passive recovery), and measured brachial artery FMD immediately after exercise. The results showed that both protocols enhanced post-exercise brachial artery FMD, with HIIT demonstrating superior yet statistically comparable improvements.[35] Other studies that maintained equivalent energy expenditure during exercise showed that both MICT and HIIT, regardless of exercise intensity and mode, increased brachial artery FMD.[24]

Compared with those in previous studies, the subjects in our study were sedentary healthy college students. The results

showed that the AT group increased FMD by 1.4% after 10 minutes of exercise. Although the improvement was not statistically significant, it had clinical significance. On the other hand, the HIIT group presented a relative decrease of 1.7% in FMD compared with baseline after 30 minutes of exercise, which was also a clinically significant decrease that may lead to adverse clinical outcomes.[36,37] The improvement in FMD in the AT group aligns with the exercise intensity-dependent FMD response, suggesting low-dose stimuli may be beneficial for increasing brachial artery FMD in young healthy college students. However, few studies have used exercise protocols that target AT intensity. Notably, the results of multiple comparisons showed no significant differences in FMD values at different time points among the groups, which may be due to an insufficient sample size. Considering the individual variability in participants' responses to single exercise sessions, the AT group showed a trend toward increasing FMD, whereas the HIIT group showed a trend toward decreasing FMD. Therefore, this study provides exploratory directions. In addition, the population of this study consisted mostly of females; accordingly, it should be prudent to generalize the results of this study to other populations, and future studies should focus on different sex components to explore how sex differences affect the changes in FMD.

Owing to the complex factors affecting FMD, including oxidative stress, brachial artery baseline diameter, vascular shear stress, sympathetic nervous system activity, and inflammatory factors, it is impossible to measure these factors in a single study. Increased oxidative stress induced by acute exercise ultimately leads to reduced production and bioavailability of NO. Acute exercise also increases blood flow and shear stress, stimulating the production of endothelial NO.[38,39] Different exercise modes lead to different shear rate patterns.[40] A study has shown that an increase in retrograde shear rate in the first 10 minutes after moderate-intensity exercise is associated with a decrease in FMD, followed by normalization.[41] This study did not measure changes in the vascular shear rate, so only speculation can be provided.

Some studies have reported a strong negative correlation between baseline artery diameter and FMD, indicating a lower dilation capacity in larger arteries than in smaller arteries.[42,43] In this study, a significant negative correlation between brachial artery baseline diameter and FMD was observed only10 minutes after exercise. The fact that the baseline diameter and FMD are not always negatively correlated reflects the complexity of the factors influencing FMD. This study further examined the correlations among the ANS, brachial artery baseline diameter, and FMD. The results showed that during the cuff inflation periods, the HRV frequency domain indices were significantly correlated with the baseline diameter. Specifically, LF and LF/HF were positively correlated with the baseline diameter, whereas HF was negatively correlated with the baseline diameter. This suggests that sympathetic nervous system activation may lead to a decrease in FMD by increasing the brachial artery baseline diameter, although this was not observed at all time points. However, it reflects, to some extent, the impact of an imbalance in ANS activation on FMD. The mechanism by which increased sympathetic activity decreases FMD is not fully understood, but it may be related to increased baseline diameter, reduced availability of NO, and/or decreased brachial artery shear stimulation. A study measured changes in brachial artery FMD in 11 healthy men under different sympathetic nervous system activation states while controlling for shear force stimulation. The results showed that sympathetic activation does not always lead to a decrease in FMD and may even improve it.[44]

4.3. Limitations

The sample size of this current study needs to be further expanded due to statistical considerations. Although sex distribution in this study was similarly proportioned but still uneven, which may introduce confounding effects on primary outcomes. Furthermore, we did not measure shear rate in this study, future investigations should incorporate temporal dynamics of shear rate variation during and after exercise. Finally, future studies should monitor the long-term effects of changes in endothelial function with exercise interventions across diverse populations and determine the minimal clinical important difference.

5. Conclusions

The present study showed that a single bout of AT exercise improved HRV by increasing parasympathetic nervous system activity. A single session of AT exercise improved vascular endothelial function 10 minutes after exercise, wherea a single session of HIIT impaired vascular endothelial function 30 minutes after exercise. The changes in vascular endothelial function were related to the changes in ANS activity induced by single exercise sessions.

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