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The effects of high-intensity interval training and moderate-intensity continuous training on visceral fat and carotid hemodynamics parameters in obese adults

Wenxia Shi [a], Jiangang Chen [a], Yufeng He [a], Pei Su [a], Mengyue Wang [a], Xulong Li [b], Donghui Tang [a,*]

[a] Department of College of P.E. and Sport, Beijing Normal University, No. 19, Xinjiekouwai St, Haidian District, Beijing, 100875, People's Republic of China
[b] Department of P.E., Qingdao University of Technology, No. 777 Jialingjiang Road, Huangdao District, Qingdao, 266520, People's Republic of China

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ABSTRACT

Objectives: The present study aimed to examine the effects of high-intensity interval training (HIIT) and moderate-intensity continuous training (MICT) on visceral fat and hemodynamic parameters in obese adults.

Methods: Fifty-two males were included in this study and divided into three groups: HIIT group (n = 21, age = 20.86 ± 1.62 years, BF (%) = 30.10 ± 5.02), MICT group (n = 22, age = 20.76 ± 1.14 years, BF (%) = 30.19 ± 5.76), and control group (CON) (n = 9, age = 21.38 ± 1.77 years, BF (%) = 30.40 ± 5.10). The HIIT and MICT groups received the exercise intervention three to four times per week for eight weeks (HIIT: exercise intensity 80–95% HRmax, circuit; MICT: exercise intensity 60–70% HRmax, running), and the control (CON) group received health education and guidance without exercise intervention. The body compositions and serum lipid indexes were tested to calculated LAP and VAI. The color doppler ultrasound diagnostic technology was used to test the artery diameter and blood velocity before and after the intervention. Based on the test data, MATLAB software and Womersley theory were used to calculate the hemodynamic parameters of the common carotid artery, including wall shear stress, flow rate, blood pressure, oscillatory shear index, elasticity modulus, dynamic resistance, artery diameter, arterial stiffness, circumferential strain and pulsatility index.

Results: We found that lipid accumulation product (LAP) was significantly decreased in both the HIIT group ($p < 0.01$) and MICT ($p < 0.05$) group but not in the CON group ($p > 0.05$). In contrast, visceral adiposity index (VAI) decreased in both the HIIT and MICT groups and increased in the CON group, although the difference among groups was not significant ($p > 0.05$). After 8 weeks of intervention, the blood velocity and wall shear stress were greater after HIIT and MICT intervention ($p < 0.01$). Artery diameter, oscillatory shear index, arterial stiffness, and pulsatility index decreased significantly, and circumferential strain increased significantly in the HIIT group (all, $p < 0.01$, $p < 0.05$) but not in the MICT group ($p > 0.05$). Dynamic resistance was significantly decreased in the MICT group. There was no difference in the CON group after the period of intervention (all, $p > 0.05$). LAP was positively related to artery diameter (r = 0.48, $p = 0.011$), blood pressure (r = 0.46, $p = 0.002$), flow rate (r = 0.31, $p = 0.04$), oscillatory shear index (r = 0.44, $p = 0.03$), and elasticity modulus (r = 0.33, $p = 0.029$) but inversely related to circumferential strain (r = −0.36, $p = 0.028$). The VAI was also positively associated with artery diameter (r = 0.33, $p = 0.03$), elasticity modulus (r = 0.38, $p = 0.009$), and arterial stiffness (r = 0.39, $p = 0.012$). In addition, the VAI was negatively correlated with the circumferential strain (r = −0.33, $p = 0.04$).

Conclusion: The present study demonstrated that both HIIT and MICT exercises for 8 weeks could effectively enhance visceral fat indices and partial hemodynamic parameters. Therefore, HIIT and MICT exert important effects on reducing fat content and improving hemodynamic environment. But HIIT on oscillatory shear index, arterial stiffness, circumferential strain, and pulsatility index was superior to

* Corresponding author.
E-mail address: tdh1964@bnu.edu.cn (D. Tang).

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MICT. In addition, there are close correlations between visceral fat and partial hemodynamic parameters of the common carotid artery.

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1. Introduction

During the past several decades, the prevalence of obesity worldwide among adults has risen sharply, which might be partially explained by an imbalance between energy intake and energy expenditure.[1] Compared with those with subcutaneous obesity, people with visceral obesity are more likely to develop diabetes, hyperlipidaemia, hypertension, and hyperuricaemia.[2] It is well known that visceral fat accumulation is related to cardiometabolic risk factors, which might lead to a series of lesions in the vascular system in obese individuals and an increased risk of cardio-cerebrovascular diseases.[3] Anthropometric parameters of general and central adiposity have become the most frequently used surrogate predictors, including body mass index (BMI), waist circumference (WC), and body fat percentage [BF (%)], but these indices commonly used in clinical practice cannot accurately reflect the degree of body fat accumulation. Currently, medical imaging techniques such as Dual-Energy X-ray Absorptiometry (DEXA), Computerized Tomography (CT) and Magnetic Resonance Imaging (MRI) can accurately assess visceral fat content. Lipid accumulation product (LAP) and visceral adiposity index (VAI), based on BMI, WC, and lipid indexes, can be also used to evaluate the individual fat distribution and visceral fat accumulation, which are emerging as valuable indicators for visceral obesity. Study confirmed a linear correlation between LAP, VAI visceral fat index and MRI, which could indirectly reflect visceral fat.[4–6] Compared with these medical imaging techniques, LAP and VAI are easier to implement and more suitable for screening. The LAP represents a clinical index based on the assessment of WC and triglycerides (TG), which estimates visceral lipid accumulation. The VAI is a simple, sex-specific marker combining anthropometric data and lipid profiles and is also a reliable indicator of visceral dysfunction, which makes up for the weakness of waist circumference.[7] Similarly, recent research has demonstrated that LAP and VAI are significantly positively correlated with metabolic syndrome and atherosclerotic cardiovascular disease.[8]

Considering the adverse effects that obesity has on vascular hemodynamic and cardiovascular structure and function, not surprisingly, obesity has a strong malignant impact on multiple cardiovascular disease risk factors, including endothelial dysfunction, carotid artery intima-media thickening, increased arterial stiffness, and an abnormal hemodynamic environment.[9,10] Investigations in recent years have demonstrated that hemodynamic abnormalities, including blood velocity and wall shear stress, caused by obesity play an important role in the occurrence and development of vascular diseases.[11] Previous studies also found that due to excessive accumulation of fat in obese people, the pumping function of the heart is reduced, resulting in lower blood velocity and wall shear stress than normal.[12] Moreover, retrospective studies strongly showed that the artery diameter and stiffness in young obese individuals who have a BMI>30 kg/m^2 were greater than those in normal-weight individuals.[12] However, little is known about the relationship between visceral fat and artery hemodynamic parameters.

Studies strongly suggest that exercise can exert direct influences on the vasculature via the impact of repetitive exposure to hemodynamic stimuli.[13] Exercise is a powerful physiological stimulus that can effectively promote cardiovascular health and reduce the risk of cardiovascular diseases by changing hemodynamic indicators.[14] Studies have found that aerobic exercise has a positive effect on enhancing carotid hemodynamic parameters, which can effectively ameliorate cardiovascular diseases in obese adults.[15] Although moderate-intensity continuous training (MICT) brings many benefits to obese individuals, it still has limitations such as long time and single exercise form, resulting in low exercise adherence for obese adults. In recent years, high-intensity interval training (HIIT) has displayed excellent adherence among obese adults.[16,17] The beneficial effects of HIIT for adults to control weight and enhance hemodynamic parameters have received widespread attention. Compared with MICT and high-intensity sustained exercise, the superior effects of HIIT have been attributed to larger blood flow responses and/or greater reductions in oxidative stress, which together may enhance endothelial cell sensitivity to shear stress and increase nitric oxide (NO) bioavailability.[18,19] Currently, the effects of HIIT and MICT on the cardiovascular health of obese people are discussed in depth from oxidative stress, inflammatory response and vasoactive substances balance,[20] this study explored HIIT and MICT on vascular health in obese from the perspective of hemodynamics.[21] Additionally, BMI, WC, and BF (%) are generally used to measure obesity, but as powerful indicators for assessing obesity-induced metabolic syndrome, there is little discussion on the effect of HIIT and MICT on LAP and the VAI in obese individuals.

In this study, our objective was to evaluate the effects of 8 weeks of HIIT and MICT on visceral fat and carotid haemodynamic parameters in obese adults. Based on the above introduction, we hypothesized that 8-week HIIT would elicit superior improvements in LAP, the VAI, and carotid haemodynamic parameters than MICT in obese adults. Based on present observations, further, we predicted that there would be close correlations among LAP, the VAI, and partial carotid hemodynamic parameters.

2. Materials and methods

2.1. Participants

Participation in the study was voluntary, and all participants were enrolled from Dalian University of Technology. Eighty-nine adults with obesity were recruited for the sports program. Of these, 72 adults were willing to take part in the study. The inclusion criteria were as follows: male, BMI$\geq$30 kg/m^2, age 18–22 years; non-smoker; not taking any vasoactive medications; sedentary and not engaged in any structured/consistent physical exercise program at any time over the prior 3 months by the Chinese version of the International Physical Activity Questionnaire (IPAQ); the Physical Assessment Risk Questionnaire (PAR-Q) was used to exclude participants with exercise risk; on the basis of verifying that the recent medical reports of all participants were normal, participants were randomly selected for electrocardiogram assessment to further ensure the safety of exercise. All participants were required to complete a written informed consent form and a health history screening. All protocols and procedures were approved by the ethics committee of the department of Physical Education and

Sports, Beijing Normal University.

Of the 72 obese adults who were preselected, there were 56 participants in the exercise groups and 16 participants in the control group. 13 were excluded due to frequent absences during the training, and 7 could not adhere to the program due to personal issues (HIIT: 7; MICT: 6; CON: 7). And finally, 43 participants in the two exercise groups (HIIT: 21; MICT: 22) and 9 participants in the CON group completed our experiment (Fig. 1).

2.2. Experimental design

This study was designed as a prospective randomized controlled trial. The participants were randomly divided into high-intensity interval training group (HIIT) and moderate-intensity continuous training group (MICT) using randomization software (GraphPad software). In addition, we also recruited the CON group according to the same inclusion criteria and ensured that the baseline levels of each indicator in the CON group were not significantly different from the exercise groups. The experimental procedure was composed of a preparation period and pre-intervention measurements (including anthropometric assessments, biochemical analyses, and body fat index and carotid hemodynamic parameter). Both exercise groups underwent an 8-week HIIT or MICT program, and the CON group did not perform the exercise intervention. Considering the ethical requirements, the participants in the CON group did not alter their current physical activity patterns during the experiment. All participants were assessed for body composition, hemodynamic index of the common carotid artery, blood lipid indicators, LAP, and VAI before and after the 8-week intervention.

2.3. Training protocols

For the two training protocols, all sessions were supervised and conducted 3–4 times a week for 8 weeks without rest during holidays. To effectively monitor the intensity of exercise interventions, the Polar Team Pro were used to monitor heart rate during exercise. The Actigraph GT3X + triaxial accelerometer was used to monitor energy expenditure in both modes of exercise to ensure consistent energy expenditure.

2.3.1. HIIT

Lessons were drawn from the classic HIIT exercise program and other studies, we adjusted according to the existing situation.[21–23] The total exercise time was 10–30 min, and the exercise intensity was set to 80%–95% of the maximal heart rate (HRmax). The HRmax was determined by calculation using the formula 220-age (in years). There was no rest on holidays, and participants exercised 3–4 times a week. Considering that the exercise intensity was higher for obese college students who were sedentary and less active, the increase in exercise intensity needed to be gradual. Therefore, the entire exercise intervention program was divided into 3 stages (Fig. 2).

2.3.2. MICT

The MICT program recommended at least 30 min of daily moderate aerobic physical activity, which was mainly based on the American College of Sports Medicine physical activity guidelines. First, all participants in the MICT group performed warm-up activities for 5 min, then 45 min of moderate-intensity aerobic exercise with a 60%–70% HRmax, and finally 5 min of relaxation activities with a total exercise time of 55 min. The entire exercise intervention program was divided into 2 stages. The specific exercise intervention plan was as follows:

Weeks 1–4: 5 min warm-up exercises + playground running (15 min jogging + 5 min variable speed running) + 25 min aerobic exercises of different types between marches + 5 min stretching and relaxation exercises.

Weeks 5–8: Considering the loss of muscle mass, a little strength exercise without equipment was added appropriately before aerobic exercise to ensure that the peripheral conditions of the blood vessels in the two groups of hemodynamic tests are consistent. In addition, study found that walking and jogging are good exercises for overweight people to control their weight.[24] Hence, we adjusted the intervention program, mainly focusing on walking and jogging.

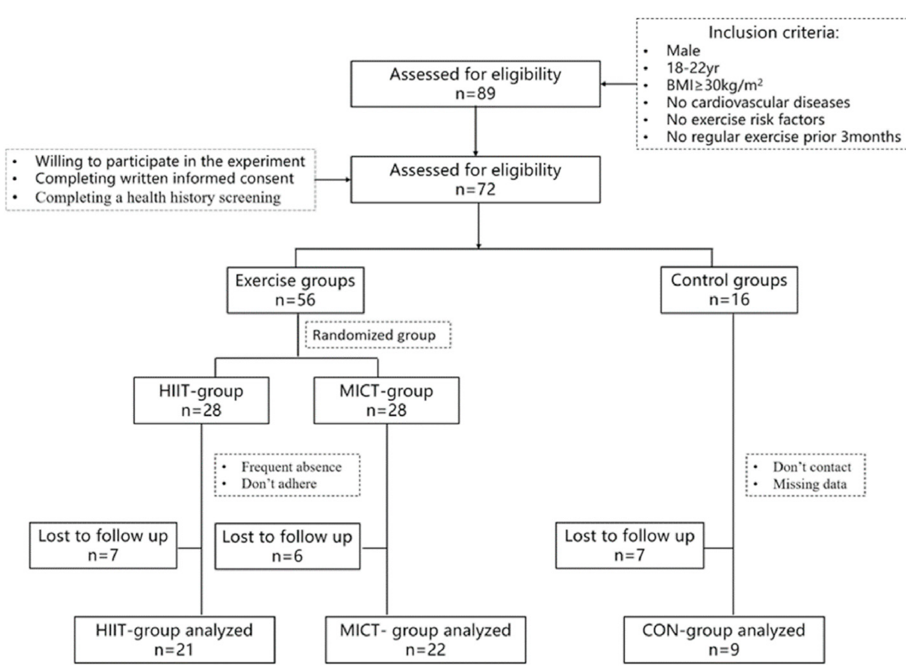

Fig. 1. Flow diagram of the selection of participants in the study.

Phase	Content of the intervention	Time schedule for each session	Total time
Phase one (1-2weeks)	Warm-up exercise: dynamic tensile exercise for 5mins, accelerative running in place for 5 mins.	Each action lasts 40 seconds and then rest for 20 seconds. Repeating for a total of 3 groups with a 60 seconds rest between each group.	25mins
	HIIT content: upward leg swing, horizontal jump, kneeling flat support, upward leg swinging, jumping jack, single hand touching shoulder, push-up with single leg-raising.		
	Relaxation exercise: streching exercise for 5mins.		
Phase two (3-5weeks)	Warm-up exercise: dynamic tensile exercise for 5mins, accelerative sprint for 5mins.	Each action lasts 60 seconds and then rest for 30 seconds. Repeating for a total of 4 groups with 60 seconds rest between each group.	35mins
	HIIT content: upward leg swing, vertical jump, jumping jack, sitting position legs open and close, interval rope skipping.		
	Relaxation exercise: streching exercise for 5mins.		
Phase three (6-8weeks)	Warm-up exercise: dynamic tensile exercise for 5mins, jogging for 5mins.	Each action lasts 60 seconds and then rest for 30 seconds. Repeating for a total of 4 groups with 60 seconds rest between each group.	45mins
	HIIT content: sprint, upward leg swinging, accelerative running in place, single leg hop, lunge, jumping jack, jump rope		
	Relaxation exercise: streching exercise for 5mins.		

Fig. 2. HIIT intervention program.

2.3.3. CON

The participants in CON group were conducted a healthy lifestyle study in various forms such as lectures and watching videos in classroom, symposiums in school playground, We-Chat group communication online. The contents mainly about the dangers of obesity, its causes and how to improve it. During intervention, body compositions and serum lipid indexes and hemodynamic testing programs were performed, but no exercise intervention was received.

2.4. Dietary advice

Diet control is essential in the process of fat reduction in obese adults. Therefore, we supervised and instructed all participants to a certain extent. During the eight weeks of exercise intervention, the participants' diets were not controlled uniformly but they were given some advice regarding diet to maximize the benefits of the exercise through nutrition education; fats, proteins, and carbohydrates were recommended to compose 65%, 25% and 10% of their daily energy intake, respectively. A WeChat group was set up, and all participants were asked to upload pictures of their food every day, which were analysed for energy intake and macronutrient content. According to these records, they were also supervised and acquired some dietary guidance from a professional. In addition, an improved version of the dietary nutrition questionnaire for college students was issued to further understand the dietary nutrition of all participants during the exercise intervention.

2.5. Pre- and- post intervention measurements

2.5.1. Anthropometric assessments

Fat mass, muscle mass, body fat percentage BF (%), visceral adipose tissue, subcutaneous fat content, and waist circumference (WC) were measured while the participants were shoeless to the nearest 0.05 cm by a Tanita MC-980MA body analyser. A tape measure was used to measure the waist and neck circumference of the participants, and the waist-to-height ratio was calculated. To minimize operating error, the before and after tests were completed by the same tester.

2.5.2. Biochemical analyses

Under aseptic precautions, venous blood was sampled for lipids from all participants before and after the intervention. To avoid the influence of biorhythm on the experimental results, blood sampling was uniformly performed at 8–10 a.m., and overnight fasting was needed. The serum was centrifuged within 2 h and stored in a −80 °C refrigerator. The 2 mL of the blood sample was used for determinations of serum high-density lipoprotein cholesterol (HDL-C) and (TG) at the School Hospital of Dalian University of Technology using an American RA-1000 automatic biochemical analyzer.

2.5.3. LAP and VAI measurement and calculation

The VAI takes full account of anthropometric and metabolic factors related to the area and volume of visceral adipose tissue but not subcutaneous adipose tissue and can more directly predict the progression and risk of CVD.[25] LAP combines anatomic and physiological metrics, such as WC, which reflects visceral fat content, and fasting triglyceride (TG) content. Both can accurately reflect the degree of lipid accumulation and metabolic abnormalities of the human body. The VAI and LAP were calculated based on waist circumference and blood indices. LAP and the VAI were calculated through anthropometric variables and laboratory parameters according to the following formula for men:

$$\text{LAP} = ([\text{WC (cm)}-65] \times \text{TG [mmol/L]}).^{26}$$

$$\text{VAI} = \text{WC (cm)}/[39.68 + 1.88 \times \text{BMI (kg/m}^2)] \times \text{TG (mmol/L)}/1.03 \times 1.31/\text{HDL-C (mmol/L)}.^{6}$$

2.5.4. Carotid hemodynamic parameters

The common carotid artery diameter and axial blood velocity waveform were measured with a high-resolution Doppler ultrasound (Prosound Alpha 7, Aloka, Japan) at 5–7 pm. First, participants were asked to rest in the supine position for 10 min. Second, the position of the common carotid artery was determined by transverse scanning. Third, the transverse section of the common carotid artery was longitudinally scanned to obtain the pictures about common carotid artery diameter waveform and the axial blood velocity waveform, as shown in Fig. 3. At the same time, the brachial systolic pressure, diastolic pressure, and heart rate (HR) on the left upper arm were measured using an electronic sphygmomanometer (Patient Monitor PM8000, Mindray). Based on the test data and pictorial information, MATLAB software (2017b, MathWorks, USA) and Womersley theory were used to calculate the hemodynamic parameters of the common carotid artery. The blood velocity, flow rate, blood pressure, wall shear stress, oscillatory shear index, peripheral resistance, elasticity modulus, dynamic resistance, artery diameter, arterial stiffness, circumferential strain and pulsatility index were included (Figs. 3 and 4).

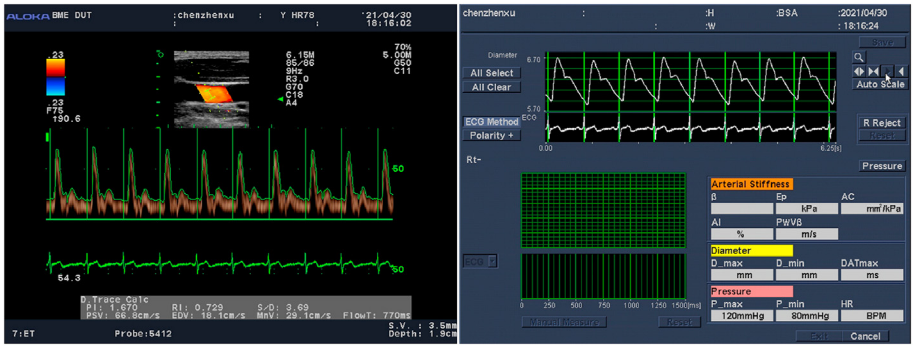

Fig. 3. Diameter and axial flow velocity waveform for the common carotid artery.

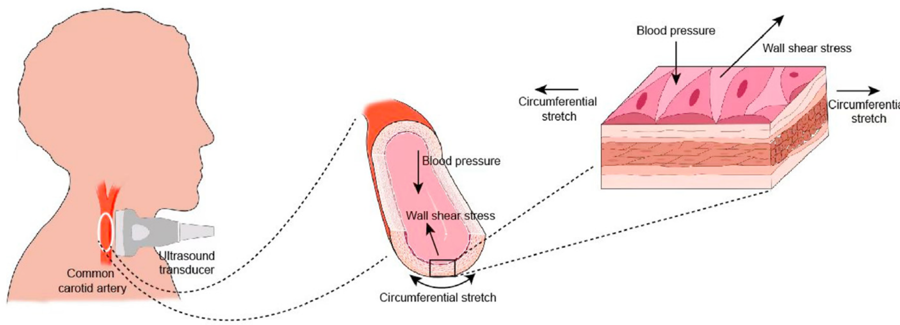

Fig. 4. Hemodynamic parameters of the common carotid artery.

2.6. Statistical analysis

First, one-way analysis of variance (ANOVA) was used to evaluate between-group differences with respect to baseline characteristics. Similarly, the approximate normality of each outcome was confirmed by examination. Second, paired t tests were carried out to detect the changes in the main outcome variables between different time points among each group. The magnitude of Cohen's d effect (ES) for changes within the group was classified as follows: "small": 0.2–0.5; "moderate": 0.5–0.8; and "large": >0.8. Third, one-way analysis of covariance (ANCOVA) was used to compare the effects of different exercise on the lipid accumulation index and carotid hemodynamic parameters. As the baseline value may affect the lipid accumulation index and carotid hemodynamic parameters, according to the requirements of one-way covariance analysis, baseline values were adjusted as covariates. Then, to compare the indicators which were adjusted with baseline level as covariable after intervention between groups. A partial eta squared (η^2) was computed to check the differences between groups, where 0.01 to 0.059 was determined as a small effect, 0.06 to 0.137 as a medium effect, and >0.138 as a large effect. Finally, the Pearson correlation method was performed to examine the correlation among the VAI, LAP, and carotid hemodynamic indices. Analyses were performed using SPSS software, version 22.0. The data are described as the mean ± SD, and values of $p < 0.05$ were considered statistically significant.

3. Results

Table 1 presents the baseline anthropometric characteristics of the 52, the differences between groups at baseline and after exercise in anthropometric, biochemical, and visceral fat indices and hemodynamic variables are described. At baseline, anthropometric measurements and body fat were similar, but there was a significant difference in some hemodynamic parameters among the groups, including the blood velocity, flow rate, blood pressure, wall shear stress, peripheral resistance, elasticity modulus, dynamic resistance.

In summary, after the 8-week intervention, compared with pre-exercise, fat mass, BF (%), and WC decreased significantly after HIIT (all, $p < 0.01$) and MICT (all, $p < 0.01$). The fat mass and BF (%) in the CON group also significantly declined, but muscle mass showed a significant increase in this study ($p < 0.01$).

Changes in visceral fat indices are summarized in Table 1. LAP in the HIIT group and the MICT group was significantly decreased ($p < 0.01$, $p < 0.05$) but not in the CON group ($p > 0.05$). In contrast, VAI values decreased in both the HIIT and MICT groups and increased in the CON group, although the difference between groups was not significant ($p > 0.05$).

The findings of carotid hemodynamic parameters are presented in Table 1. After 8 weeks of intervention, the blood velocity and wall shear stress were greater after HIIT and MICT ($p < 0.01$). Artery diameter, oscillatory shear index, arterial stiffness, and pulsatility index were increased significantly, and circumferential strain was significantly greater in the HIIT group (all, $p < 0.01$, $p < 0.05$), but not in the MICT group ($p > 0.05$). Dynamic resistance was significantly decreased in the MICT group. There was no difference in the CON group after the period of intervention (all, $p > 0.05$).

As shown in Table 2, a homogeneity test was conducted among the three groups using one-way analysis of variance, and we found that there was a difference in some hemodynamic parameters. Moreover, considering that baseline values of LAP, VAI, and hemodynamic parameters may affect postexercise changes, baseline values of indicators were used as covariates to explore differences in changes in indicators among the three groups.

The analysis of covariance showed that after 8 weeks of HIIT and

Table 1
Anthropometric, fat, and hemodynamic parameters in all groups at baseline and after exercise.

	HIIT			MICT			CON		
	Pre	**Post**	***P/ES***	**Pre**	**Post**	***P/ES***	**Pre**	**Post**	***P/ES***
Anthropometric Assessments and Biochemical Analyses									
FM (kg)	29.07 ± 9.13	24.83 ± 8.29$^{\&\&}$	**0.001/1.78**	28.19 ± 9.88	24.90 ± 9.39$^{\&\&}$	**0.001/1.18**	29.48 ± 8.55	27.23 ± 8.23$^{\&\&}$	**0.001/1.62**
MM (kg)	62.15 ± 5.43	61.71 ± 5.55	0.16/0.32	59.38 ± 6.05	58.41 ± 6.34$^{\&}$	**0.04/0.48**	62.46 ± 6.72	60.13 ± 6.81$^{\&\&}$	**0.01/0.72**
BF (%)	30.10 ± 5.02	27.01 ± 5.14$^{\&\&}$	**0.001/1.91**	30.19 ± 5.76	27.89 ± 5.82$^{\&\&}$	**0.001/1.13**	30.40 ± 5.10	28.50 ± 5.18$^{\&\&}$	**0.001/2.04**
WC (cm)	98.05 ± 10.33	92.20 ± 9.31$^{\&\&}$	**0.001/1.43**	97.84 ± 12.81	92.37 ± 11.47$^{\&\&}$	**0.001/0.89**	99.92 ± 7.97	98.02 ± 9.57	0.12/0.58
HDL-C (mg/dL)	1.24 ± 0.22	1.25 ± 0.15	0.72/0.08	1.27 ± 0.20	1.27 ± 0.22	0.85/-0.06	1.27 ± 0.16	1.17 ± 0.12	0.08/0.67
TG (mg/dL)	1.21 ± 0.35	1.11 ± 0.37	0.19/0.32	1.18 ± 0.47	1.03 ± 0.45	0.08/0.43	1.33 ± 0.54	1.61 ± 0.64	0.35/-0.33
Visceral Fat Indices Measurement and Calculation									
LAP	42.70 ± 21.51	32.62 ± 19.17$^{\&\&}$	**0.001/0.32**	31.81 ± 15.66	22.31 ± 11.81$^{\&}$	**0.03/0.59**	39.78 ± 19.56	53.70 ± 33.94	0.30/-0.43
VAI	1.35 ± 0.56	1.16 ± 0.52	0.07/0.45	1.14 ± 0.53	0.97 ± 0.52	0.13/0.38	1.42 ± 0.68	1.87 ± 0.71	0.22/-0.48
Carotid Hemodynamic Parameters									
BV(m/s)	0.26 ± 0.07*$^{\#}$	0.29 ± 0.06$^{\&\&}$	**0.01/-0.50**	0.22 ± 0.06	0.26 ± 0.05$^{\&\&}$	**0.01/-0.57**	0.19 ± 0.05	0.20 ± 0.03	0.72/-0.17
AD (cm)	0.66 ± 0.06	0.62 ± 0.07$^{\&\&}$	**0.01/0.57**	0.63 ± 0.09	0.62 ± 0.09	0.48/0.14	0.66 ± 0.08	0.67 ± 0.08	0.85/-0.09
BP (kPa)	13.25 ± 0.61$^{\#\#}$	12.92 ± 0.87	0.12/0.33	12.29 ± 0.97	12.66 ± 1.23	0.10/-0.38	12.69 ± 1.33	12.74 ± 1.23	0.91/-0.04
WSS (Pa)	0.60 ± 0.20*	0.73 ± 0.22$^{\&\&}$	**0.001/0.68**	0.59 ± 0.21	0.69 ± 0.16$^{\&}$	**0.02/-0.59**	0.49 ± 0.18	0.51 ± 0.11	0.87/-0.05
FR	4.27 ± 1.33*$^{\#\#}$	3.93 ± 0.65	0.27/0.27	3.30 ± 0.81	3.85 ± 1.09	0.07/-0.44	3.30 ± 1.12	3.52 ± 0.92	0.57/-0.20
OSI	0.15 ± 0.06	0.09 ± 0.04$^{\&\&}$	**0.001/1.04**	0.08 ± 0.05	0.08 ± 0.05	0.59/0.02	0.12 ± 0.07	0.12 ± 0.05	0.70/-0.14
EM (kPa)	55.36 ± 10.52$^{\#}$	48.07 ± 9.74	0.14/0.61	45.99 ± 11.77	44.89 ± 17.31*	0.78/0.06	59.19 ± 17.94	55.14 ± 17.87	0.59/0.19
AS	4.04 ± 0.62	3.28 ± 0.60$^{\&\&}$	**0.001/1.10**	3.80 ± 0.89	3.53 ± 1.29	0.41/0.18	4.37 ± 1.03	4.18 ± 1.27	0.75/0.12
DR ($Pa \cdot s \cdot m^{-1}$)	0.83 ± 0.23*$^{\#}$	0.81 ± 0.22	0.79/0.07	1.03 ± 0.24	0.83 ± 0.28$^{\&}$	**0.02/0.57**	1.04 ± 0.33	1.03 ± 0.30	0.92/0.03
Rq ($Pa \cdot s \cdot m^{-3}$)	23.68 ± 7.46$^{\#}$	23.44 ± 5.61	0.89/0.03	27.50 ± 6.56	24.20 ± 7.35	0.14/0.35	31.94 ± 10.92	28.62 ± 7.17	0.39/0.31
CS	0.11 ± 0.02	0.13 ± 0.03$^{\&\&}$	**0.02/-0.64**	0.12 ± 0.02$^{\#}$	0.12 ± 0.04	0.84/-0.03	0.09 ± 0.03	0.10 ± 0.03	0.61/-0.25
PI	2.00 ± 0.21	1.86 ± 0.29$^{\&\&}$	**0.03/0.54**	1.90 ± 0.32	1.88 ± 0.28	0.80/0.06	2.10 ± 0.36	2.01 ± 0.29	0.62/0.20

Note: Outcome variables are shown as the means ± standard deviations. One-way analysis of variance (ANOVA) was used to compare the differences among groups at baseline, and LSD post hoc testing was used for pairwise comparisons. Significant differences in the CON group at $**p < 0.01$, $*p < 0.05$, significant differences in the MICT group at $^{\#\#}p < 0.01$, $^{\#}p < 0.05$. Paired t tests were used to compare the significant differences within groups at $^{\&}p < 0.05$ and $^{\&\&}p < 0.01$. ES: effect size, P/ES: p value and effect size. FM: fat mass, MM: muscle mass, BF (%): percentage of body fat, WC: waist circumference, HDL-C: high-density lipoprotein cholesterol, TG: triglycerides, LAP: lipid accumulation product, VAI: visceral adiposity index, BV: blood velocity, AD: artery diameter, BP: blood pressure, WSS: wall shear stress, FR: flow rate, OSI: oscillatory shear index, EM: elasticity modulus, AS: arterial stiffness, DR: dynamic resistance, Rq: peripheral resistance, CS: circumferential strain, PI: pulsatility index.

Table 2
Comparison of the change in body fat and hemodynamic parameters after exercise.

	Comparison Among Different Groups											
	HIIT	MICT	*P*	η^2	HIIT	CON	*P*	η^2	MICT	CON	*P*	η^2
Visceral Fat Indices Measurement and Calculation												
LAP	29.83 ± 2.84	25.59 ± 3.10	0.33	0.03	32.10 ± 4.27	55.18 ± 7.22	**0.01**	**0.24**	23.23 ± 4.82	51.46 ± 7.58	**0.01**	**0.32**
VAI	1.10 ± 0.09	1.03 ± 0.10	0.63	0.01	1.17 ± 0.12	1.84 ± 0.19	**0.01**	**0.28**	1.00 ± 0.13	1.79 ± 0.20	**0.00**	**0.33**
Carotid Hemodynamic Parameters												
BV (m/s)	0.29 ± 0.10	0.27 ± 0.10	0.30	0.03	0.29 ± 0.01	0.22 ± 0.02	**0.00**	**0.30**	0.26 ± 0.01	0.20 ± 0.02	**0.00**	**0.28**
AD (cm)	0.61 ± 0.02	0.63 ± 0.02	0.26	0.03	0.62 ± 0.02	0.68 ± 0.02	0.08	0.11	0.63 ± 0.02	0.66 ± 0.03	0.34	0.03
BP (kPa)	12.57 ± 0.23	12.98 ± 0.22	0.23	0.04	12.85 ± 0.21	12.90 ± 0.32	0.89	0.00	12.73 ± 0.23	12.56 ± 0.35	0.69	0.01
WSS (Pa)	0.71 ± 0.03	0.63 ± 0.04	0.10	0.07	0.71 ± 0.04	0.51 ± 0.06	**0.01**	**0.23**	0.60 ± 0.03	0.47 ± 0.04	**0.01**	**0.23**
FR	3.84 ± 0.23	3.87 ± 0.22	0.94	0.00	3.87 ± 0.17	3.53 ± 0.25	0.29	0.05	3.79 ± 0.24	3.40 ± 0.35	0.37	0.03
OSI	0.08 ± 0.01	0.13 ± 0.01	**0.00**	**0.24**	0.09 ± 0.01	0.13 ± 0.01	**0.01**	**0.24**	0.09 ± 0.01	0.07 ± 0.01	0.19	0.07
EM (kPa)	46.40 ± 3.22	46.48 ± 3.13	0.99	0.00	48.38 ± 2.79	54.44 ± 4.18	0.24	0.05	46.21 ± 3.86	52.07 ± 6.12	0.44	0.02
AS	3.24 ± 0.24	3.57 ± 0.23	0.33	0.03	3.30 ± 0.20	4.14 ± 0.31	**0.03**	**0.18**	3.56 ± 0.29	4.10 ± 0.47	0.35	0.04
DR ($Pa \cdot s \cdot m^{-1}$)	0.82 ± 0.06	0.82 ± 0.06	0.93	0.00	0.83 ± 0.06	0.99 ± 0.09	0.11	0.10	0.83 ± 0.06	1.03 ± 0.10	0.09	0.10
Rq ($Pa \cdot s \cdot m^{-3}$)	23.84 ± 1.46	23.79 ± 1.50	0.98	0.00	24.09 ± 1.34	27.19 ± 2.08	0.24	0.05	24.45 ± 1.69	28.10 ± 2.49	0.24	0.05
CS	0.13 ± 0.007	0.12 ± 0.006	0.12	0.07	0.13 ± 0.01	0.11 ± 0.01	0.15	0.09	0.12 ± 0.01	0.11 ± 0.01	0.89	0.01
PI	1.84 ± 0.06	1.91 ± 0.06	0.44	0.02	1.87 ± 0.07	1.99 ± 0.11	0.33	0.04	1.90 ± 0.06	1.97 ± 0.11	0.56	0.01

Note: One-way analysis of covariance (ANCOVA) was used to compare the change of different groups on the lipid accumulation index and carotid hemodynamic parameters. The data in Table 2 were adjusted value based on baseline level as covariable; outcome variables are shown as the means ± standard deviations. Statistical significance was set at an alpha level of 95% ($p < 0.05$) for all statistical measures. Partial η^2 value for effect size.
LAP: lipid accumulation product, VAI: visceral adiposity index, BV: blood velocity, AD: artery diameter, BP: blood pressure, WSS: wall shear stress, FR: flow rate, OSI: oscillatory shear index, EM: elasticity modulus, AS: arterial stiffness, DR: dynamic resistance, Rq: peripheral resistance, CS: circumferential strain, PI: pulsatility index.

MICT, except for the oscillatory shear index in the HIIT group, which was significantly decreased compared with the MICT group ($F = 11.59$, $p < 0.01$, $\eta^2 = 0.24$), there was no difference between the two exercise groups (all, $p > 0.05$). Compared with the CON group, LAP and VAI values of the two exercise groups were significantly lower than those of the CON group (LAP: $F = 7.57$, $p < 0.01$, $\eta^2 = 0.24$, $F = 9.68$, $p < 0.01$, $\eta^2 = 0.32$; VAI: $F = 9.35$, $p < 0.01$, $\eta^2 = 0.28$; $F = 10.99$, $p < 0.01$, $\eta^2 = 0.33$). The results also showed that the blood velocity and wall shear stress were significantly greater in the HIIT group and the MICT group than in the CON group (the blood velocity: $F = 11.81$, $p < 0.01$, $\eta^2 = 0.30$, $F = 10.47$, $p < 0.01$, $\eta^2 = 0.28$; wall shear stress: $F = 7.88$, $p < 0.01$, $\eta^2 = 0.23$, $F = 7.33$, $p < 0.01$, $\eta^2 = 0.23$). In addition, compared with the CON group, the oscillatory shear index and arterial stiffness were also significantly decreased in the HIIT group (oscillatory shear index: $F = 8.29$, $p < 0.01$, $\eta^2 = 0.24$, arterial stiffness: $F = 5.11$, $p < 0.03$, $\eta^2 = 0.18$) but not in the MICT group ($p > 0.05$). However, there was no significant difference in the hemodynamic parameters except for the oscillatory shear index between the two training groups. See Table 2 for a summary of these reports.

We further analysed the correlations among the variables to explore the relationships of LAP (Fig. 5) and the VAI (Fig. 6) with carotid hemodynamic parameters, respectively. The results of the Pearson correlation method demonstrated that a strong correlation emerged between fat parameters and the main hemodynamic indices. LAP was positively related to artery diameter ($r = 0.48$, $p = 0.011$), blood pressure ($r = 0.46$, $p = 0.002$), flow rate ($r = 0.31$, $p = 0.04$), oscillatory shear index ($r = 0.44$, $p = 0.03$), and elasticity modulus ($r = 0.33$, $p = 0.029$) but inversely related to circumferential strain ($r = -0.36$, $p = 0.028$). The VAI was also positively associated with artery diameter ($r = 0.33$, $p = 0.03$), elasticity modulus ($r = 0.38$, $p = 0.009$), and AS ($r = 0.39$, $p = 0.012$). In addition, the VAI was negatively correlated with circumferential strain ($r = -0.33$, $p = 0.04$).

4. Discussion

The number of obese adults around the world has soared drastically in recent years due to sedentary lifestyles and high-calorie dietary habits, which have led to a rapid increase in metabolic syndrome. In this study, we assessed the effects of HIIT and MICT on LAP, the VAI, and hemodynamic parameters in obese adults. The primary findings in our study were as follows: 1) male obese participants reduced their fat mass, muscle mass, and BF (%) after 8 weeks of HIIT and MICT intervention; 2) LAP and VAI were improved in the HIIT and MICT groups but not the CON group; and 3) compared with the CON group, HIIT and MICT improved the partial hemodynamic parameters, including the blood velocity and wall shear stress, but only the HIIT group significantly improved in the oscillatory shear index and arterial stiffness after intervention. Furthermore, compared with MICT, oscillatory shear index was significantly different in the HIIT group. 4) Visceral obesity was significantly correlated with partial hemodynamic parameters (Fig. 7).

4.1. Different types of exercise and visceral fat

LAP and VAI are considered independent predictors of cardiovascular diseases in healthy populations. In this study, our results validated our initial hypothesis. Intriguingly, we found that after 8 weeks of exercise intervention, fat mass and BF (%) in the HIIT, MICT, and CON groups were significantly decreased compared with the baseline, but LAP and the VAI decreased markedly only in the HIIT and MICT groups, and there was no significant difference in the CON group. Combined with the results of the changes in muscle content, visceral fat, and carotid hemodynamic indicators among the three groups, it is possible that the CON group was provided more information about diet, which could generate a kind of

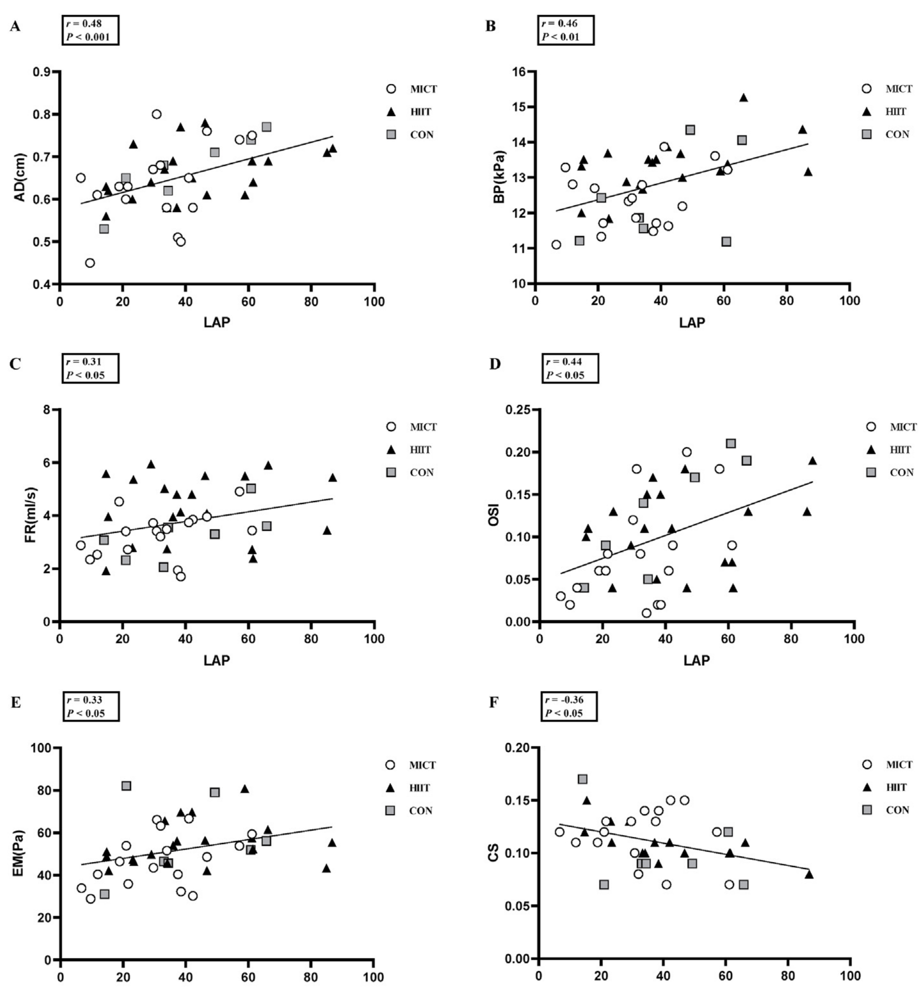

Fig. 5. Correlation analysis of LAP and partial hemodynamic parameters.
Note: LAP: lipid accumulation product, AD: artery diameter, BP: blood pressure, FR: flow rate, OSI: oscillatory shear index, EM: elasticity modulus, CS: circumferential strain.

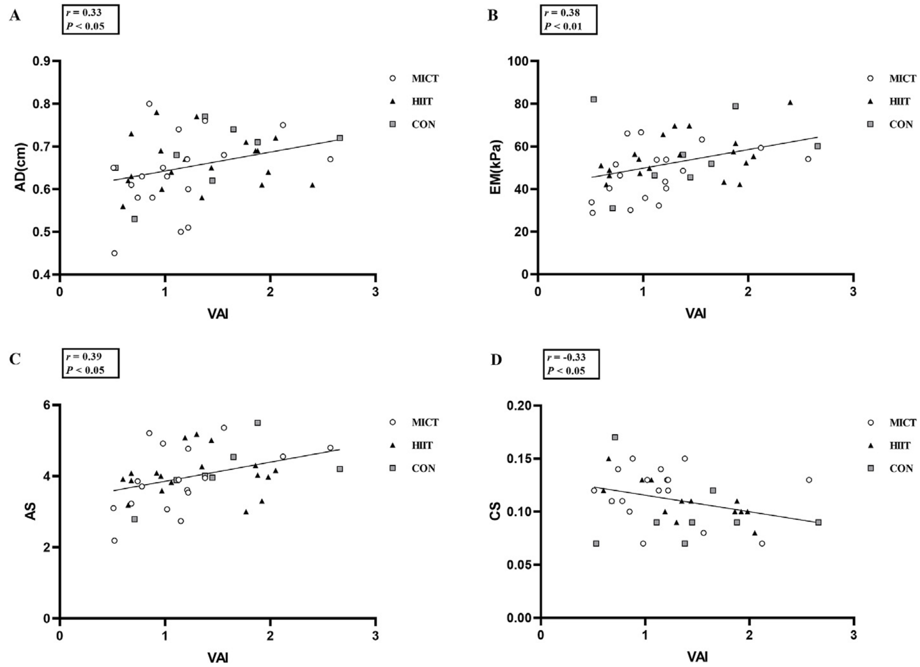

Fig. 6. Correlation analysis of VAI and partial hemodynamic parameters.
Note: VAI: visceral adiposity index, AD: artery diameter, EM: elasticity modulus, AS: arterial stiffness, CS: circumferential strain.

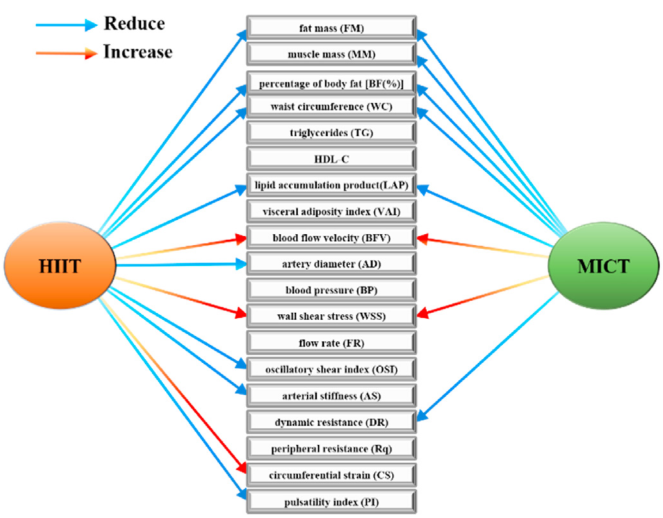

Fig. 7. The differences between HIIT and MICT on physiological parameters.

awareness that induce the participants in the CON group to eat better. Moreover, this non-active approach does not promote visceral fat loss. Therefore, our results further confirmed the positive effect of HIIT and MICT in this study from another perspective.

However, an unexpected finding in the present study was that no significant difference was found between HIIT and MICT in improving visceral fat. Notably, a previous study reported that a 12-week exercise intervention with resistance training (RT) and aerobic training (AT) could effectively induce a significant improvement in LAP and VAI values in adults with type 2 diabetes. Moreover, there was no significant difference in the improvement effect between the two groups, which was consistent with our findings.[27] We infer that it may be related to the similar energy expenditure after HIIT and MICT exercise. Energy consumed by exercise includes the energy consumed and excess post-exercise oxygen consumption (EPOC).[28] Study has found that compared with MICT, HIIT might has more obvious phenomenon of EPOC, although MICT might have a more energy consumed during exercise.[29] From the perspective of fat oxidation, we speculated that there was no significant difference about visceral fat between HIIT and MICT, probably because they both effectively promote fat oxidation. Study has confirmed that moderate-intensity aerobic exercise, especially when the intensity is 60%–65% of the maximum oxygen uptake, has the highest amount of fat oxidation,[30] and someone have found that HIIT stimulates more and makes catecholamines, growth hormone and other hormone levels rise, promote fat hydrolysis.[31]

4.2. Different types of exercise, blood velocity, wall shear stress, and oscillatory shear index

The common carotid artery plays a crucial role in the early diagnosis of cardio-cerebrovascular diseases, and the changes in carotid hemodynamic parameters can reflect the overall hemodynamic condition of the cardio-cerebrovascular system.[32] Therefore, this study mainly focused on the common carotid artery to explore the effects of HIIT and MICT on the hemodynamic parameters of the common carotid artery in obese adults. Our data showed that blood velocity and wall shear stress increased significantly after HIIT and MICT. Consistent with our findings, many studies have shown that exercise could increase blood velocity and wall shear stress and effectively improve vascular endothelial function.[33] The underlying mechanism may be that a certain intensity of exercise can increase circulating blood volume, further increase the wall shear stress on the vascular wall, and promote the phosphorylation of vascular endothelial nitric oxide synthase (eNOS), leading to vasodilation.[34] Nevertheless, there was no significant difference in blood velocity or wall shear stress between HIIT and MICT. We speculate that both HIIT and MICT can effectively improve the hemodynamic environment of obese adults and improve vascular endothelial function to a certain extent.

In this study, we also found that only the oscillatory shear index decreased significantly after HIIT exercise intervention, and it was significantly lower than that in the MICT group and the CON group. The oscillatory shear index represents the proportion of the reverse wall shear stress and the total wall shear stress in a cardiac cycle and the oscillatory degree of the forward and backwards wall shear stress.[35] The greater the oscillatory shear index is, the higher the risk of endothelial dysfunction.[36] Although we did not measure retrograde shear, we extrapolated that the oscillatory shear index value decreased significantly after HIIT when there was no difference in anterograde wall shear stress between the HIIT group and the MICT groups, which suggests that HIIT can effectively reduce retrograde shear and improve endothelial function.[37] One possible explanation for the positive impact of HIIT on oscillatory shear index was that HIIT has regular rest periods during exercise, which could inhibit the endothelial damage caused by retrograde shear stress. However, the underlying mechanisms remain unclear, and future studies should be conducted to explore the biomechanical mechanism of the impact on oscillatory shear index after HIIT.

Another potential mechanism of oscillatory shear index decrease is a decrease in body fat. As previously described, oscillatory shear index reflects the degree of the shock of blood flowing in blood vessels, and obesity plays a crucial role in regulating hemodynamic parameters.[9] Although LAP and the VAI are currently considered new indicators for evaluating visceral obesity, how visceral fat may underlie this regulation has been much less explored. We speculated that alterations in LAP and VAI values may affect oscillatory shear index to a certain extent. Our study provided a novel finding that the oscillatory shear index was significantly positively correlated with the visceral fat index LAP and VAI through correlation analysis, which was consistent with our inference. Thus, combined with the results of the significant decrease in the VAI, LAP, and oscillatory shear index after HIIT exercise, we further deduced that the positive effect of HIIT on oscillatory shear index may also be related to the reduction in visceral fat content in obese participants. Unfortunately, we have not found a correlation between the changes in each indicator before and after, which may be due to the small sample size, and future research needs to be executed to determine whether an oscillatory shear index reduction led to decreased VAI and LAP values.

4.3. Different types of exercise, arterial stiffness and artery diameter

Another novel finding was the training-induced reduction in arterial stiffness in the HIIT group. arterial stiffness is an indicator used to evaluate vascular structure, and obesity can lead to a remarkable increase in arterial stiffness.[38–40] The improvement in arterial stiffness after HIIT, but not MICT, can be explained by the mode and intensity of exercise. Study has indicated that both moderate-intensity continuous exercise and interval exercise can improve systemic arterial stiffness, and the effect of interval exercise is superior to that of continuous exercise, which may be related to the cumulative effect of interval exercise.[41] One possible explanation for the positive effect of HIIT on arterial stiffness might be the change in circumferential strain, which can reflect the circumferential deformation capacity of the arterial wall after cardiac pumping. Research has demonstrated that circumferential strain is an important factor influencing arterial stiffness, which has a negative association with arterial stiffness. The higher the deformability of the common carotid artery, the lower its stiffness.[42] In this study, we observed that circumferential strain increased significantly after HIIT but not MICT, so we can infer that HIIT exercise can enhance the deformability of the common carotid artery, thereby effectively reducing arterial stiffness.

Based on previous studies and our present research results, it was necessary to explore whether there was a certain relationship between the visceral fat index and arterial stiffness. We hypothesized that the visceral fat index was positively correlated with arterial stiffness and negatively correlated with circumferential strain. Therefore, we further verified the underlying correlations between LAP, the VAI, arterial stiffness, and circumferential strain by the Pearson correlation method. More specifically, we found that the VAI was positively related to arterial stiffness, and there was a negative correlation with circumferential strain, which is consistent with our original hypothesis. Previous study has shown that the VAI was closely related to atherosclerosis, which could effectively support the current results of this study, and we also found that the VAI and arterial stiffness were significantly decreased and circumferential strain was significantly increased after HIIT exercise.[43] Thus, these results further provide new evidence for the improvement of common carotid artery stiffness.

An enlarged artery lumen diameter is associated with numerous cardiovascular risk factors and is considered one of the early and easily detectable measures of vascular remodelling.[44] Study has indicated that obesity can lead to arterial enlargement, which might be a compensatory mechanism for regulating wall shear stress.[45] In our study, we also found that artery diameter was positively related to the LAP and VAI, which is consistent with the results of previous studies. Chung et al. suggested that arterial enlargement in obese participants was an adaptative response due to proportionate changes in blood flow to maintain shear stress.[46] Moreover, other studies revealed that a larger artery diameter was accompanied by a decrease in shear stress in obese participants with BMI>30 kg/m^2.[47] We utilized vascular diameter as an indicator to evaluate arterial structure. We found that there was a significant decrease in vascular diameter in the HIIT group after 8 weeks of exercise intervention. However, there was no change in vascular diameter in the CON and MICT groups. Many studies have demonstrated that exercise training increases artery diameter in clinical populations, elite athletes, and young adult humans.[48–50] It has been shown that young adults who undergo regular endurance training have larger lumen diameters of the main conduit arteries of trained limbs than young adults of the same age who do not perform regular exercise. Meanwhile, Dinenno et al. also found an increase in the femoral artery lumen diameter after 3 months of exercise training in healthy individuals.[50] However, these studies mainly focus on nonobese people and nonobese people with BMI≥30 kg/m^2. Therefore, it is not clear how the common carotid artery diameter changes in obese adults after systemic HIIT and MICT exercise.

According to the results of our study, we speculated that exercise can curb or relieve the pathological vascular dilatation of obese people to some extent. When vascular diameter returns to the normal state, exercise might promote positive exercise adaptation of the vasculature for a longer time. In addition, in our study, we also found that both HIIT and MICT could significantly improve the shear stress and velocity of blood flow in obese adults, which were significantly higher than those in the CON group. Therefore, we further speculated that exercise increased the shear stress of blood flow in obese people, improving the state of blood vessel dilation to compensate for the decreased blood shear stress.[45] In addition, another possible explanation was that there was a greater demand for oxygen and nutrients in obese individuals with increased tissue mass.[44] After 8 weeks of exercise intervention, body shape indicators such as BF (%), WC, LAP, and the VAI in HIIT were significantly reduced, so these data showed that visceral fat in these young men was associated with artery remodelling and was also accompanied by partial hemodynamic changes. Similarly, the reduced artery diameter was consistent with reduced LAP and VAI after HIIT prevention.

4.4. Practical implications

A strength of this study is that we included several hemodynamic indices that had not been reported in most previous studies comparing HIIT and MICT in obese adults. To our knowledge, this is the first investigation that explored the effectiveness of HIIT and MICT intervention in decreasing LAP and VAI values and improving hemodynamic parameters. We also found that HIIT could effectively reduce the oscillatory shear index, and the improvement effect was superior to MICT, which was first found in studies on the effects of HIIT and MICT on carotid hemodynamic parameters in obese adults. Moreover, in this study, we also concluded that the lipid accumulation index and visceral fat index may be closely related to partial carotid hemodynamic parameters. In addition, to ensure the safety and efficiency of each session as much as possible, each session was supervised by research technicians while the heart rate was monitored. Finally, our research was an intervention study outside the laboratory and with more experimental participants, which was closer to reality and can more truly reflect the effect of exercise on weight loss and improve carotid hemodynamic parameters of obese adults in the real world.

4.5. Study limitations and future research

There were also some limitations to our study to be considered. First, one major limitation was the small sample size of the CON group, which was due to the participants in the CON group who had no intervention being particularly vulnerable to loss to follow-up. Second, to control for potential confounding factors of sex and eliminate the effect of menstrual cycles in females, only men were included in our sample. Therefore, it is unknown whether our findings also apply to women. Sex differences may exist in vascular responses to exercise, thereby limiting the generalizability of our results. Third, we supervised and guided participants' diets in the WeChat group, and all participants were required to submit pictures of their meals on WeChat, but we did not perform diet control and we will try to control for dietary factors as tightly as possible in future studies. Fourth, considering ethical requirements, we did not randomize the control and exercise groups in this study, although the baselines were homogeneous between the control and exercise groups, so the control group in this study, to some extent, might not be a true control group, which may have some influence on the results of the study.

5. Conclusions

The findings of this study demonstrated that 8 weeks of HIIT and MICT could decrease visceral fat indices and improve the BFV and WSS of the common carotid artery. The HIIT group exhibited lower oscillatory shear index and artery stiffness and greater circumferential strain, which indicated that HIIT may produce a clinically meaningful improvement in vascular endothelial function and artery stiffness in the short term. Therefore, HIIT and MICT exert important effects on reducing fat content and improving hemodynamic environment. In addition, HIIT should be considered for improving vascular structure and function in obese adults. Moreover, the results of this study found that visceral fat is associated with the partial hemodynamic parameters of the common carotid artery, which plays a significant role in hemodynamic homeostasis.

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Author contributions

Conceptualization, W.S., D.T.; methodology, W.S., J.C., P.S., M.W., Y.H.; investigation, J.C.; resources, D.T. X.L.; writing—original draft preparation, W.S., D.T.; writing—review and editing, W.S., D.T. All authors have read and agreed to the published version of the manuscript.

Institutional review board statement

This study was conducted according to the guidelines of the Declaration of Helsinki, and approved by the ethics committee of the department of Physical Education and Sports, Beijing Normal University.

Informed consent statement

Informed consent was obtained from all subjects involved in the study.

Declaration of competing interest

The authors declare no conflict of interest.

References

1. Collaboration NCDRF. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *Lancet (London, England)*. 2017;390(10113):2627–2642.
2. Rosenquist KJ, Pedley A, Massaro JM, et al. Visceral and subcutaneous fat quality and cardiometabolic risk. *JACC Cardiovasc Imaging*. 2013;6(7):762–771.
3. Alessi MC, Peiretti F, Morange P, Henry M, Nalbone G, Juhan-Vague I. Production of plasminogen activator inhibitor 1 by human adipose tissue: possible link between visceral fat accumulation and vascular disease. *Diabetes*. 1997;46(5):860–867.
4. Maskarinec G, Shvetsov YB, Wong MC, et al. Subcutaneous and visceral fat assessment by DXA and MRI in older adults and children. *Obesity*. 2022;30(4): 920–930.
5. Lemos T, Gallagher D. Current body composition measurement techniques. *Curr Opin Endocrinol Diabetes Obes*. 2017;24(5):310–314.
6. Amato MC, Giordano C, Galia M, et al. Visceral Adiposity Index A reliable indicator of visceral fat function associated with cardiometabolic risk. *Diabetes Care*. 2010;33(4):920–922.
7. Nusrianto R, Ayundini G, Kristanti M, et al. Visceral adiposity index and lipid accumulation product as a predictor of type 2 diabetes mellitus: the Bogor cohort study of non-communicable diseases risk factors. *Diabetes Res Clin Pract*. 2019;155, 107798.
8. Ioachimescu AG, Brennan DM, Hoar BM, Hoogwerf BJ. The lipid accumulation product and all-cause mortality in patients at high cardiovascular risk: a PreCIS database study. *Obesity*. 2010;18(9):1836–1844.
9. Alpert MA, Omran J, Bostick BP. Effects of obesity on cardiovascular hemodynamics, cardiac morphology, and ventricular function. *Curr Obes Rep*. 2016;5(4):424–434.
10. Tounian P, Aggoun Y, Dubern B, et al. Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study. *Lancet*. 2001;358(9291):1400–1404.
11. Lemmens HJ, Bernstein DP, Brodsky JB. Estimating blood volume in obese and morbidly obese patients. *Obes Surg*. 2006;16(6):773–776.
12. Kappus RM, Fahs CA, Smith D, et al. Obesity and overweight associated with increased carotid diameter and decreased arterial function in young otherwise healthy men. *Am J Hypertens*. 2014;27(4):628–634.
13. Liao ZY, Peng MC, Yun CH, et al. Relation of carotid artery diameter with cardiac geometry and mechanics in heart failure with preserved ejection fraction. *J Am Heart Assoc*. 2012;1(6).
14. Green DJ, Hopman MT, Padilla J, Laughlin MH, Thijssen DH. Vascular adaptation to exercise in humans: role of hemodynamic stimuli. *Physiol Rev*. 2017;97(2): 495–528.
15. Zeigler ZS, Swan PD, Buman MP, Mookadam F, Gaesser GA, Angadi SS. Post-exercise hemodynamic responses in lean and obese men. *Med Sci Sports Exerc*. 2018;50(11):2292–2300.
16. Bartlett JD, Close GL, MacLaren DP, Gregson W, Drust B, Morton JP. High-intensity interval running is perceived to be more enjoyable than moderate-intensity continuous exercise: implications for exercise adherence. *J Sports Sci*. 2011;29(6):547–553.
17. Bluher S, Kapplinger J, Herget S, et al. Cardiometabolic risk markers, adipocyte fatty acid binding protein (aFABP) and the impact of high-intensity interval

training (HIIT) in obese adolescents. *Metabolism*. 2017;68:77–87.
18. Vita JA, Holbrook M, Palmisano J, et al. Flow-induced arterial remodeling relates to endothelial function in the human forearm. *Circulation*. 2008;117(24):3126–3133.
19. Lyall GK, Davies MJ, Ferguson C, Porter KE, Birch KM. In-exercise vascular shear rate during acute continuous and interval exercise: impact on endothelial function and miR-21. *J Appl Physiol*. 2019;127(6):1754–1762.
20. Ramos JS, Dalleck LC, Tjonna AE, Beetham KS, Coombes JS. The impact of high-intensity interval training versus moderate-intensity continuous training on vascular function: a systematic review and meta-analysis. *Sports Med*. 2015;45(5):679–692.
21. Dias KA, Ingul CB, Tjonna AE, et al. Effect of high-intensity interval training on fitness, fat mass and cardiometabolic biomarkers in children with obesity: a randomised controlled trial. *Sports Med*. 2018;48(3):733–746.
22. Batitucci G, Junior EFV, Nogueira JE, et al. Impact of intermittent fasting combined with high-intensity interval training on body composition, metabolic biomarkers, and physical fitness in women with obesity. *Front Nutr*. 2022;9.
23. Chin EC, Leung CK, Yu DJ, et al. Effects of one-year once-weekly high-intensity interval training on body adiposity and liver fat in adults with central obesity: study protocol for a randomized controlled trial. *Journal of Exercise Science & Fitness*. 2022;20(2):161–171.
24. Loftin M, Waddell DE, Robinson JH, Owens SG. Comparison of energy expenditure to walk or run a mile in adult normal weight And overweight men and women. *J Strength Condit Res*. 2010;24(10):2794–2798.
25. Yu J, Yi Q, Chen G, et al. The visceral adiposity index and risk of type 2 diabetes mellitus in China: a national cohort analysis. *Diabetes Metab Res Rev*. 2022;38(3), e3507.
26. Kahn HS. The "lipid accumulation product" performs better than the body mass index for recognizing cardiovascular risk: a population-based comparison. *BMC Cardiovasc Disord*. 2005;5:26.
27. Mohammadi A, Bijeh N, Moazzami M, Kazem K, Rahimi N. Effect of exercise training on spexin level, appetite, lipid accumulation product, visceral adiposity index, and body composition in adults with type 2 diabetes. *Biol Res Nurs*. 2022;24(2):152–162.
28. Laforgia J, Withers RT, Gore CJ. Effects of exercise intensity and duration on the excess post-exercise oxygen consumption. *J Sports Sci*. 2006;24(12):1247–1264.
29. Schaun GZ, Pinto SS, Praia ABD, Alberton CL. Energy expenditure and EPOC between water-based high-intensity interval training and moderate-intensity continuous training sessions in healthy women. *J Sports Sci*. 2018;36(18):2053–2060.
30. Romijn JA, Coyle EF, Sidossis LS, et al. Regulation of endogenous fat and carbohydrate-metabolism in relation to exercise intensity and duration. *Am J Physiol*. 1993;265(3):E380–E391.
31. Zouhal H, Jacob C, Delamarche P, Gratas-Delamarche A. Catecholamines and the effects of exercise, training and gender. *Sports Med*. 2008;38(5):401–423.
32. Kim GH, Lee SN, Moon DG, Kim JH, Moon KW, Yoo KD. Hemodynamic parameters of common carotid artery are associated with atherosclerotic cardiovascular disease and cardiac function. *Eur Heart J*. 2017;38, 1162-1162.
33. Green DJ, Smith KJ. Effects of exercise on vascular function, structure, and health in humans. *Cold Spring Harb Perspect Med*. 2018;8(4).
34. Souilhol C, Serbanovic-Canic J, Fragiadaki M, et al. Endothelial responses to shear stress in atherosclerosis: a novel role for developmental genes. *Nat Rev Cardiol*. 2020;17(1):52–63.
35. Phillips AT, Pankaj P, Howie CR, Usmani AS, Simpson AH. Finite element modelling of the pelvis: inclusion of muscular and ligamentous boundary conditions. *Med Eng Phys*. 2007;29(7):739–748.
36. Hernandez-Lopez P, Cilla M, Martinez M, Pena E. Effects of the haemodynamic stimulus on the location of carotid plaques based on a patient-specific mechanobiological plaque atheroma formation model. *Front Bioeng Biotechnol*. 2021;9, 690685.
37. Thijssen DH, Dawson EA, Tinken TM, Cable NT, Green DJ. Retrograde flow and shear rate acutely impair endothelial function in humans. *Hypertension*. 2009;53(6):986–992.
38. Bonafini S, Tagetti A, Giontella A, et al. Markers of arterial stiffness and subclinical vascular damages in obese children. *J Hypertens*. 2018;36. E115-E115.
39. Derevyanchenko M, Statsenko M, Fabritskaya S, Rindina Y. Arterial stiffness in patients with chronic heart failure: the role of visceral obesity. *Eur Heart J*. 2021;42, 982-982.
40. Safar ME, Czernichow S, Blacher J. Obesity, arterial stiffness, and cardiovascular risk. *J Am Soc Nephrol*. 2006;17(4 Suppl 2):S109–S111.
41. Zheng L, Zhang X, Zhu W, Chen X, Wu H, Yan S. Acute effects of moderate-intensity continuous and accumulated exercise on arterial stiffness in healthy young men. *Eur J Appl Physiol*. 2015;115(1):177–185.
42. Zou C, Sun H, Huang P. Correlativity between strain and elasticity of common carotid artery in patients with type 2 diabetes by ultrasonography. *Chin J Ultrasound Med*. 2010;26(6):542–544.
43. Kahn HS. The lipid accumulation product is better than BMI for identifying diabetes - a population-based comparison. *Diabetes Care*. 2006;29(1):151–153.
44. Pasterkamp G, Galis ZS, de Kleijn DP. Expansive arterial remodeling: location, location, location. *Arterioscler Thromb Vasc Biol*. 2004;24(4):650–657.
45. Hamburg NM, Mott MM, Bigornia SJ, et al. Maladaptive enlargement of the brachial artery in severe obesity is reversed with weight loss. *Vasc Med*. 2010;15(3):215–222.
46. Chung WB, Hamburg NM, Holbrook M, et al. The brachial artery remodels to maintain local shear stress despite the presence of cardiovascular risk factors. *Arterioscler Thromb Vasc Biol*. 2009;29(4):606–612.
47. Chatzizisis YS, Coskun AU, Jonas M, Edelman ER, Feldman CL, Stone PH. Role of endothelial shear stress in the natural history of coronary atherosclerosis and vascular remodeling: molecular, cellular, and vascular behavior. *J Am Coll Cardiol*. 2007;49(25):2379–2393.
48. Dopheide JF, Rubrech J, Trumpp A, et al. Supervised exercise training in peripheral arterial disease increases vascular shear stress and profunda femoral artery diameter. *Eur J Prev Cardiol*. 2017;24(2):178–191.
49. Park J, Park H. Effects of 6 months of aerobic and resistance exercise training on carotid artery intima media thickness in overweight and obese older women. *Geriatr Gerontol Int*. 2017;17(12):2304–2310.
50. Dinenno FA, Tanaka H, Monahan KD, et al. Regular endurance exercise induces expansive arterial remodelling in the trained limbs of healthy men. *JOURNAL OF PHYSIOLOGY-LONDON*. 2001;534(1):287–295.