Vascular Health and Risk Management

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Association Between Recovery Systolic Blood Pressure After Spot Marching Exercise and Vascular Function in Middle-Aged Adults

Teonchit Nuamchit[1], Weerapong Chidnok[2], Tomon Thongsri[3], Nuttanit Rodvinit[1], Thanaphon Chaitawong[1], Noppawit Rattanawan[1], Nisakan Juntarach[1], Noppakoon U-suwan[1], Sorrawee Lamoonkit[1], Nannalat Onchaiya[1], Paniwara Borirakwanich[1], Suwiporn Kawila[1], Pussadee Paensuwan[4], Wanvisa Treebuphachatsakul[5], Duangduan Siriwittayawan[1], Piyanuch Thitiwuthikiat[1]

[1]Department of Cardio-Thoracic Technology, Faculty of Allied Health Sciences, Naresuan University, Phitsanulok, Thailand; [2]Department of Physical Therapy, Exercise and Rehabilitation Sciences Research Unit, Faculty of Allied Health Sciences, Naresuan University, Phitsanulok, Thailand; [3]Buddhachinaraj Hospital Medical School, Phitsanulok, Thailand; [4]Department of Optometry, Faculty of Allied Health Sciences, Naresuan University, Phitsanulok, Thailand; [5]Department of Medical Technology, Faculty of Allied Health Sciences, Naresuan University, Phitsanulok, Thailand

Correspondence: Piyanuch Thitiwuthikiat, Department of Cardio-Thoracic Technology, Faculty of Allied Health Sciences, Naresuan University, Phitsanulok, 65000, Thailand, Tel +66 55 966371, Email piyanucht@nu.ac.th

Purpose: Blood pressure (BP) response observed during exercise stress tests has been recognized as a predictor of the onset of hypertension and arterial stiffness. However, access to such testing is often limited to specialized clinical settings. The purpose of this study was to assess the utility of a simple, equipment-free exercise, self-paced spot marching exercise (SME), along with the subsequent recovery BP in evaluating vascular functions.

Patients and Methods: A cross-sectional study was conducted with 107 participants aged 40–59 years, including those with and without hypertension. During the 6-min recovery period following SME, systolic BP (SBP) and diastolic BP (DBP) were measured every 2 min. Flow-mediated dilation (FMD) and cardio-ankle vascular index (CAVI) were used to evaluate endothelial function and arterial stiffness. Pearson's correlation and multiple linear regression analyses were performed to assess the associations between recovery BP and vascular parameters.

Results: Among total participants, 4-min recovery SBP was independently associated with FMD, whereas resting BP showed no such association. CAVI was not correlated with any recovery BP. In non-hypertensive individuals, 4-min recovery SBP remained independently associated with FMD, whereas age and resting SBP were linked to CAVI.

Conclusion: These findings suggest that recovery SBP following SME reflects early endothelial dysfunction and vascular impairment. Therefore, using recovery SBP after the SME demonstrates the potential to be a tool for the early detection of vascular risk in middle-aged adults.

Keywords: cardio-ankle vascular index, flow-mediated dilation, moderate-intensity exercise, recovery blood pressure

Introduction

Blood pressure (BP) responses observed during exercise have gained attention as predictive and diagnostic indicators of cardiovascular disorders, which sometimes cannot be captured by resting BP measurements. Hypertensive response to exercise (HRE) has been linked to the likelihood of developing future hypertension, myocardial infarction, autonomic dysfunction, coronary artery disease, and cardiovascular-related morbidity and mortality.[1–5] Furthermore, BP response to exercise also reflects vascular health and vascular risk.[6] Arterial stiffness was found to be significantly involved in the abnormal systolic BP (SBP) response during exercise and delayed BP recovery due to its inability to relax and reduce vascular resistance during exercise.[7] Previous studies have shown that delayed BP recovery is correlated with increased arterial stiffness in hypertensive patients compared to normotensive individuals.[8,9] Recent research indicates that cardio-

Received: 11 May 2025
Accepted: 23 July 2025
Published: 31 July 2025

ankle vascular index (CAVI), which measures overall arterial stiffness, serves as an independent predictor of an HRE in individuals who took part in the health prevention program.[10] Although the mechanism of HRE is unclear, endothelial dysfunction is believed to play a pivotal role and is the initial step in vascular impairment. Therefore, the detection of endothelial dysfunction is a focus of researchers to enhance the prevention and management of cardiovascular diseases. Previous studies have demonstrated an association between endothelial function, as measured by flow-mediated dilation (FMD), and the BP response to exercise.[11,12] A large study, the Framingham Offspring cohort, revealed a negative correlation between FMD and exercise SBP in 2,115 participants.[12]

Although HRE serves as a valuable tool for predicting future hypertension and cardiovascular risk, it has some limitations. First, BP measurement during exercise is difficult and inaccurate.[13,14] Furthermore, a consensus on an appropriate exercise regimen has not yet been established, and the maximum intensity of current protocols may not be feasible for certain populations such as older individuals. This concern has led to the ongoing adoption of an exaggerated BP response to exercise at moderate intensities.[13] Research indicates that exaggerated BP response during moderate-intensity exercise can predict hypertension and is linked to cardiovascular risk.[15] Studies have suggested that BP response to submaximal exercise provides a better prognosis than maximal exercise.[16,17] To overcome these limitations, the use of recovery BP, a potential marker of subclinical CVD,[18] following by submaximal or moderate intensity exercise may be a solution. Second, HRE is often detected during standard exercise stress testing (EST). However, EST requires specialized equipment, trained personnel, and clinical settings, making it less feasible for widespread use in community-based screening or routine monitoring. Thus, developing a simple exercise program that does not require special tools and is accessible to a particular group is challenging. Recently, a study introduced a simple and practical combined arm and leg exercise test, known as the spot marching exercise (SME), which can be performed by both adults and older individuals.[19] The studies showed that the heart rate (HR) at the end of SME was approximately 70% of the maximum HR, which offers moderate exercise intensity.[19,20] However, studies investigating the impact of immediate post-exercise recovery BP following moderate-intensity exercise as a predictor of vascular function are limited. Therefore, this study aimed to investigate the association between recovery BP following moderate-intensity SME and vascular function in middle-aged adults. We hypothesized that the recovery BP response to SME could predict vascular health, such as arterial stiffness, endothelial function, and parameters associated with cardiovascular risk in middle-aged adults.

Materials and Methods

Study Design and Participants

This cross-sectional study received approval from the Naresuan University Institutional Review Board (COA No. 495/2022) and was conducted in accordance with the principles outlined in the Declaration of Helsinki. Participants were recruited between April and October 2023. Written informed consent was obtained from all participants. The study was conducted in the Department of Cardio-Thoracic Technology, Faculty of Allied Health Sciences, Naresuan University. Participants in the middle-aged category, aged 40–59 years, were recruited for the study. A total of 107 individuals were enrolled. According to the hypertension management guideline JNC 7, participants with resting SBP $\geq$ 140 mmHg or diastolic BP (DBP) $\geq$ 90 mmHg at the time of measurement were categorized as having hypertension for subgroup analysis. This classification has also been applied to individuals with a prior diagnosis of hypertension or those currently receiving antihypertensive treatment. However, this grouping did not imply a formal clinical diagnosis of hypertension, which typically requires multiple elevated readings over time or medical confirmation. Participants diagnosed with or treated for other cardiovascular diseases, cardiac arrhythmia, or respiratory disease were excluded. Participants with a resting BP $\geq$ 160/100 mmHg on the day of SME performing were also excluded.

Participant Visits and Study Procedures

All participants were scheduled for two separate visits. The first visit was conducted to obtain informed consent and perform initial screening procedures in accordance with the study's inclusion and exclusion criteria. These procedures included medical history taking and resting BP assessment. Participants were asked to refrain from vitamins or dietary supplements, alcohol, and caffeine-containing beverages for at least 12 h, smoking for at least 6 h, and exercise for at least 24 h prior to the testing day. The second visit was conducted in the early morning, following overnight fasting for at least 8 h. Upon arrival, blood tests were conducted to measure C-peptide, insulin, uric acid, fasting blood sugar (FBS), HbA1c, creatinine, and lipid

profiles. Creatinine levels were used to determine the estimated glomerular filtration rate (eGFR) by using the CKD-EPI creatinine equation.[21] Blood tests were performed according to the manufacturer's protocol (Human Diagnostics, Worldwide, Germany). Anthropometric measurements, including body weight and body fat percentage, were obtained using a body composition monitor (Omron Karada Scan Body Composition Monitor HBF-214, Japan). Waist and hip circumferences were measured and calculated as waist-to-hip ratio (WHR). To measure resting BP and HR, participants sat quietly for 5 min before readings were taken with an automatic brachial sphygmomanometer (HEM-7130, Omron, Japan). BP readings were obtained twice, and the average of these measurements was recorded. Subsequently, the participants underwent non-invasive vascular assessments, FMD and CAVI. After a brief rest period, participants performed a SME protocol.

Self-Paced Spot Marching Exercise

The self-paced spot marching exercise (SME) was performed in a standing position followed by walking in the same spot with an arm rise of at least 90° and an alternate hip rise of at least 70° in the vertical plane.[19] Self-paced work rate was regulated by walking as fast as possible by themselves with at least speed of 70 spm for 6 min. During the exercise, participants were consistently reminded of the specific targets for their arms and knees. Participants were informed that they could stop the exercise at any time if they experienced symptom-limited discomfort, such as a breathlessness rating of 5 out of 10 or arm or leg fatigue rating of 7 out of 10. However, all participants were able to complete the 6-min self-paced SME without interruption, and none required early termination or rest during the protocol. BP response to exercise was assessed at immediate end-exercise and during the recovery phases at 2, 4, and 6 min post-exercise.

Cardio-Ankle Vascular Index Measurement

The cardio-ankle vascular index (CAVI) was assessed using a vascular screening device (VaSera1500, Fukuda Denshi Co. Ltd, Tokyo). Participants were positioned supine, with four blood pressure cuffs attached to both upper arms and ankles, two electrocardiogram electrodes placed on each wrist, and a microphone for a phonocardiogram positioned on the sternum between the second rib.[22] After a 10-minute rest period, the measurement was taken. The CAVI value was determined automatically using Equation 1, which relies on the stiffness parameter β and the Bramwell-Hill equation.

$$\mathrm{CAVI} = a\left[\frac{2\rho}{\mathrm{P_s} - \mathrm{P_d}} \times \ln\left(\frac{\mathrm{Ps}}{\mathrm{Pd}}\right) \times \mathrm{PWV}^2\right] + b \tag{1}$$

Flow-Mediated Dilation Measurement

Brachial flow-mediated dilation (FMD) was examined according to expert consensus recommendations using an ultrasound machine with a high-resolution linear probe (Affiniti50 and linear probe L12-3 MHz, Philips, USA).[23] After a 10-minute rest period, the measurement was taken. A sphygmomanometer cuff was positioned on the right forearm, 1–2 cm below the antecubital fossa. The linear probe was placed 5 cm above the antecubital fossa, color and pulsed wave Doppler were used to confirm the artery, and the artery size was adjusted to maximize the actual brachial artery diameter. The B-mode image was optimized to display an approximately 10 cm longitudinal axis and clear edges of the artery. Before cuff inflation, the baseline diameter was measured at three locations and reported as the average. The cuff was then inflated above the SBP for 50 mmHg for 5 min. The hyperemic diameter was measured at 30, 60, and 90 s after cuff deflation at the exact locations. The maximum diameter during the hyperemic phase was used to calculate the FMD. The FMD percentage was calculated using Equation 2.

$$\%\,\mathrm{FMD} = \left[\frac{\text{peak diameter} - \text{baseline diameter}}{\text{baseline diameter}}\right] \times 100 \tag{2}$$

Statistical Analysis

The Shapiro–Wilk test was utilized to assess the normality of data distribution. For data with normal distribution, continuous variables were presented as mean ± standard deviation (SD), while for non-normal distributions, they were shown as median (interquartile range, IQR). To compare two independent means, the Student's *t*-test was used for

normally distributed data, and the Mann–Whitney *U*-test was applied for non-normally distributed data. Categorical variables were represented as counts and percentages, with differences between the two groups examined using the chi-squared test. Pearson's correlation analyses were conducted to investigate the relationship between vascular parameters, including FMD and CAVI, and other clinical variables. A two-step analytic approach was employed to identify independent clinical predictors of vascular parameters. First, a grouped stepwise selection strategy was used to reduce collinearity and variable redundancy by selecting the strongest representative from predefined physiological categories: hemodynamic relevant—systolic blood pressure, metabolic relevant—blood parameters, and anthropometric variables. Clinical relevance and the selected variables from the first step were subsequently included in the stepwise multiple linear regression models for each outcome variable, FMD and CAVI, analyzed separately in the total participant group and the non-hypertensive (non-HTN) subgroup. The analysis of all data was conducted using IBM SPSS Statistics 30, with statistical significance determined at a *p*-value of less than 0.05.

Results

Demographic Characteristics of the Participants

A total of 107 individuals participated in the study. Table 1 summarizes the demographic and clinical characteristics of the total participants, as well as the non-HTN and hypertensive (HTN) subgroups. In general, individuals in the HTN group were older and had a higher BMI than those in the non-HTN group. Regarding metabolic parameters, the HTN group exhibited elevated uric acid levels. Additionally, participants in the HTN group showed a higher CAVI than those in the non-HTN group.

Blood Pressure Response to Exercise and During Recovery

Table 2 presents the SBP and DBP responses to the self-paced SME. The immediate end-exercise HR and percentage of age-predicted maximum HR were reported. The percent maximum HR values for the non-HTN and HTN groups were approximately 58% and 59%, respectively, which fall within the range of moderate-intensity exercise. Participants in the HTN group exhibited consistently higher BP values than those in the non-HTN group at all measurement time points, including the immediate end-exercise and throughout the post-exercise recovery period.

Table 1 Demographic Characteristics Stratified by Groups of Participants

Variables	Total (n=107)	Non-HTN (n=73)	HTN (n=34)	*p*-value
Age (years)	50 (9)	49 (10)	52.5 (5)	0.017
Gender (Female) (n (%))	86 (80.4%)	58 (79.5%)	28 (82.4%)	0.725
BMI (kg/m^2)	26.1 ± 4.2	25.4 ± 3.6	27.8 ± 5.0	0.005
Body fat (%)	33.9 (7.0)	33.3 (7.0)	35.5 (7.4)	0.080
WHR	0.86 ± 0.06	0.86 ± 0.06	0.86 ± 0.06	0.846
Resting HR (bpm)	74 (11)	73 (11)	75.5 (14.5)	0.128
Resting SBP (mmHg)	123.7 ± 15.9	116.3 ± 11.8	139.6 ± 10.8	< 0.001
Resting DBP (mmHg)	74 (18)	69 (13)	82.5 (11.3)	< 0.001
Laboratory tests				
C-peptide (nmol/L)	2.2 ± 0.8	2.2 ± 0.7	2.4 ± 0.9	0.132
Insulin (mIU/L)	7.7 (6.0)	7.2 (5.6)	10.4 (8.5)	0.075
Uric acid (mg/dL)	5.6 ± 1.5	5.4 ± 1.5	6.0 ± 1.3	0.045

(*Continued*)

Table 1 (Continued).

Variables	Total (n=107)	Non-HTN (n=73)	HTN (n=34)	*p*-value
FBS (mg/dL)	93 (14.0)	92 (13.5)	95 (15.8)	0.268
HbA1c (%)	5.8 (0.6)	5.8 (0.7)	5.9 (0.5)	0.517
Creatinine (mg/dL)	0.72 (0.18)	0.72 (0.16)	0.71 (0.21)	0.551
eGFR (mL/min/1.73m^2)	99.8 (16.6)	100.0 (17.1)	99.5 (20.7)	0.222
Cholesterol (mg/dL)	196.8 ± 32.5	198.1 ± 34.2	194.1 ± 28.9	0.561
Triglyceride (mg/dL)	114 (76)	114 (67)	115.5 (90.5)	0.844
HDL (mg/dL)	50.8 ± 12.5	50.8 ± 13.5	50.8 ± 10.2	0.997
LDL (mg/dL)	118.4 ± 29.7	119.6 ± 30.8	115.8 ± 27.4	0.540
Antihypertensive drugs (n (%))	12 (11.2%)	–	12 (35.3%)	< 0.001
Diabetes (n (%))	3 (2.8%)	1 (1.4%)	2 (5.9%)	0.188
Dyslipidemia (n (%))	15 (14%)	6 (8.2%)	9 (26.5%)	0.011
Smoking (n (%))	10 (9. 3%)	7 (9.6%)	3 (8.8%)	0.899
Vascular tests				
FMD (%)	7.2 ± 2.3	7.4 ± 2.3	6.6 ± 2.2	0.098
Average CAVI	7.9 ± 0.8	7.7 ± 0.8	8.2 ± 0.6	0.001

Notes: Values are displayed as mean ± SD, median (IQR), and n (%).
Abbreviations: BMI, body mass index; WHR, waist-to-hip ratio; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; FBS, fasting blood glucose; eGFR, estimated glomerular filtration rate; FMD, flow-mediated dilation; CAVI, cardio-ankle vascular index.

Table 2 Blood Pressure Response to Exercise and During Recovery Stratified by Groups of Participants

Exercise-Related Variables	Total (n=107)	Non-HTN (n=73)	HTN (n=34)	*p*-value
End-exercise HR (bpm)	100 (29)	100 (32.5)	101.5 (27)	0.514
End-exercise %HRmax (%)	58.2 (16.2)	57.8 (18.3)	59.4 (13.8)	0.841
End-exercise SBP (mmHg)	156.6 ± 21.2	149.2 ± 18.8	172.6 ± 17.3	< 0.001
End-exercise DBP (mmHg)	86.6 ± 13.8	83.4 ± 13.7	93.7 ± 11.3	< 0.001
2-min recovery SBP (mmHg)	138 (32)	127 (25)	152 (21.3)	< 0.001
2-min recovery DBP (mmHg)	86.1 ± 13.2	82.5 ± 13.3	93.9 ± 9.0	< 0.001
4-min recovery SBP (mmHg)	130 (27)	122 (25)	140.5 (22.5)	< 0.001
4-min recovery DBP (mmHg)	85.6 ± 12.4	82.4 ± 12.7	92.6 ± 8.5	< 0.001
6-min recovery SBP (mmHg)	128.6 ± 17.0	122.4 ± 13.4	141.9 ± 16.4	< 0.001
6-min recovery DBP (mmHg)	86.4 ± 12.8	82.6 ± 12.5	94.7 ± 9.1	< 0.001

Notes: Values are displayed as mean ± SD and median (IQR).
Abbreviations: HR, heart rate; %HRmax, percent maximum HR; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Correlation Between Vascular Parameters and Clinical Variables

Table 3 and Table 4 present the Pearson's correlation between vascular parameters, including FMD and CAVI, and clinical variables. For FMD, 4-min recovery SBP exhibited consistent negative associations with FMD across both the total participant group and the non-HTN subgroup. In addition, eGFR was positively correlated with FMD, particularly in the non-HTN subgroup.

Table 3 Pearson's Correlation Between Flow-Mediated Dilation (FMD) and Clinical Variables in Total Participants and Non-Hypertensive Subgroup

Clinical Variables	Total Participants		Non-HTN	
	r	*p*-value	*r*	*p*-value
Age (years)	−0.167	0.086	−0.208	0.077
Resting HR (bpm)	0.017	0.862	0.030	0.799
Resting SBP (mmHg)	−0.204	0.035	−0.137	0.247
Resting DBP (mmHg)	−0.230	0.017	−0.152	0.199
Body compositions BMI (kg/m^2)	−0.033	0.733	−0.050	0.674
Body fat (%)	−0.027	0.782	0.030	0.799
WHR	0.055	0.572	0.003	0.978
Laboratory tests C-peptide (nmol/L)	−0.042	0.667	−0.086	0.469
Insulin (mIU/L)	0.005	0.963	0.046	0.702
Uric acid (mg/dL)	−0.092	0.347	−0.114	0.335
FBS (mg/dL)	0.123	0.207	0.159	0.180
HbA1c (%)	0.021	0.828	−0.015	0.899
Creatinine (mg/dL)	−0.005	0.955	−0.109	0.359
eGFR (mL/min/1.73m^2)	0.095	0.332	0.235	0.046
Cholesterol (mg/dL)	−0.097	0.318	−0.056	0.637
Triglyceride (mg/dL)	−0.034	0.730	−0.060	0.614
HDL (mg/dL)	−0.077	0.431	−0.092	0.439
LDL (mg/dL)	−0.058	0.551	0.004	0.973
Exercise SBPs End-exercise SBP (mmHg)	−0.171	0.077	−0.114	0.336
2-min recovery SBP (mmHg)	−0.215	0.026	−0.187	0.114
4-min recovery SBP (mmHg)	−0.258	0.007	−0.241	0.040
6-min recovery SBP (mmHg)	−0.209	0.031	−0.181	0.126

Notes: Correlation coefficients (*r*) and *p*-values are presented for each variable in both groups.
Abbreviations: BMI, body mass index; WHR, waist-to-hip ratio; HR, heart rate; % HRmax, percent maximum HR; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; FBS, fasting blood glucose; eGFR, estimated glomerular filtration rate; FMD, flow-mediated dilation; CAVI, cardio-ankle vascular index.

Table 4 Pearson's Correlation Between Cardio-Ankle Vascular Index (CAVI) and Clinical Variables in Total Participants and Non-Hypertensive Subgroup

Clinical Variables	Total Participants		Non-HTN	
	r	*p*-value	*r*	*p*-value
Age (years)	0.402	< 0.001	0.350	0.002
Resting HR (bpm)	0.009	0.923	−0.065	0.587
Resting SBP (mmHg)	0.432	< 0.001	0.381	0.001
Resting DBP (mmHg)	0.297	0.002	0.160	0.177
Body compositions				
BMI (kg/m^2)	0.041	0.674	−0.049	0.683
Body fat (%)	0.019	0.849	−0.137	0.249
WHR	0.258	0.007	0.283	0.015
Laboratory tests				
C-peptide (nmol/L)	0.266	0.006	0.294	0.012
Insulin (mIU/L)	0.079	0.416	0.002	0.987
Uric acid (mg/dL)	0.166	0.087	0.152	0.198
FBS (mg/dL)	0.035	0.717	0.121	0.308
HbA1c (%)	0.060	0.539	0.102	0.390
Creatinine (mg/dL)	0.008	0.934	0.071	0.549
eGFR (mL/min/1.73m^2)	−0.179	0.065	−0.251	0.032
Cholesterol (mg/dL)	−0.090	0.356	−0.108	0.363
Triglyceride (mg/dL)	0.117	0.231	0.145	0.222
HDL (mg/dL)	−0.212	0.029	−0.224	0.056
LDL (mg/dL)	−0.067	0.490	−0.088	0.458
Exercise SBPs				
End-exercise SBP (mmHg)	0.229	0.018	0.136	0.252
2-min recovery SBP (mmHg)	0.252	0.009	0.161	0.172
4-min recovery SBP (mmHg)	0.343	< 0.001	0.262	0.025
6-min recovery SBP (mmHg)	0.397	< 0.001	0.309	0.008

Notes: Correlation coefficients (*r*) and *p*-values are presented for each variable in both groups.

Abbreviations: BMI, body mass index; WHR, waist-to-hip ratio; HR, heart rate; % HRmax, percent maximum HR; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; FBS, fasting blood glucose; eGFR, estimated glomerular filtration rate; FMD, flow-mediated dilation; CAVI, cardio-ankle vascular index.

CAVI showed significant positive correlations with age, resting SBP, WHR, C-peptide level, and 4-min recovery SBP in both groups. In the total participant group, HDL was negatively correlated with CAVI, whereas eGFR was negatively correlated with CAVI in the non-HTN subgroup.

Table 5 Multiple Linear Regression Analyses of Flow-Mediated Dilation (FMD) Stratified by Participant Groups

Independent Predictors	Total Participants			Non-HTN		
	B (95% CI)	β	*p*-value	B (95% CI)	β	*p*-value
4-min recovery SBP	−0.034 (−0.058, −0.009)	−0.258	0.007	−0.037 (−0.072, −0.002)	−0.241	0.040
Adjusted R^2	0.058			0.045		
Model *p*-value	0.007			0.040		

Notes: Unstandardized coefficients (B) with 95% Confidence Interval (95% CI), standardized coefficients (β), and *p*-values are presented for each predictor variable in both groups. Adjusted R^2 and model *p*-values are reported to indicate the overall model performance. Adjusted variables: Total participant group – age, gender, body mass index (BMI), resting systolic blood pressure (SBP), and C-peptide; Non-HTN subgroup – age, gender, BMI, resting SBP, C-peptide, and estimated glomerular filtration rate (eGFR).

Table 6 Multiple Linear Regression Analyses of Cardio-Ankle Vascular Index (CAVI) Stratified by Participant Groups

Independent Predictors	Total Participants			Non-HTN		
	B (95% CI)	β	*p*-value	B (95% CI)	β	*p*-value
Resting SBP	0.016(0.008, 0.025)	0.331	< 0.001	0.025(0.011, 0.039)	0.371	0.001
Age	0.045(0.021, 0.070)	0.313	< 0.001	0.048(0.019, 0.077)	0.338	0.002
WHR	2.625(0.341, 4.909)	0.188	0.025	–	–	–
Adjusted R^2	0.297			0.239		
Model *p*-value	< 0.001			< 0.001		

Notes: Unstandardized coefficients (B) with 95% confidence interval (CI), standardized coefficients (β), and *p*-values are presented for each predictor variable in both groups. Adjusted R^2 and model *p*-values are reported to indicate the overall model performance. Adjusted variables: Total participant group – age, gender, waist-to-hip ratio (WHR), resting systolic blood pressure (SBP), C-peptide, insulin, and high-density lipoprotein cholesterol (HDL); Non-HTN subgroup – age, gender, WHR, resting SBP, C-peptide, estimated glomerular filtration rate (eGFR), and insulin.

Multiple Regression Analysis for Predictors of FMD and CAVI

Table 5 and Table 6 present the results of multiple linear regression analyses conducted using the stepwise approach to determine clinical predictors of FMD and CAVI in both the total participant group and the non-HTN subgroup. According to FMD, the final regression model for both groups revealed that a 4-min recovery SBP was negatively associated with FMD. The final regression model showed that age and resting SBP were positively associated with CAVI in both groups. Additionally, in the total participant group, WHR was positively associated with CAVI. Figure 1 illustrates the relationship between vascular parameters, FMD and CAVI, and BP variables.

Discussion

This study employed SME, an equipment-free physical activity, along with post-exercise recovery BP measurements to evaluate endothelial function and arterial stiffness. The results show that recovery SBP at 4 min post-SME is inversely related to FMD, suggesting potential links among post-exercise hemodynamics and vascular function.

This study included 107 participants with varying BP levels ranging from normotension to mild hypertension. While both total participants and the non-HTN subgroup were analyzed, the key statistical findings were primarily focused on the non-HTN group, allowing clearer examination of the associations between vascular function and post-exercise BP recovery across different BP profiles. FMD was assessed independently before the exercise session. A key finding is that the significant negative association between 4-min recovery SBP and FMD was observed even among non-HTN participants, suggesting that impaired post-exercise recovery may reflect early endothelial dysfunction despite normal or borderline resting BP. This finding aligns with

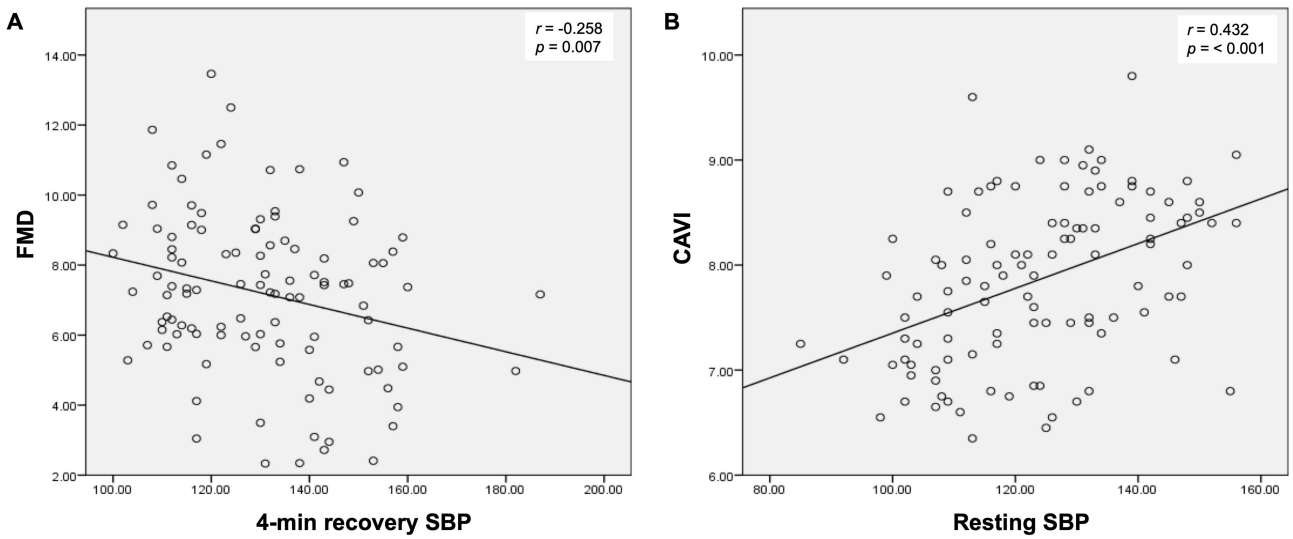

Figure 1 Scatter plots illustrating the associations between vascular parameters and systolic blood pressure (SBP). (**A**) shows the negative correlation between flow-mediated dilation (FMD) and 4-min recovery SBP. (**B**) shows the positive correlation between cardio-ankle vascular index (CAVI) and resting SBP.

previous research indicating that delayed post-exercise blood pressure recovery is linked to impaired vascular health and autonomic dysfunction.[24–26] A slower SBP decline after exercise may reflect reduced nitric oxide (NO) bioavailability and endothelial dysfunction, as FMD is a widely accepted indicator of endothelial health.[27] The finding that non-HTN participants with slower SBP recovery had lower FMD suggests that post-exercise BP patterns may help identify early endothelial dysfunction before hypertension becomes more clinically apparent. This supports the idea that post-exercise recovery BP could serve as a sensitive functional marker of vascular health independent of traditional BP classifications. These results align with previous research showing that delayed BP recovery after exercise is predictive of future hypertension, major adverse cardiovascular events, and mortality even in participants with normal resting BP.[12,28]

The finding that recovery SBP at 4 min post-exercise is significantly associated with FMD suggests that this time point may represent a critical physiological window during which endothelial function influences BP regulation. This timing is important because, after exercise, BP recovery depends on the balance between reduced sympathetic activity and reactivation of the parasympathetic system.[26] Better endothelial function, indicated by higher FMD, promotes faster peripheral vasodilation, lowers vascular resistance, and helps BP to return to baseline more quickly. Since delayed SBP recovery is linked to cardiovascular risk, slower BP recovery associated with lower FMD could indicate early signs of vascular dysfunction.[12,24,29,30] The 4-min mark in post-exercise BP recovery likely reflects a phase in which BP decreases due to vasodilation, making it a useful point for assessing how endothelial function influences BP regulation. The 4-min post-exercise time point was supported based on accumulating evidence showing its physiological relevance in assessing vascular recovery. SBP recovery was significantly delayed beyond 3 min, especially in the uncontrolled HTN group, which showed no notable change in SBP recovery over 7 min of recovery. Although derived from a chronic kidney disease cohort, evidence indicates that delayed SBP recovery is linked to impaired autonomic regulation and sustained vasoconstriction, particularly among individuals with hypertension.[31] Similarly, a large-scale study reported that SBP recovery in healthy, normotensive adults continues well beyond 3 min, with average recovery times ranging from 5 to 7 min depending on age, sex, and exercise intensity.[32] More recently, Lee et al (2023) found that recovery SBP at 4 min post-exercise was significantly associated with arterial stiffness and autonomic dysfunction in middle-aged adults.[26] Both SBP recovery and HRE are considered markers of hemodynamic regulation and vascular responsiveness during and after exertion.[29,33] However, recovery SBP may be a more precise and practical predictor, especially for evaluating vascular function and early cardiovascular risk. Compared to HRE, the recovery SBP assessed by SME offers a simpler and safer alternative, requiring only moderate effort and allowing for more stable BP measurement during the recovery phase. In contrast, HRE relies on peak-exercise BP, which is often prone to recording errors due to motion artifacts and cuff displacement.[12,20,33,34] Previous research has reported that SBP measurements at peak exercise can be

imprecise, with potential errors reaching up to 40 mmHg due to these technical limitations.[14] Moreover, SME elicits moderate-intensity activity (≈ 4.4 METs) through the activation of large muscle groups, including the legs, core, and arms, thereby increasing energy demand. It also imposes a greater cardiovascular load, resulting in elevated HR and BP responses.[19,35] Prior research indicates that low-to-moderate exercise intensity may more accurately reflect the cardiovascular demands encountered in daily life.[33] The present study demonstrated that recovery SBP at 4 min is associated with FMD, while resting BP is not. This suggests that post-exercise BP dynamics offer an additional predictive value beyond resting BP measures. Accordingly, the use of SME to derive the recovery SBP appears appropriate for the initial screening of cardiovascular risk markers, particularly in preclinical or middle-aged populations.

Moreover, the findings indicate that CAVI was significantly related to age and resting SBP in both groups. Importantly, because CAVI is measured at rest, it provides a non-invasive, pre-exercise estimate of arterial stiffness. As the CAVI reflects the structural characteristics of the arteries and is minimally influenced by acute hemodynamic variations, measuring it prior to exercise ensures that the results remain unaffected by transient fluctuations in BP or HR induced by physical activity. The strong correlation between CAVI and age in both the total participants and the non-HTN subgroup suggests that arterial stiffening occurs as part of physiological aging, even in the absence of hypertension, highlighting that arterial stiffness increases over time, even in the absence of apparent cardiovascular disease.[36] Therefore, CAVI may serve as a useful indicator of early age-related vascular changes, even before the onset of clinical hypertension. Elevated resting SBP is a well-established consequence of increased arterial stiffness because stiffer arteries lead to higher pulse wave velocity (PWV) and reduced arterial compliance.[37] Although a previous study linked elevated CAVI (CAVI > 8) to hypertensive responses to exercise and impaired SBP recovery,[10] no significant association was observed in our study. This may be explained by the relatively low arterial stiffness in our cohort, as reflected by a mean CAVI < 8, indicating a preserved vascular structure. Additionally, SBP recovery may be more influenced by dynamic physiological mechanisms, such as autonomic reactivation and endothelial vasodilation, than by baseline arterial stiffness, particularly under moderate-intensity exercise conditions. While CAVI remains a valuable tool for assessing long-term arterial health, it may be less effective in detecting early impairments in BP regulation following exercise. In contrast, FMD appears to be more sensitive in capturing early vascular dysfunction, as evidenced by its association with post-exercise recovery BP.

SME, a simple, equipment-free, self-paced aerobic activity performed in place, offers a practical and low-impact option for cardiovascular assessment or training, particularly in resource-limited or home-based settings. Although SME appears safe and feasible for the general population, its application in hypertensive individuals has not been widely studied. However, evidence supports the safety and effectiveness of similar low-to-moderate-intensity home-based exercise programs. A systematic review of 27 trials reported significant BP reductions following home-based endurance, isometric strength, and respiratory exercises with no major adverse events.[38] Similarly, a 12-week low-intensity walking program improved BP, HR variability, and baroreflex sensitivity in mildly hypertensive adults.[39] Isometric handgrip training, comparable in intensity to SME, has also shown consistent BP-lowering effects in hypertensive populations.[40] These findings support the presumed safety of SME in this group, though dedicated trials are needed to confirm its efficacy and safety in individuals at elevated cardiovascular risk.

Limitations and Perspectives for Future Research

1. This study focused on middle-aged adults (40–59 years), which may not be applicable to younger or older populations. Blood pressure recovery responses may differ across age groups, as endothelial function and arterial stiffness vary throughout the lifespan. Expanding the study to include a broader age range and a larger cohort could enhance the generalizability of the results.
2. This study is predominance of female participants, which may limit the generalizability of the findings. Sex-related physiological differences, such as those involving vascular tone, autonomic regulation, endothelial function, and BP responses to exercise, could influence the observed outcomes. Future studies should aim for a more balanced sex distribution to better account for these potential differences.
3. The SME is a moderate-intensity, self-paced activity which may result in variability in exercise intensity across participants. Differences in fitness levels could influence BP recovery and vascular responses, thereby introducing individual variability into the results. Compared to standardized treadmill or cycling protocols, SME may offer less precise control over exercise intensity.

4. Dietary habits, physical activity levels, and sleep quality were not controlled in this study but could influence vascular function and BP recovery. A more detailed analysis of medication use, diet, and physical activity could help to adjust for confounding variables.

Clinical Implications

This study integrated SME, recovery BP, FMD, and CAVI to evaluate cardiovascular health. These findings have important clinical implications for early vascular assessment, risk stratification, and intervention strategies. The SME is an equipment-free, accessible exercise protocol that can be used in clinical and community settings to assess cardiovascular function. Unlike high-intensity exercise tests, the SME is safe and feasible for populations with different fitness levels including older individuals. The association between recovery SBP and FMD suggests that vascular factors influence BP regulation after exercise. Measuring recovery SBP at 4 min post-exercise could serve as a simple screening tool for detecting early endothelial dysfunction. FMD assessment, along with recovery SBP measurement, could help identify individuals at risk of hypertension or cardiovascular disease before symptom development.

Conclusion

In summary, recovery SBP at 4 min post-exercise appears to reflect vascular function, as evidenced by its inverse association with FMD. These findings support the potential utility of post-exercise SBP as an early integrative tool for risk stratification in endothelial dysfunction, particularly in middle-aged populations.

Acknowledgments

This work was supported by the National Science, Research and Innovation Fund (NSRF), Thailand [grant number R2566B084].

Disclosure

The authors declare that they have no competing interests for this work.

References

1. Gottdiener JS, Brown J, Zoltick J, Fletcher RD. Left ventricular hypertrophy in men with normal blood pressure: relation to exaggerated blood pressure response to exercise. *Ann Intern Med*. 1990;112(3):161–166. doi:10.7326/0003-4819-112-3-161
2. Wiech P, Würzburger L, Rossi V, Caselli S, Schmied C, Niederseer D. Hypertensive response to exercise, hypertension and heart failure with preserved ejection fraction (HFpEF)-a continuum of disease? *Wien Klin Wochenschr*. 2023;135(23–24):685–695. doi:10.1007/s00508-023-02195-3
3. Lovic D, Narayan P, Pittaras A, Faselis C, Doumas M, Kokkinos P. Left ventricular hypertrophy in athletes and hypertensive patients. *J Clin Hypertens*. 2017;19(4):413–417. doi:10.1111/jch.12977
4. Mottram PM, Haluska B, Yuda S, Leano R, Marwick TH. Patients with a hypertensive response to exercise have impaired systolic function without diastolic dysfunction or left ventricular hypertrophy. *J Am Coll Cardiol*. 2004;43(5):848–853. doi:10.1016/j.jacc.2003.08.057
5. Laukkanen JA, Kurl S, Salonen R, TA L, Rauramaa R, Salonen JT. Systolic blood pressure during recovery from exercise and the risk of acute myocardial infarction in middle-aged men. *Hypertension*. 2004;44(6):820–825. doi:10.1161/01.HYP.0000148460.95060.f2
6. Nayor M, Gajjar P, Murthy VL, et al. Blood pressure responses during exercise: physiological correlates and clinical implications. *Arterioscler Thromb Vasc Biol*. 2023;43(1):163–173. doi:10.1161/ATVBAHA.122.318512
7. Saz-Lara A, Cavero-Redondo I, Álvarez-Bueno C, Notario-Pacheco B, Ruiz-Grao M, Martínez-Vizcaíno V. The acute effect of exercise on arterial stiffness in healthy subjects: a meta-analysis. *J Clin Med*. 2021;10(2):291. doi:10.3390/jcm10020291
8. Kontsas K, Triantafyllidi H, Trivilou P, et al. Delayed blood pressure recovery ratio might indicate increased arterial stiffness in hypertensive patients with reduced aerobic exercise capacity. *Blood Press*. 2013;22(5):290–296. doi:10.3109/08037051.2012.759694
9. Tsioufis C, Dimitriadis K, Thomopoulos C, et al. Exercise blood pressure response, albuminuria, and arterial stiffness in hypertension. *Am J Med*. 2008;121(10):894–902. doi:10.1016/j.amjmed.2008.05.035
10. Wuttichaipradit C, Yodwut C, Sukhum P, et al. CAVI (Cardio-Ankle Vascular Index) as an independent predictor of hypertensive response to exercise. *BMC Cardiovasc Disord*. 2024;24(1):165. doi:10.1186/s12872-024-03807-0
11. Olson KM, Augeri AL, Seip RL, Tsongalis GJ, Thompson PD, Pescatello LS. Correlates of endothelial function and the peak systolic blood pressure response to a graded maximal exercise test. *Atherosclerosis*. 2012;222(1):202–207. doi:10.1016/j.atherosclerosis.2012.01.027
12. Thanassoulis G, Lyass A, Benjamin EJ, et al. Relations of exercise blood pressure response to cardiovascular risk factors and vascular function in the Framingham Heart Study. *Circulation*. 2012;125(23):2836–2843. doi:10.1161/CIRCULATIONAHA.111.063933
13. Kosowski W, Aleksandrowicz K. Hypertensive response to exercise as an early marker of disease development. *Biomedicines*. 2025;13(1):30. doi:10.3390/biomedicines13010030
14. McHam SA, Marwick TH, Pashkow FJ, Lauer MS. Delayed systolic blood pressure recovery after graded exercise: an independent correlate of angiographic coronary disease. *J Am Coll Cardiol*. 1999;34(3):754–759. doi:10.1016/S0735-1097(99)00269-7

15. Schultz MG, Picone DS, Nikolic SB, Williams AD, Sharman JE. Exaggerated blood pressure response to early stages of exercise stress testing and presence of hypertension. *J Sci Med Sport*. 2016;19(12):1039–1042. doi:10.1016/j.jsams.2016.04.004
16. Schultz MG, La Gerche A, Sharman JE. Blood pressure response to exercise and cardiovascular disease. *Curr Hypertens Rep*. 2017;19(11):89. doi:10.1007/s11906-017-0787-1
17. Schultz MG, Otahal P, Picone DS, Sharman JE. Clinical relevance of exaggerated exercise blood pressure. *JACC*. 2015;66(16):1843–1845. doi:10.1016/j.jacc.2015.08.015
18. Lee J, Vasan RS, Xanthakis V. Association of blood pressure responses to submaximal exercise in midlife with the incidence of cardiovascular outcomes and all-cause mortality: the Framingham Heart Study. *J Am Heart Assoc*. 2020;9(11):e015554. doi:10.1161/JAHA.119.015554
19. Jones C, Phimphasak C, Pukdeechat M, Jones D. Spot marching exercise for home-based pulmonary rehabilitation in COPD: physical performance and quality of life. *Eur Respir J*. 2018;52(suppl 62):PA3651.
20. Sitthiracha P, Eungpinichpong W, Chatchawan U. Effect of progressive step marching exercise on balance ability in the elderly: a cluster randomized clinical trial. *Int J Environ Res Public Health*. 2021;18(6):3146. doi:10.3390/ijerph18063146
21. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med*. 2009;150(9):604–612. doi:10.7326/0003-4819-150-9-200905050-00006
22. Asmar R. Principles and usefulness of the cardio-ankle vascular index (CAVI): a new global arterial stiffness index. *Eur Heart J Suppl*. 2017;19 (suppl_B):B4–B10. doi:10.1093/eurheartj/suw058
23. Thijssen DHJ, Bruno RM, van Mil ACCM, et al. Expert consensus and evidence-based recommendations for the assessment of flow-mediated dilation in humans. *Eur Heart J*. 2019;40(30):2534–2547. doi:10.1093/eurheartj/ehz350
24. Mahfouz RA, Dewedar A, Elawady W, Salem A, Goda M. Delayed blood pressure recovery ratio and its relation to endothelial function and left ventricular diastolic function in prediabetics. *Echocardiography*. 2014;31(7):858–864. doi:10.1111/echo.12489
25. Nishiyama Y, Sakaue A, Matsuo S, et al. Systolic blood pressure during recovery from exercise is related to flow-mediated dilatation in patients with coronary artery disease. *Heart Asia*. 2014;6(1):83–86. doi:10.1136/heartasia-2013-010457
26. Lee H, Kim H, Oh S, et al. Delayed blood pressure recovery after exercise stress test is associated with autonomic dysfunction and pulse pressure in a middle-aged healthy group. *PLoS One*. 2023;18(10):e0285961. doi:10.1371/journal.pone.0285961
27. Thijssen DH, Black MA, Pyke KE, et al. Assessment of flow-mediated dilation in humans: a methodological and physiological guideline. *Am J Physiol Heart Circ Physiol*. 2011;300(1):H2–12. doi:10.1152/ajpheart.00471.2010
28. Campbell R, Fisher JP, Sharman JE, McDonnell BJ, Frenneaux MP. Contribution of nitric oxide to the blood pressure and arterial responses to exercise in humans. *J Hum Hypertens*. 2011;25(4):262–270. doi:10.1038/jhh.2010.53
29. Costa EC, Boreskie KF, Scott Kehler D, et al. Immediate post-exercise blood pressure and arterial compliance in middle-aged and older normotensive females: a cross-sectional study. *Sci Rep*. 2020;10(1):9205. doi:10.1038/s41598-020-66104-8
30. Costa EC, Browne RAV, Câmara M, et al. Immediate post-exercise blood pressure and arterial stiffness in hypertensive and normotensive older females. *J Clin Hypertens*. 2022;24(6):704–712. doi:10.1111/jch.14490
31. Agarwal R, Pappas MK. Delayed systolic blood pressure recovery following exercise as a mechanism of masked uncontrolled hypertension in chronic kidney disease. *Nephrol Dial Transplant*. 2017;32(10):1710–1717. doi:10.1093/ndt/gfw266
32. Dimpka U, Ugwu AC. Determination of systolic blood pressure recovery time after exercise in apparently healthy, normotensive, non-athletic adults and the effects of age, gender, and exercise intensity. *Int J Exerc Sci*. 2009;2(2):115–130. doi:10.70252/MLAP9661
33. Kim D, Ha JW. Hypertensive response to exercise: mechanisms and clinical implication. *Clin Hypertens*. 2016;22:17. doi:10.1186/s40885-016-0052-y
34. Ellestad MH. Reliability of blood pressure recordings. *Am J Cardiol*. 1989;63(13):983–985. doi:10.1016/0002-9149(89)90153-7
35. Ainsworth BE, Haskell WL, Herrmann SD, et al. 2011 compendium of physical activities: a second update of codes and MET values. *Med Sci Sports Exerc*. 2011;43(8):1575–1581. doi:10.1249/MSS.0b013e31821ece12
36. Matsushita K, Ding N, Kim ED, et al. Cardio-ankle vascular index and cardiovascular disease: systematic review and meta-analysis of prospective and cross-sectional studies. *J Clin Hypertens*. 2019;21(1):16–24. doi:10.1111/jch.13425
37. Ghosh A, Dharmarajan A, Swain PK, Das D, Verma P, Tripathy PR. Impact of cardiovascular factors on pulse wave velocity and total vascular resistance in different age group patients with cardiovascular disorders. *Curr Aging Sci*. 2019;11(4):261–268. doi:10.2174/1874609812666190226151500
38. Rodrigues GD, Lima LS, da Silva NCS, et al. Are home-based exercises effective to reduce blood pressure in hypertensive adults? A systematic review. *Clin Hypertens*. 2022;28(1):28. doi:10.1186/s40885-022-00211-8
39. Hua LP, Brown CA, Hains SJ, Godwin M, Parlow JL. Effects of low-intensity exercise conditioning on blood pressure, heart rate, and autonomic modulation of heart rate in men and women with hypertension. *Biol Res Nurs*. 2009;11(2):129–143. doi:10.1177/1099800408324853
40. Cornelissen VA, Smart NA. Exercise training for blood pressure: a systematic review and meta-analysis. *J Am Heart Assoc*. 2013;2(1):e004473. doi:10.1161/JAHA.112.004473