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ORIGINAL PAPER

Association between arterial stiffness and autonomic recovery following graded aerobic exercise in healthy young adults – an exploratory pilot study

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ABSTRACT

Introduction and aim. Arterial stiffness reflects vascular properties that can influence blood pressure (BP) and heart rate (HR) responses to exercise, but its association with post-exercise autonomic recovery in healthy young adults remains unclear. This exploratory pilot study investigated the relationship between baseline aortic stiffness and post-exercise hemodynamic and autonomic recovery following graded submaximal aerobic exercise.

Material and methods. Thirty healthy young adults (17 women, 13 males; mean age 22.7±2.5 years) underwent baseline evaluation of carotid–femoral pulse wave velocity (cf-PWV), central BP, and pulse wave analysis. Participants completed a graded submaximal cycling protocol from 20 to 80% of the maximum oxygen uptake in 3-minute stages, followed by 5 minutes of seated recovery. HR was continuously recorded and BP measured at 1, 3, and 5 minutes of recovery. Autonomic recovery was assessed using heart rate recovery (HRR) and heart rate variability (HRV) indices. Associations were analyzed using correlation and multivariate linear regression adjusted for age, sex, and body mass index.

Results. Higher baseline cf-PWV was associated with higher peak HR and systolic BP during exercise ($p<0.01$), and slower HRR at 1 min ($r=-0.517$, $p=0.002$). Cf-PWV independently predicted early HRR after adjustment.

Conclusion. In this pilot sample of healthy young adults, greater aortic stiffness was associated with higher BP responses during exercise and slower early autonomic recovery. These findings suggest an association between vascular properties and early postexercise recovery, which should be confirmed in larger studies.

Keywords. aortic stiffness, carotid–femoral pulse wave velocity, autonomic recovery, heart rate variability, heart rate recovery, submaximal aerobic exercise

Abbreviations

AIx – augmentation index, **AIx@75** – heart rate–corrected augmentation index, **AP** – augmentation pressure, **BMI** – body mass index, **BP** – blood pressure, **cDBP** – central diastolic blood pressure, **cf-PWV** - carotid - femoral pulse wave velocity, **cSBP** – central systolic blood pressure, **DBP** – diastolic blood pressure, **ECG** – electrocardiogram, **HF** - high frequency power, **HFnu** – high-frequency normalized units, **HR** – heart rate, **HRpeak** – peak heart rate, **HRR** – heart rate recovery, **HRR1** – heart rate recovery at 1 minute, **HRR3** – heart rate recovery at 3 minutes, **HRR5** – heart rate recovery at 5 minutes, **HRV** – heart rate variability, **LF** - low frequency power, **LF/HF** – low-frequency to

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high-frequency ratio, **LFnu** – low-frequency normalized units, **PP** – pulse pressure, **PWV** – pulse wave velocity, **PWA** – pulse wave analysis, **RMSSD** – root mean square of successive differences, **RR** – RR interval, **SBP** – systolic blood pressure, **SDNN** – standard deviation of normal-to-normal intervals, **VO_2max** – maximal oxygen uptake

Introduction

The importance of arterial integrity for cardiovascular health has long been recognized, famously captured by Thomas Sydenham's assertion that 'a man is as old as his arteries".[1] In modern preventive medicine, arterial function is seen not only as a marker of vascular aging, but also as a determinant of physiological resilience to hemodynamic stress. Physical activity, a potent modulator of arterial structure and function, therefore, provides a useful physiological model to understand early vascular adaptations in healthy young adults.[1,2]

Arterial stiffness is a robust surrogate marker of vascular aging.[3] By influencing central blood pressure, ventricular afterload, and coronary perfusion, aortic stiffness plays an important role in cardiovascular adaptation to physiological stress.[3,4] Pulse wave velocity (PWV) is the gold standard noninvasive measure of aortic stiffness, with carotid–femoral PWV (cf-PWV) specifically reflecting central hemodynamics and possible cardiovascular risk.[4,5]

Wave reflection indices, including augmentation index (AIx), heart rate corrected AIx (AIx@75), and augmentation pressure (AP), provide additional information on ventricular–vascular coupling and mechanical load on the left ventricle.[6-8] During and after exercise, cardiovascular homeostasis depends on coordinated interactions between vascular function and autonomic regulation dynamically modulating heart rate (HR), vascular tone and central hemodynamics.[9,10] These integrated autonomic vascular responses can be monitored non-invasively via heart rate variability (HRV), heart rate recovery (HRR), cfPWV, AIx@75, and central blood pressures (BP), including central systolic BP (cSBP), central diastolic BP (cDBP), and pulse pressure (PP), offering a comprehensive view of cardiovascular recovery after exercise.[9-11]

Although acute aerobic exercise responses to arterial stiffness and autonomic function have been studied, the extent to which baseline aortic stiffness is early after exercise autonomic and hemodynamic recovery remains insufficiently explored.[11,12] Baseline assessment of arterial stiffness may provide additional information on interindividual differences in vascular responses to physiological stress.[13,14] Most previous studies have focused on resting measures or acute changes during exercise without integrating preexercise vascular properties with recovery dynamics. Investigating this relationship in healthy young adults can provide insight into early physiological variability in vascular-autonomic interactions, even in the absence of overt diseases. Such findings may serve as a reference framework for future studies in populations with increased metabolic risk.

Aim

This exploratory pilot study aimed to assess whether baseline aortic stiffness, measured by cf-PWV prior to exercise, is associated with early post-exercise cardiovascular and autonomic recovery in healthy young adults undergoing graded submaximal cycling. As secondary exploratory analyses, we examined whether a higher baseline aortic stiffness is associated with a slower post-exercise HRR and altered autonomic regulation during recovery, and a higher peak HR and peak systolic BP achieved during graded submaximal exercise. We also explore whether cf-PWV independently predicts early HRR after adjustment for age, sex, and body mass index (BMI).

Material and methods

Ethical considerations

The study was conducted in accordance with the Declaration of Helsinki and all relevant national guidelines for research involving human participants. Before data collection, ethical approval was obtained from the Medical Ethics Committee of the Republic of Slovenia (approval number: 0120-388/2025-2711-5). All participants were fully informed about the aims, procedures, and potential risks of the study and provided their written informed consent prior to enrollment. Participation was voluntary and participants were allowed to withdraw from the study at any time without consequences. All collected data were fully anonymized prior to analysis, with any direct or indirect personal identifiers removed or replaced by coded numerical identifiers. Data were stored on secure servers and handled exclusively by authorized research personnel. All procedures for data storage, processing, and protection complied with the applicable local legislation on personal data protection.

Study design

This study was carried out as an exploratory pilot investigation using a prospective observational design under controlled laboratory conditions. It represents the healthy reference cohort of a larger ongoing clinical research project that will subsequently include hypertensive patients with and without impaired autonomic regulation. Within this framework, the present phase aimed to examine associations between baseline aortic stiffness and post-exercise hemodynamic and autonomic recovery in healthy young adults. No formal sample size calculation was performed due to the pilot nature of the study. The sample size was determined pragmatically based on feasibility and is consistent with recommen-

dations for exploratory physiological studies. Given its hypothesis-generating nature and limited sample size, this pilot phase was intended to provide preliminary information and inform the refinement of experimental protocols and analytical approaches for future large-scale investigations.

Participants

A total of 30 apparently healthy adult volunteers (17 women, 13 males; mean age 22.7±2.5 years, mean BMI 22.3±1.82 kg/m^2) were enrolled in the study. Participants were recruited through campus postings, announcements distributed through the university student email list, and targeted outreach within university-affiliated organizations. The sampling approach did not involve randomization; instead, convenience sampling was used, relying on individuals who voluntarily expressed interest in participating. Eligible participants were required to be between 18 and 30 years of age, non-smokers, free of any history of acute or chronic cardiovascular, metabolic or musculoskeletal disease, not taking any supplements or medications that could influence vascular or autonomic function, and regularly physically active without previous exertional symptoms. All participants underwent a resting electrocardiogram (ECG) and an orthostatic test, both of which yielded normal findings.

Pulse-wave analysis and pulse-wave velocity

Central hemodynamic parameters, PWV and PWA were obtained during the baseline supine assessment using the SphygmoCor XCEL device (Cardiex, Sydney, Australia). All measurements were made after at least 5 minutes of rest in the supine position to ensure hemodynamic stability. The assessment protocol enabled the comprehensive acquisition of central blood pressure and waveform-derived indices, including measures of arterial stiffness and wave reflection. For the purposes of the present analysis, cf-PWV was used as the primary index of aortic stiffness, alongside selected central hemodynamic and PWA parameters that are commonly reported and relevant to vascular evaluation. These included cSBP, cDBP, PP, AIx, and AIx@75. Other waveform-derived parameters obtained during the assessment were recorded but not included in the current analyzes. PWA was performed using an upper arm cuff while participants remained supine, allowing non-invasive estimation of central BP and augmentation indices. cf-PWV was measured using the same device, with a thigh cuff positioned over the femoral artery and carotid waveforms obtained by applanation tonometry. Transit distances were determined using standardized surface measurements. Specifically, three anatomical distances were measured: (1) the distance from the carotid artery pulse site to the suprasternal notch, (2) the distance from the suprasternal notch to the upper edge of the cuff, and (3) the distance from the femoral artery pulse site to the upper edge of the cuff. The device software to calculate cf-PWV based on the pulse transit time between the carotid and femoral recording sites and the corresponding anatomical path length. This combined baseline assessment provided an integrated characterization of aortic stiffness and central hemodynamic before the graded submaximal exercise test. All SphygmoCor measurements were subject to device-specific quality control criteria, and only recordings that met the recommended quality thresholds were accepted for analysis. Quality control was performed for each individual measurement.

Exercise stress test

The graded cycling exercise protocol was carried out using a cycle ergometer (Ergosana ERG 911 plus; Schiller AG, Baar, Switzerland) in the exercise physiology laboratory of the outpatient clinic for hypertensive patients at Dr. Peter Draj Hospital, part of the University Medical Center Ljubljana. All tests were supervised by trained personnel. Participants were instructed to refrain from vigorous physical activity for at least 24 hours before testing, to arrive well-rested and adequately hydrated, and to avoid heavy meals and caffeine for at least 4 hours before the session. All evaluations were scheduled in the afternoon to minimize circadian variability and were performed in a climate-controlled environment to ensure consistent temperature and reduce environmental variability. The exercise protocol consisted of several sequential phases. The participants first completed a 3 minute rest period while seated and breathing normally. This was followed by the cycling phase, which included progressively increasing workloads: The initial workload was set at 20% of the maximum oxygen uptake (VO_2max) and increased by 20% every 3 minutes until it reached 80% of predicted VO_2max. The predicted VO_2max values were derived from age and sex-specific reference tables from the updated FRIEND registry (2022),[15] and the corresponding cycling power outputs were calculated using the Storer equation, which incorporates body mass to determine individualized maximum workload.[16] Participants were instructed to maintain a cadence of 65 rpm throughout the cycling phase, regardless of workload. HR was continuously measured throughout the entire session using 12-lead ECG monitoring, while systolic BP (SBP) and diastolic BP (DBP) were recorded automatically with a preprogrammed upper arm cuff pre-programmed in the device software. During the cycling phase, BP was measured once during each 3-minute exercise stage. After completion of the cycling phase, participants underwent a 5-minute seated recovery period, during which continuous HR monitoring was maintained and BP measured at 1, 3, and 5 minutes to assess early post-exercise hemodynamic responses. This protocol was selected to

provide a controlled and progressive physiological stimulus in standardized submaximal intensities.

Heart rate variability and heart rate recovery assessment

Electrocardiographic signals were recorded and stored for subsequent HRV analysis using Kubios HRV Analysis Software (Kubios Oy, Kuopio, Finland). The signals were sampled at 500 Hz, providing the high temporal resolution required for accurate extraction of HRV parameters. Artifacts and ectopic beats were identified and corrected using the Kubios automatic artifact correction algorithm, with manual verification to ensure signal quality. The HRV indices were calculated according to the guidelines of the Task Force of the European Society of Cardiology Task Force and the North American Society of Pacing and Electrophysiology (1996).17 The HRV indices were averaged throughout the 5-minute recovery period. This approach was chosen to provide a stable summary estimate of post-exercise autonomic recovery and to reduce the influence of short-term fluctuations and signal noise. However, it should be acknowledged that averaging across the entire recovery period can obscure transient, minute-to-minute changes in autonomic dynamics.

The time domain analysis included the mean RR interval (RR), the root mean square of successive differences (RMSSD), reflecting short-term and vagally mediated HRV, and the standard deviation of all normal to normal intervals (SDNN), representing overall autonomic modulation.[18] The frequency domain analysis comprised low-frequency (LF; 0.04–0.15 Hz) and high-frequency (HF; 0.5–0.40 Hz) power, expressed in both absolute (ms^2) and normalized units (nu). High-frequency normalized units (HFnu) predominantly indicate parasympathetic activity, low-frequency normalized units (LFnu) reflect sympathetic modulation, and their ratio (LF/HF) provides a measure of sympathovagal balance.[18,19] HRR was assessed as the decline in HR from peak exercise (HRpeak) at 1 (HRR1), 3 (HRR3), and 5 minutes (HRR5) after exercise. HRR was calculated as the absolute decrease in heart rate from peak exercise according to the following definitions: HRR1=HRpeak HR at 1 minute of recovery, HRR3=HRpeak HR at 3 minutes of recovery, and HRR5=HRpeak HR at 5 minutes of recovery. Early HRR (HRR1) predominantly reflects rapid parasympathetic reactivation, while later recovery phases (HRR3, HRR5) are influenced by both parasympathetic reactivation and sympathetic withdrawal, providing additional information on autonomic recovery after exertion.[20]

Statistical analysis

Data were analyzed using JASP version 0.17 (JASP Team, Amsterdam, The Netherlands). Continuous variables were tested for normality using the Shapiro–Wilk test and are presented as mean±standard deviation. Associations between baseline central hemodynamic parameters, cf-PWV, AIx @ 75 and post-exercise autonomic recovery indices were evaluated using Pearson or Spearman correlation coefficients, depending on the data distribution, and visualized with scatterplots. For HRV analysis, time-domain and frequency-domain indices were averaged over the entire 5-minute recovery period to obtain a single summary value per participant. Univariate linear regression analyzes were performed to identify potential predictors of autonomic recovery, followed by multivariate linear regression models adjusted for age, sex, and BMI. Statistical significance was established at $p<0.05$.

Although sex was included as a covariate in multivariate models, no stratified analyses were performed. Therefore, potential sex-specific physiological differences in autonomic and vascular responses may not be fully captured. Given the number of correlations tested, the analyzes should be interpreted with caution because of an increased risk of type I error. No formal correction for multiple comparisons was applied, as the study was exploratory. Analyses examining associations between cf-PWV and autonomic recovery indices were predefined, while additional exploratory analyses were performed to generate hypotheses for future studies.

Results

A total of 30 participants were included in the final analysis, of which 17 (56.7%) were women. The baseline characteristics of the study population, including demographic and anthropometric measures, central BP, PWA, and cf-PWV are summarized in Table 1. All variables were approximately normally distributed and no extreme outliers were identified.

Table 1. Demographic, anthropometric and central hemodynamic characteristics of study participants, values are presented as mean±SD unless otherwise indicated (n=30)

Variable (Unit)	Mean±SD or n (%)
Demographics	
Sex (female/male, n)	17 (56.7%)/13 (43.3%)
Age (years)	22.7±2.5
Body mass (kg)	69.2±9.6
Height (cm)	175.8±8.8
Body mass index (kg/m^2)	22.3±1.8
Central hemodynamic parameters	
Central systolic blood pressure (mmHg)	108.9±8.6
Central diastolic blood pressure (mmHg)	77.3±7.6
Central pulse pressure (mmHg)	31.6±5.2
Pulse wave analysis	
Augmentation pressure (AP, mmHg)	1.57±2.86
Augmentation index (AIx, %)	4.23±9.22
Augmentation index, HR-corrected (AIx@75, %)	2.30±11.45
Pulse wave velocity	
Carotid–femoral pulse wave velocity (cf-PWV, m/s)	5.57±0.75

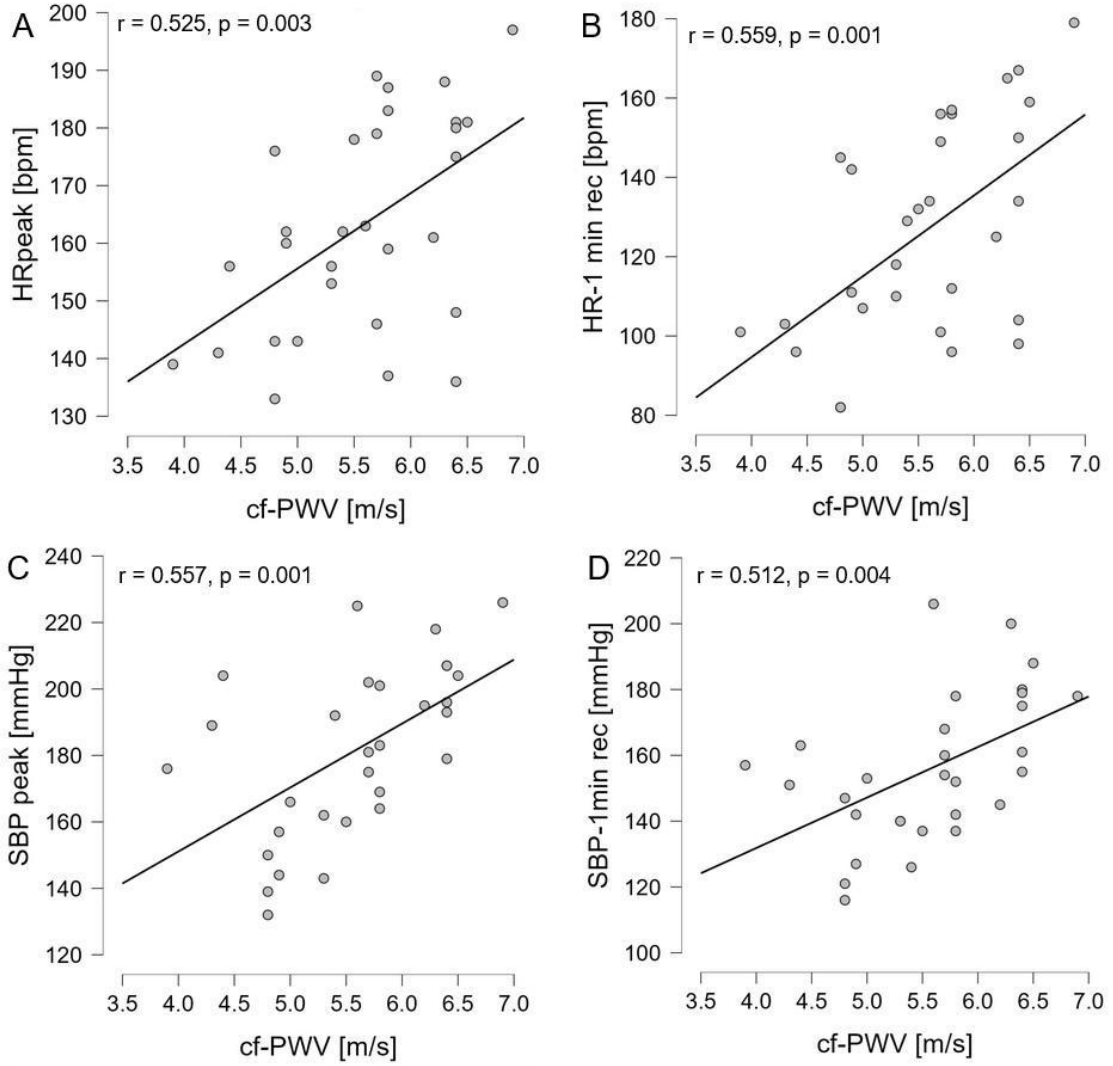

Fig. 1. The figure presents four scatterplots depicting correlations between cf-PWV and cardiovascular responses to graded submaximal cycling exercise: A: HR achieved during exercise, B: HR measured at 1-minute recovery following exercise, C: peak SBP achieved during exercise, and D: SBP measured at 1 minute recovery following exercise, all correlations are presented with the correlation coefficient (r) and the corresponding p-value. Statistical significance was established a priori at $p<0.05$

Table 2. Hemodynamic responses at rest, during peak exercise, and throughout recovery (1, 3, and 5 minutes), values are presented as mean±standard deviation

Variable (Unit)	Rest (mean±SD)	Peak exercise (mean±SD)	Recovery 1 min. (mean±SD)	Recovery 3 min (mean±SD)	Recovery 5 min (mean±SD)
Heart rate (bpm)	81.0±14.4	163.1±18.6	126.7±27.3	108.0±23.8	101.4±19.8
Systolic BP (mmHg)	117.6±12.9	181.3±25.8	155.9±22.4	136.4±20.3	127.6±16.8
Diastolic BP (mmHg)	73.5±7.9	82.6±11.5	74.3±8.8	72.7±8.3	73.7±10.6

All participants successfully completed the graded submaximal exercise protocol up to 80% of the predicted VO_2max without adverse events. The mean HR peak increased from a resting value of 81.0±14.4 bpm to 163.1±18.6 bpm during exercise. SBP increased progressively with workload, reaching a mean peak value of 181.3±25.9 mmHg (resting value: 117.6±12.9 mmHg), while DBP remained relatively stable throughout the test. During the 5-minute seated recovery period, both HR and BP gradually decreased toward baseline values (Table 2).

Associations of baseline arterial stiffness with peak exercise and post-exercise hemodynamic responses

cf-PWV was positively associated with HRpeak ($r=0.525$, $p=0.003$) and peak systolic blood pressure (SBPpeak; $r=0.557$, $p<0.001$) during graded submaximal exercise (Fig. 1). Baseline AIx@75 showed weaker but statistically significant positive associations with HRpeak ($r=0.368$, $p=0.045$) and HR measured at 1, 3, and 5 minutes during recovery (HR1: $r=0.378$, $p=0.039$; HR3: $r=0.504$, $p=0.005$; HR5: $r=0.416$, $p=0.022$). During recovery, cf-PWV was positively correlated with SBP for 1 minute (SBP1, $r=0.512$, $p=0.004$) and 3 minutes (SBP3, $r=0.486$, $p=0.008$), while the correlation for 5 minutes

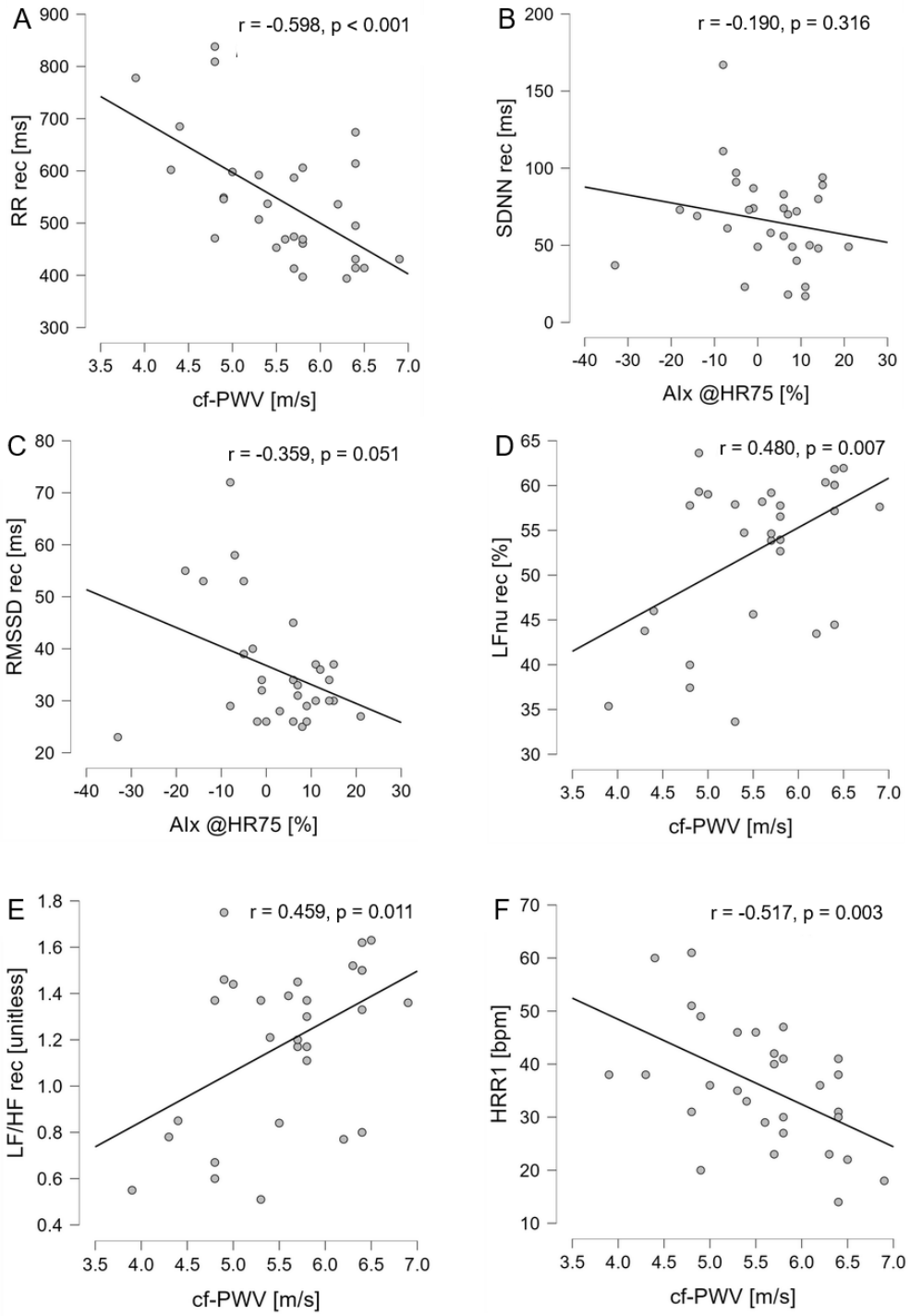

Fig. 2. The figure presents six scatterplots that illustrate correlations between arterial stiffness, heart rate variability and recovery parameters during postexercise recovery: A: cf-PWV and RR intervals, B: AIx@75 and SDNN, C: AIx@75 and RMSSD, D: cf-PWV and LFnu, E: cf-PWV and LF/HF ratio, and F: cf-PWV and early heart rate recovery during the first minute of recovery (HRR1), all correlations are presented with the correlation coefficient (r) and the corresponding p-value, statistical significance was established a priori at $p<0.05$

(SBP5) did not reach statistical significance (r=0.307, p=0.099). HR during recovery at 1, 3, and 5 minutes (HR1, HR3, HR5) showed significant positive correlations with baseline cf-PWV (HR1: r=0.559, p=0.001; HR3: r=0.603, p=0.002; HR5: r=0.541, p=0.003). No significant correlations were observed between cf-PWV and DBP at any recovery time point (all p>0.05).

Associations of baseline arterial stiffness with autonomic recovery following exercise

During the recovery period, cf-PWV showed a significant inverse association with the mean RR interval (r=−0.598, p<0.001) (Fig. 2). On the contrary, cf-PWV was not significantly associated with HRV indices, including SDNN and RMSSD (all p>0.05). However,

a higher cf-PWV was significantly associated with a lower HFnu (r=0.480, p=0.004), a higher LFnu (r=0.480, p=0.004) and a higher LF/HF ratio (r=0.49, p=0.005).

AIx@75 also demonstrated an inverse association with the mean RR interval during recovery (r=0.481, p=0.007). Furthermore, AIx @ 75 showed a trend towards an inverse association with RMSSD during recovery that did not reach statistical significance (r=<0.359, p=0.051) and was not associated with SDNN (p>0.05). Similarly to cf-PWV, AIx@75 was significantly associated with HRV indices during recovery, including a higher LFnu (r=0.376, p=0.041), lower HFnu (r=0.376, p=0.041) and a higher LF/HF ratio (r=0.367, p=0.046). No significant correlations were observed between other baseline central hemodynamic parameters and recovery HRV indices (all p>0.05).

cf-PWV was inversely associated with early HRR during the first minute of recovery (HRR1; r=<0.517, p=0.002) (Fig. 2). The associations with later recovery phases at 3 and five minutes (HRR3 and HRR5) were weaker and did not reach statistical significance (HRR3: r=−0.104, p=0.292; HRR5: r=0.146, p=0.210). The baseline cSBP was inversely correlated with HRR1 (r=<0.370, p=0.044) but not with HRR3 or HRR5 (HRR3: r=−0.211, p=0.264; HRR5: r=<0.075, p=0.692). In contrast, the augmentation index (AIx@75) was not significantly associated with HRR at any post-exercise time point (all p>0.05).

Table 3. Univariate and multivariate linear regression analyses identifying predictors of early heart rate recovery*

Model	Predictor (Unit)	B	SE	β (standardized)	p	Model statistics
Univariate	cf-PWV (m/s)	-0.033	0.010	−0.517	0.003	R=0.517; R^2=0.268; Adjusted R^2=0.241
Multivariable	cf-PWV (m/s)	-0.030	0.012	−0.465	0.045	R=0.560; R^2=0.313; Adjusted R^2=0.203
	Age (years)	−0.019	0.051	−0.063	0.715	
	Sex (female/male)	−0.324	0.292	†	0.277	
	BMI (kg/m^2)	−0.054	0.075	−0.132	0.478	

* values are regression coefficients from linear regression models, standardized β for sex is not reported because sex is a binary variable

Regression analysis of arterial stiffness as a determinant of early autonomic recovery

In the univariate linear regression analysis, cf-PWV was a significant predictor of early HRR 1 minute post-exercise, accounting for 26.8% of the variance in HRR1 (β=−0.517, R^2=0.268, p=0.003). After adjustment for age, sex and body mass index, cf-PWV remained independently associated with HRR1, although the strength of the association was attenuated (adjusted R^2=0.203, p=0.045). None of the covariates were significant independent predictors in the multivariable model (all p>0.05) (Table 3).

Exploratory regression analyzes for selected HRV indices showed that, after adjustment for age, sex, and body mass index, no baseline arterial stiffness or central hemodynamic parameters were independently associated with post-exercise HRV measures (all adjusted p>0.05).

Discussion

This exploratory pilot study investigated the association between baseline aortic stiffness and cardiovascular and autonomic recovery following graded submaximal aerobic exercise in healthy young adults. The main findings were that a higher baseline aortic stiffness (assessed by cf-PWV) was associated with higher peak HR and SBP during exercise, slower HRR at the beginning of the exercise, and an altered autonomic recovery profile characterized by patterns in the HRV indices (lower HFnu, higher LFnu and LF/HF ratio). cf-PWV remained an independent predictor of early HRR after adjustment for age, sex, and BMI. Such findings suggest an association between baseline vascular properties and early cardiovascular and autonomic recovery in a controlled physiological setting. These findings are consistent with the results of the cardiovascular risk in young Finns study by Haarala et al.[21], which showed that greater arterial stiffness was associated with exaggerated exercise blood pressure responses during the maximal cardiopulmonary exercise test. Together, these findings are consistent with the idea that baseline arterial pressure is associated with cardiovascular load during exercise and recovery, even in young, otherwise healthy individuals.

Beyond peak exercise responses, our findings suggest that a higher baseline cf-PWV is associated with slower early postexercise recovery, reflected by higher HR and BP during the recovery period and delayed hemodynamic normalization. These findings indicate that higher arterial stiffness may also be associated with early cardiovascular readjustment after exercise. Previous studies have also linked a higher PWV to altered exercise responses and delayed recovery.[22,23] In our study, higher HR and SBP during graded submaximal exercise reflect cardiovascular responses to physiological stress. Previously, exaggerated exercise BP responses have been associated with vascular dysfunction and adverse cardiovascular profiles.[24]

Given that postexercise hemodynamic recovery is tightly regulated by autonomic mechanisms, we further examined whether a higher baseline cf-PWV is associated with altered autonomic regulation during recovery. A higher baseline cf-PWV was associated with a lower HFnu, a higher LFnu, and an elevated LF/HF ratio, commonly interpreted as reflecting changes in autonomic balance.[25]

These findings are consistent with Park et al.[26], who reported similar associations between arterial stiffness and altered autonomic recovery after aerobic exercise,

and with findings from resistance exercise studies, suggesting that arterial stiffness may also be relevant to autonomic recovery across different exercise modalities.[27]

Previous studies suggest a bidirectional relationship between autonomic activity and arterial stiffness, although causality cannot be inferred in the present study.[28] Recent work by Gronwald et al.[29] further highlights the influence of exercise intensity on post-exercise HRV, showing that more vigorous exercise produces greater and longer lasting autonomic perturbations compared to moderate exercise. Our findings suggest that even in healthy young adults, interindividual differences in arterial stiffness may be associated with autonomic recovery. Higher cf-PWV was associated with higher LFnu and LF/HF ratio during recovery, even under graded submaximal exercise (20–80% VO_2max). This suggests that vascular properties may be associated with interindividual differences in autonomic responses during recovery.

Our findings suggest that variations in arterial stiffness and autonomic regulation are detectable even in apparently healthy young adults. HRV and related autonomic indices provide sensitive measures of autonomic cardiovascular control during the recovery phase after exercise, reflecting the ability of the cardiovascular system to adapt to physiological stress.[25] Furthermore, cf-PWV emerged as a more consistent predictor of frequency domain HRV indices than AIx@75, possibly reflecting differences in intrinsic aortic stiffness and baroreflex-mediated autonomic modulation.[30] These results suggest the potential utility of combining vascular and autonomic evaluation to characterize physiological differences in cardiovascular regulation in a controlled setting. To support this interpretation, a systematic review by Pierce et al.[31] reported that PWV shows more consistent responses to exercise in different modalities, whereas the increase indices are more variable and strongly influenced by transient hemodynamic conditions.

Cf-PWV was associated with HRV indices but not time-domain measures, reflecting differences in the physiological information captured by these parameters.[31,32] This pattern can be explained by the fact that the time domain indices reflect overall or short-term variability without distinguishing sympathetic and parasympathetic contributions, while the frequency domain measures more directly capture autonomic modulation and baroreflex sensitivity.[32-34] The present study focused on the early post-exercise recovery phase, with HRV, HRR, and BP monitored during the first 5 minutes after exercise cessation. This time window captures rapid parasympathetic reactivation and the initial phase of cardiovascular readjustment, but it does not reflect the full trajectory of post-exercise recovery, which can extend beyond 20 to 60 minutes or longer.[35] For this reason, the present findings should be interpreted as relating specifically to early recovery kinetics rather than complete post-exercise recovery dynamics.

Beyond exercise performance, an important observation of the present study is related to early postexercise recovery. HRR within the first minute after exercise cessation is mainly governed by rapid parasympathetic reactivation and is widely used as a marker of autonomic regulation.[36,37] In our study, a higher baseline cf-PWV was associated with slower HRR at 1 minute, and this relationship remained significant after adjustment for age, sex and BMI. While previous studies linking greater arterial stiffness to slower HRR were conducted in older or clinical populations,[38,39] the present findings suggest that vascular-autonomic associations are also detectable in a controlled physiological setting in healthy young adults. In summary, baseline cf-PWV was associated with early cardiovascular and autonomic recovery after graded submaximal exercise in healthy young adults and remained an independent predictor of early HRR. These results should be interpreted as evidence of physiological variation in vascular–autonomic regulation rather than indicators of clinical risk. In general, the study provides information on early recovery dynamics under controlled experimental conditions, while confirmation in larger and clinical cohorts is still needed.

Study limitations and future perspectives

This exploratory study has several limitations that should be considered when interpreting the findings. First, the relatively small sample size (n=30) and the inclusion of a homogeneous cohort of healthy young adults within a narrow age range (18-30 years) can limit the generalizability. Although the study detected several moderate to strong associations between cf-PWV and autonomic and hemodynamic indices, the results should be interpreted with caution given the sample size and exploratory design. Future studies involving larger and more diverse populations, including older adults and individuals with cardiovascular risk factors, are warranted to confirm and extend these observations.

Second, post-exercise recovery was monitored for only 5 minutes. Although this period captures the early phase of parasympathetic reactivation and allowed simultaneous evaluation of HRR, HRV, and BP dynamics, longer recovery monitoring would provide a more complete characterization of post-exercise autonomic and hemodynamic adjustments. Third, cf-PWV and central hemodynamics were evaluated only at rest. Although resting cf-PWV provides a robust index of intrinsic aortic stiffness, future studies may benefit from evaluating vascular responses during post-exercise recovery to better characterize dynamic vascular–autonomic interactions.

Fourth, aerobic capacity was not measured directly using cardiopulmonary exercise testing but estimat-

ed from age- and sex-based prediction tables. Given the relatively homogeneous fitness levels of young participants and the derived nature of predicted VO_2max, its inclusion in regression models may have introduced collinearity with exercise HR variables. However, direct VO_2max measurement would be valuable in future studies involving more heterogeneous or clinical populations.

Finally, the inclusion of multiple covariates in regression models relative to the modest sample size may have reduced statistical power. These adjustments were included to account for potential confounders and, therefore, the findings should be interpreted as exploratory and hypothesis-generating. In addition, the observational design limits causal inference. Future larger-scale studies, particularly in populations with cardiovascular risk factors, are needed to clarify the relationship between arterial stiffness and post-exercise autonomic recovery.

Conclusion

This exploratory pilot study demonstrates that baseline aortic stiffness, assessed by cf-PWV, is associated with cardiovascular and autonomic responses to graded submaximal exercise in healthy young adults. Higher cf-PWV was associated with higher peak HR and SBP during exercise, as well as slower early HRR after cessation of exercise. Cf-PWV also remained independently associated with early HRR after adjustment for age, sex, and BMI. These findings support an association between baseline vascular properties and early cardiovascular and autonomic recovery dynamics in a controlled physiological setting. However, given the observational and pilot nature of the study, these results should be interpreted as preliminary. More studies in larger and clinical populations to confirm these associations and better understand their physiological and clinical relevance.

Declarations

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Author contributions

Conceptualization: A.K. and T.M.; Methodology: A.K.; Software: M.R.; Validation: M.R. and T.M.; Investigation: A.K. and T.M.; Resources: A.K.; Data Curation: T.M.; Writing – Original Draft Preparation: T.M.; Writing – Review & Editing: A.K. and T.M.; Visualization: M.R. and T.M.; Supervision: A.K.; Project Administration: A.K. and T.M.

Conflict of interest

The authors declare no conflict of interest.

Data availability

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethics approval

Ethical approval was granted by the Medical Ethics Committee of the Republic of Slovenia (approval number: 0120-388/2025-2711-5).

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