European Journal of Applied Physiology
https://doi.org/10.1007/s00421-026-06419-y

ORIGINAL ARTICLE

Integrated hemodynamic and autonomic responses following aerobic versus resistance exercise in young Black men

Ann Claire E. Blalock[1] · Alex T. Pierce[1] · Carlos D. Miller[1] · Sydney L. Bricker[1] · Gregory J. Grosicki[2] · Andrew A. Flatt[1]

Received: 25 June 2026 / Accepted: 24 August 2026

Abstract

Purpose Acute cardiovascular recovery from exercise remains poorly characterized in young Black men, a population at elevated cardiovascular risk. Therefore, we compared integrated hemodynamic and cardiac autonomic responses following aerobic (AE) versus resistance exercise (RE) in this group.

Methods Fifteen Black men (18–29 years) completed counterbalanced bouts of AE (30 min cycling, 65% of peak heart rate) and RE (six barbell exercises, 3 × 10 repetitions, 95% of 10-repetition maximum). Supine pulse wave analysis assessed augmentation index normalized to 75 bpm (AIx75), wave-reflection components, subendocardial viability ratio (SEVR), and central and brachial blood pressure before and at 5, 15, and 30 min post-exercise. HR and the natural log of the root mean square of successive differences (LnRMSSD) were derived from electrocardiography.

Results Exercise duration and mean HR were similar between modes (Ps > 0.05), whereas peak HR was higher during RE ($P < 0.01$). RE produced larger and more sustained elevations in AIx75 and wave-reflection indices, greater reductions in SEVR and central and brachial diastolic blood pressure, and higher HR with lower LnRMSSD throughout recovery compared with AE (mode × time interactions, all $P < 0.001$). Mode differences persisted independent of peak HR.

Conclusion In young Black men, RE induces a more pronounced and sustained cardiovascular and autonomic strain during the early post-exercise period than AE, characterized by elevated central afterload, reduced myocardial oxygen supply–demand balance, and delayed parasympathetic reactivation. These findings offer novel mechanistic insight into modality-specific cardiovascular responses in a high-risk population and support consideration of recovery kinetics in exercise programming.

Keywords Pulse wave analysis · Heart rate variability · Blood pressure · Sports medicine · Exercise physiology

Abbreviations

AE	Aerobic exercise
AIx75	Augmentation index normalized to 75 bpm
ANCOVA	Analysis of covariance
AP	Augmentation pressure
DBP	Diastolic blood pressure
ECG	Electrocardiography
HR	Heart rate
HRpeak	Peak heart rate
HRV	Heart rate variability
LnRMSSD	Natural logarithm of the root mean square of successive differences
Pb	Backward wave amplitude
Pf	Forward wave amplitude
RE	Resistance exercise
RMSSD	Root mean square of successive differences
SBP	Systolic blood pressure
SEVR	Subendocardial viability ratio
10RM	10-Repetition maximum
Wpeak	Peak power output

Communicated by Lauro C. Vianna.

✉ Andrew A. Flatt
aflatt@georgiasouthern.edu

[1] Department of Health Sciences and Kinesiology, Biodynamics and Human Performance Center, Georgia Southern University-Armstrong Campus, 11935 Abercorn St., Savannah, GA 31419, USA

[2] Performance Science., WHOOP Inc, Boston, MA, USA

Published online: 02 September 2026

Introduction

Cardiovascular disease remains a leading cause of morbidity and mortality that disproportionately affects Black men in the United States (Kyalwazi et al. 2022), yet young Black men remain underrepresented in studies of post-exercise cardiovascular recovery. Acute post-exercise responses are clinically relevant because the early minutes after exertion involve transient shifts in vascular load and autonomic control and coincide with a brief window of elevated risk for acute cardiac events in susceptible individuals (Franklin et al. 2020). Additionally, acute cardiovascular responses have been shown to predict chronic exercise-induced changes (Wegmann et al. 2018). A limited number of race comparison studies following acute aerobic exercise (AE) indicate altered vascular and blood pressure responses in Black versus White men (Heffernan et al. 2007b; Yan et al. 2014), highlighting the need to further investigate acute cardiovascular recovery in this population. Moreover, despite being recommended alongside AE (Ozemek et al. 2025), acute responses to resistance exercise (RE) in young Black men remain largely unexamined, creating a critical gap in our understanding of mode-specific responses.

Comprehensive evaluation of post-exercise cardiovascular dynamics involves concurrent assessment of hemodynamic parameters and cardiac autonomic modulation. Measures of central blood pressure and wave reflection characterize ventricular–vascular coupling and systolic loading (Sharman et al. 2009; Wilkinson et al. 2000), while the subendocardial viability ratio (SEVR) reflects the balance between myocardial oxygen supply and demand (Tagawa et al. 2019). Meanwhile, heart rate (HR) and HR variability (HRV) can be used to reflect parasympathetic reactivation during recovery (Michael et al. 2017). However, these responses do not occur in isolation. Alterations in wave reflection and central loading may influence myocardial oxygen supply–demand balance, while autonomic recovery modulates both vascular tone and cardiac function. Although acute AE typically lowers central stiffness and yields modest, transient changes in wave reflection, RE elevates central pulsatile load (e.g., higher absolute and normalized augmentation index [AIx]), depresses SEVR (Pierce et al. 2018a), and delays cardiac parasympathetic reactivation (Marasingha-Arachchige et al. 2022; Michael et al. 2017). Nevertheless, few studies integrate these domains within a single protocol. Consequently, the interaction between late-systolic pressure augmentation, myocardial oxygen supply–demand balance, and parasympathetic reactivation during early recovery remains poorly defined, especially in young Black men.

Although composite markers like AIx normalized to a HR of 75 bpm (AIx75) are widely used to index late-systolic ventricular-vascular load during post-exercise recovery (Pierce et al. 2018a), detailed pulse wave analysis allows for the separation of the central waveform into its forward (Pf) and backward (Pb) wave components and the calculation of reflection magnitude (Pb/Pf) (Wilkinson et al. 2000; Wakeham et al. 2025). These individual waveform metrics offer superior mechanistic insight into whether post-exercise AIx75 increases are driven by larger reflected waves, amplified forward waves, or both, while also clarifying how those changes affect central systolic loading, late-systolic pressure augmentation, and myocardial oxygen supply–demand balance during recovery. Integrating waveform characteristics with HRV may provide mechanistic insight into how alterations in central hemodynamics, ventricular–vascular coupling, and autonomic regulation collectively shape cardiovascular recovery. This may be particularly relevant following RE, which imposes substantial transient increases in central hemodynamic loading but remains less well characterized in terms of its interaction with myocardial oxygen supply–demand balance and autonomic restoration.

Whether the differential cardiovascular responses to AE and RE reported in prior studies (Heffernan et al. 2007a; Pierce et al. 2018b) similarly occur in young Black men remains unknown. Given prior evidence of distinct vascular and blood pressure responses to acute exercise in Black adults (Heffernan et al. 2007b; Yan et al. 2014) and the lack of acute RE research in this population, this study was designed to extend previous mode-comparison work into an understudied group for whom exercise recovery may differ. Clarifying these integrated responses is important for improving our understanding of early post-exercise cardiovascular stress and its potential implications for exercise prescription. Therefore, we compared the acute effects of AE and RE on central hemodynamics, wave reflection characteristics, SEVR, and cardiac autonomic modulation in young Black men. We hypothesized that RE would elicit greater elevations in wave reflection and central loading indices, larger reductions in SEVR, and delayed cardiac parasympathetic recovery compared with AE.

Materials and methods

Study design

A counterbalanced, crossover study design was used to compare acute cardiovascular responses to isolated bouts of AE and RE in young Black men. Participation involved three laboratory visits. Visit 1 included baseline assessments and familiarization. Visits 2 and 3 consisted of the counterbalanced exercise bouts (AE and RE) separated by ≥72 h. Cardiovascular assessments were performed pre-exercise

and at 5, 15, and 30 min post-exercise, each preceded by 5 min of stabilization. HR was monitored continuously during exercise using a chest-strap telemetry system. Participants were instructed to abstain from food and beverages for ≥2 h, alcohol, caffeine, and dietary exercise supplements for ≥24 h, and intense exercise for ≥72 h prior to each visit. A standardized 250 ml cup of water was provided before exercise after pre-exercise cardiovascular assessment. No other fluids were consumed to control for their confounding influence on cardiovascular parameters (Christiani et al. 2021). All testing occurred in a temperature-controlled laboratory (22° C, 50% relative humidity) between 0800 and 1200 h to control for circadian influences. Compliance with pre-visit restrictions was verified via self-report upon arrival.

Participants

Apparently healthy non-Hispanic Black men (age: 18–29 years) were recruited for this study via convenience sampling from the university community and surrounding areas. To qualify for participation, volunteers needed to be proficient in performing standard barbell exercises and be free from known cardiovascular, metabolic, pulmonary, or musculoskeletal diseases, as well as contraindications to exercise as determined by a health history and physical activity readiness questionnaire. Exclusion criteria included being a current smoker, use of antihypertensive or vasoactive medications, acute illness, and allergies to adhesives. The study was approved by the Georgia Southern University Institutional Review Board (Protocol H25054), and all procedures conformed to the ethical standards outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to any testing.

Although multiple cardiovascular outcomes were assessed, the study was powered based on AIx75 as the primary index of central pulsatile loading. Sample size was determined a priori using G*Power (3.1.9.7, Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany) for a mode × time interaction in a 2 × 4 repeated-measures analysis of variance (ANOVA). We assumed Cohen's f = 0.30, informed by aggregated acute exercise effects on AIx75 (Pierce et al. 2018b), α = 0.05, power = 0.80, correlation among repeated measures = 0.60, based on the reliability of pre- and post-exercise AIx/AIx75 (Perissiou et al. 2019), nonsphericity correction ε = 1, and 4 measurements. This resulted in a total sample size of 14 participants. Accordingly, 17 individuals were recruited to account for potential attrition.

Testing and familiarization

Following written informed consent and eligibility screening, participants underwent baseline assessments in the laboratory. Participants were first familiarized with the cardiovascular assessment protocol (including oscillometric and ECG procedures) to minimize novelty effects during subsequent experimental visits. Height was measured using a stadiometer (Seca 213, Seca GmbH, Hamburg, Germany). Body mass, body fat percentage, and visceral fat rating were determined using a calibrated bioelectrical impedance analysis system (InBody 570, InBody Co., Ltd., Seoul, South Korea) while participants stood barefoot on the device holding hand electrodes for ~1 min.

After a standardized warm-up consisting of 5 min of light cycling and dynamic stretches for the upper and lower limbs, 10-repetition maximum (10RM) strength was assessed for six barbell exercises (back squat, Romanian deadlift, bench press, bent-over row, standing press, and arm curl). Each exercise began with warm-up sets consisting of 10 repetitions at 50% of the self-estimated 10RM, 10 repetitions at 75% of the estimated 10RM, followed by 10 repetitions at 100% of the estimated 10RM, with 3 min of rest between sets. If necessary, loads were adjusted over 1–2 subsequent attempts until the 10RM was established. Exercises were performed using a full range of motion and strict technique.

Baseline assessments concluded with a graded maximal exercise test on a mechanically braked cycle ergometer (939E, Monark Exercise AB, Vansbro, Sweden). Participants began at 50 W, with resistance increasing by 25 W every 2 min until volitional exhaustion. Heart rate was monitored continuously via a Bluetooth HR monitor (H10, Polar Electro Oy, Kempele, Finland). Peak HR (HRpeak) and peak power output (Wpeak) were recorded as the highest achieved HR and completed workload (in watts), respectively.

Exercise protocols

The AE session consisted of 30 min of continuous cycling on the Monark ergometer at 65% of HRpeak. Cadence was maintained at 60–70 rpm throughout. Heart rate was monitored continuously to ensure the target intensity was achieved and adjusted as needed. This intensity and duration were selected in accordance with American College of Sports Medicine (ACSM) guidelines for near-daily moderate-intensity aerobic exercise (Ozemek et al. 2025).

The RE session involved a total-body protocol consisting of 3 sets of 10 repetitions at 95% of the 10RM (determined on Visit 1) for all six barbell exercises with 90 s of rest between sets and 2 min between exercises. Participants performed 1 warm-up set of 10 repetitions at ~50% of 10RM

for each exercise prior to working sets. This protocol is consistent with ACSM resistance training guidelines for healthy adults (Ozemek et al. 2025). Appropriate technique was enforced by members of the research team for all exercises and loads were adjusted if necessary to ensure completion of 10 repetitions. Continuous HR was monitored and total exercise duration was recorded for comparison with AE.

Hemodynamics

All hemodynamic measurements were performed in the supine position in a private examination room following 5 min of quiet stabilization. Assessments occurred at baseline (pre-exercise) and at 5, 15, and 30 min post-exercise for both the AE and RE bouts. A brachial cuff was applied to the right arm for systolic (SBP) and diastolic (DBP) blood pressure assessment via automated oscillometry using the SphygmoCor XCEL device (AtCor Medical, Naperville, IL, USA). This system has been validated against invasive aortic pressure recordings for assessment of central blood pressure, augmentation index, and wave-separation parameters, and demonstrates high intra- and inter-session reliability for AIx75 and related indices in young adults at rest and during post-exercise recovery (Perissiou et al. 2019; Pierce et al. 2018b). The cuff was then re-inflated to a sub-diastolic level for peripheral waveform acquisition via volumetric displacement of the brachial artery. Pulse wave analysis was used to derive AIx and AIx75, along with central SBP and DBP, Pf, Pb, Pb/Pf, and SEVR. Central hemodynamic parameters were derived using a generalized transfer function applied to the peripheral waveform, and wave separation was performed using pressure-only methods to estimate Pf, Pb and Pb/Pf. Because AIx is strongly heart rate dependent, and recovery HR may differ between AE and RE, we prespecified AIx75 as our primary augmentation metric (Wilkinson et al. 2000). Blood pressure measurements were taken in duplicate and averaged. A third measurement was taken if the difference exceeded 5 mmHg, and the two closest values were used.

Cardiac autonomic modulation

Cardiac autonomic modulation was assessed via supine HR and HRV derived from continuous ECG recordings using a bioamplifier system (Biopac MP150, Goleta, CA, USA) sampled at 1000 Hz as previously described (Vondrasek et al. 2026). Three Ag-AgCl electrodes were placed on the participant's trunk in a modified lead II configuration to capture ECG signals. Five-min ECG segments from immediately following hemodynamic measurements at each time point were used for analysis. Data were extracted and analyzed offline using Kubios HRV Scientific software (4.1.0, Kuopio, Finland). R-R intervals were first detrended using the smoothness priors method (Tarvainen et al. 2002) and were subsequently filtered using the automatic correction algorithm (Lipponen and Tarvainen 2019). Recorded parameters included HR and the root mean square of successive differences (RMSSD). RMSSD was selected as the primary HRV metric due to its established use as an index of cardiac parasympathetic modulation during postexercise recovery (Michael et al. 2017) and its satisfactory reproducibility in healthy young adults following exercise (Araújo et al. 2020).

Statistical analyses

Linear mixed models were used to examine within-person variation in cardiovascular parameters. Fixed effects included mode (AE vs. RE), time (pre-exercise, 5 min, 15 min, and 30 min post-exercise), and the mode × time interaction; subject identification was included as a random effect. Separate models were fit for each dependent variable. Model residuals were assessed for normality using Shapiro-Wilk tests. Natural logarithm (Ln) transformations were applied to non-normally distributed variables. Tukey's Honest Significant Difference tests were used for post-hoc analyses. Exercise duration as well as mean and peak HR were compared between sessions using paired t-tests. Standardized differences were calculated using Hedges' g effect sizes for parametric pairwise comparisons (Lakens 2013). Effect sizes are reported without qualitative magnitude labels (e.g., small, medium, large), consistent with recommendations that interpretation of standardized effects should be context-specific rather than based on arbitrary thresholds (Lakens 2013). P values < 0.05 were considered statistically significant. Analyses were conducted in JMP Pro 18 (SAS Institute Inc. Cary, NC, USA) and figures were constructed in JASP 0.19.3 (University of Amsterdam, Amsterdam, The Netherlands). Data are reported as mean ± standard deviation or median (interquartile range).

Results

Participant characteristics

Fifteen young Black men completed the study protocol and were included in the final analysis (Table 1). Although no participants had a clinical diagnosis of hypertension, baseline brachial SBP fell within the stage 1 hypertension range (≥ 130 mmHg) (Whelton et al. 2018). When compared with recently established 10RM norms in young men (Piper et al. 2021), our participants strength levels corresponded to the 60th percentile, consistent with recreational resistance

Table 1 Participant characteristics ($n = 15$ young Black men)

Descriptor	Mean ± SD or median (IQR)
Anthropometrics characteristics	
Age, years	23.7 ± 3.2
Height, cm	179.1 ± 6.1
Mass, kg	86.8 ± 18.5
Body fat, %	17.9 ± 7.6
Visceral fat rating	7 (8)
Cardiovascular characteristics	
Brachial systolic blood pressure, mmHg	131.5 ± 12.7
Brachial diastolic blood pressure, mmHg	72.4 ± 7.6
Resting heart rate, bpm	56.7 ± 5.4
Fitness characteristics	
Peak heart rate, bpm	176.5 ± 12.0
Peak power output, W	170 ± 15
Strength characteristics	
Back squat 10RM, kg	92.5 ± 27.4
Romanian deadlift 10RM, kg	91.0 ± 28.1
Bench press 10RM, kg	73.9 ± 22.7
Bent-over row 10RM, kg	53.2 ± 17.6
Overhead press, 10RM, kg	35.1 ± 10.3
Arm curl 10RM, kg	29.8 ± 7.7

SD standard deviation, *IQR* interquartile range; *RM* repetition maximum

training experience. Contrastingly, peak W during cycle ergometry (~170 W) is consistent with lower cardiorespiratory fitness relative to norms for healthy young men (Kaminsky et al. 2017), indicating a non-endurance-trained sample.

Exercise session characteristics

Total exercise duration was similar between AE and RE (30.0 ± 0.0 vs. 31.0 ± 5.0 min; $P = 0.266$, g = 0.28). Mean HR during exercise also did not differ significantly, (129.9 ± 9.0 vs. 122.1 ± 14.6 bpm; $P = 0.06$, g = 0.63), whereas peak HR was significantly lower during AE (148.4 ± 12.8 vs. 162.8 ± 12.5 bpm; $P < 0.01$, g = − 1.11).

Wave reflection responses

Significant mode × time interactions were observed for all wave reflection indices (Figs. 1 and 3), including AIx75 ($F_{3,42} = 25.4$, $P < 0.001$), AP ($F_{3,42} = 13.8$, $P < 0.001$), Pb ($F_{3,42} = 7.1$, $P < 0.001$), Pf ($F_{3,42} = 8.2$, $P < 0.001$), Pb/Pf ($F_{3,42} = 11.6$, $P < 0.001$), and SEVR ($F_{3,42} = 24.3$, $P < 0.001$), accompanied by significant main effects of mode and time (Ps < 0.01).

Post-hoc analyses revealed that AIx75 transiently increased at 5 min post-AE relative to pre-AE ($P < 0.001$; g = 0.96) but returned toward baseline at 15 and 30 min post-AE (Ps > 0.05). In contrast, AIx75 was elevated at all post-RE time points compared with pre-RE (Ps < 0.001; g = 1.00–3.83). Additionally, post-5 min RE was higher than post-15 and post-30 RE (Ps < 0.001), and post-15 was higher than post-30 RE ($P < 0.0001$). Finally, all post-RE AIx75 values were higher than corresponding post-AE values throughout recovery (Ps < 0.001; g = 1.37–2.87).

AP was unchanged from pre-AE at all post-AE time points (Ps > 0.05). Conversely, AP increased at 5 min post-RE relative to all time points and, although decreased at 15 min post-RE, remained elevated relative to pre-RE (Ps < 0.01, g = 0.84–1.82), returning to baseline by 30 min post-RE ($P > 0.05$). Between-mode comparisons showed higher AP at all post-RE time points relative to corresponding post-AE time points (Ps < 0.05, g = 0.91–1.72).

Pb exhibited a similar pattern of significant differences as AP, except values at 5 and 15 min post-RE were not different ($P = 0.323$), nor were RE and AE values different at 30 min post-exercise ($P = 0.054$). Pf remained unchanged post-AE (Ps > 0.05) but increased at all post-RE time points relative to pre-RE (Ps < 0.05, g = 0.94–1.16) and was higher at 15 and 30 min post-RE relative to corresponded post-AE values (Ps < 0.05, g = 0.91–1.06). Pb/Pf, increased at 5 min post-RE and was greater than all other time points (Ps < 0.05, g = 1.16–1.54), with higher values at 5 min post-RE than 5 min post-AE ($P < 0.0001$, g = 1.97).

SEVR decreased at all post-exercise time points in both modes relative to pre-exercise (Ps < 0.001, g = − 1.01 to − 4.72), with values at 5 and 15 min post-exercise being significantly lower than at 30 min for RE (Ps < 0.01, g = − 0.94 to − 1.09), and values at 5 min post-exercise being lower than at 30 min for AE (Ps < 0.01, g = − 1.34). Between-mode comparisons showed that all post-RE values were significantly lower than corresponding post-AE values (Ps < 0.001, g = − 1.83 to − 2.58).

Blood pressure responses

Mode × time plots for blood pressure are presented in Fig. 2. A significant main effect of time was observed for central SBP ($F_{3,42} = 6.9$, $P < 0.001$). Irrespective of mode, central SBP decreased during recovery, with values at 15 min post-exercise lower than at 5 min post-exercise ($P < 0.05$, g = − 0.35) and further reduced at 30 min post-exercise relative to pre- and 5 min post-exercise (Ps < 0.05, g = − 0.34 to − 0.55). Contrastingly, a significant mode × time interaction was observed for brachial SBP ($F_{3,42} = 3.1$, $P < 0.05$), along with a significant main effect of time ($P < 0.05$). Post-hoc comparisons showed no changes across time post-RE (Ps > 0.05), whereas brachial SBP decreased at 15 and 30 min post-AE relative to 5 min post-AE (Ps < 0.05, g = − 0.66 to − 0.69).

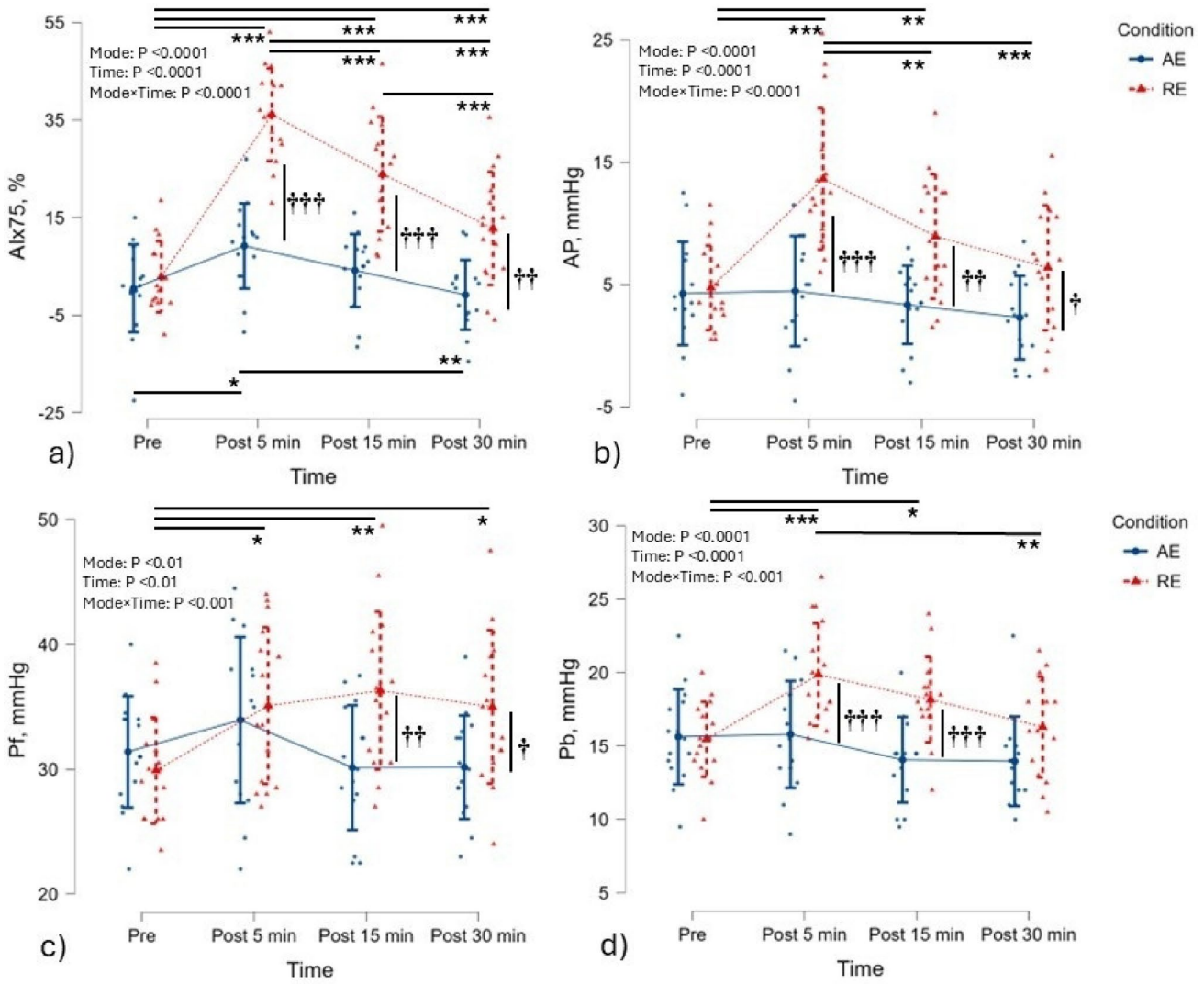

Fig. 1 Mode × time plots showing changes in **a** augmentation index normalized to 75 bpm (AIx75), **b** augmentation pressure (AP), **c** forward wave amplitude (Pf), and **d** backward wave amplitude (Pb) measured pre-exercise and at 5, 15, and 30 min post-exercise following aerobic (AE) and resistance exercise (RE). Data are presented as individual responses with group means ± standard deviation. Horizontal black lines indicate significant within-mode post-hoc comparisons, with significance denoted by $*P<0.05$, $**P<0.01$, and $***P<0.001$. Vertical black lines indicate significant between-mode differences at corresponding time points, with significance denoted by $\dagger P<0.05$, $\dagger\dagger P<0.01$, and $\dagger\dagger\dagger P<0.001$

A significant mode × time interaction was observed for central DBP ($F_{3,42}$ = 7.3, $P<0.001$), along with significant main effects of mode and time (Ps < 0.05). Post-hoc comparisons showed no changes across time post-AE (Ps > 0.05). Conversely, central DBP was reduced at all post-RE time points relative to pre-RE (Ps < 0.01, g = −0.61 to −0.87) and was lower than corresponding post-AE values throughout recovery (Ps < 0.01, g = −0.88 to −0.98). Brachial DBP exhibited an identical pattern of significant effects and post-hoc differences.

Cardiac autonomic responses

Mode × time plots for HR and HRV are presented in Fig. 3. A significant mode × time interaction was observed for HR ($F_{3,42}$ = 235.5, $P<0.0001$), along with significant main effects of mode and time (Ps < 0.001). Post-hoc comparisons showed that HR increased at all post-RE time points relative to pre-RE (Ps < 0.0001, g = 2.60–4.10), and that HR at 5 min post-RE was greater than 15 and 30 min post-RE (Ps < 0.001, g = 0.57–1.50), and HR at 15 min post-RE was greater than 30 min post-RE ($P<0.0001$, g = 0.88). Although similar temporal patterns occurred post-AE (Ps < 0.01, g = 0.66–2.31),

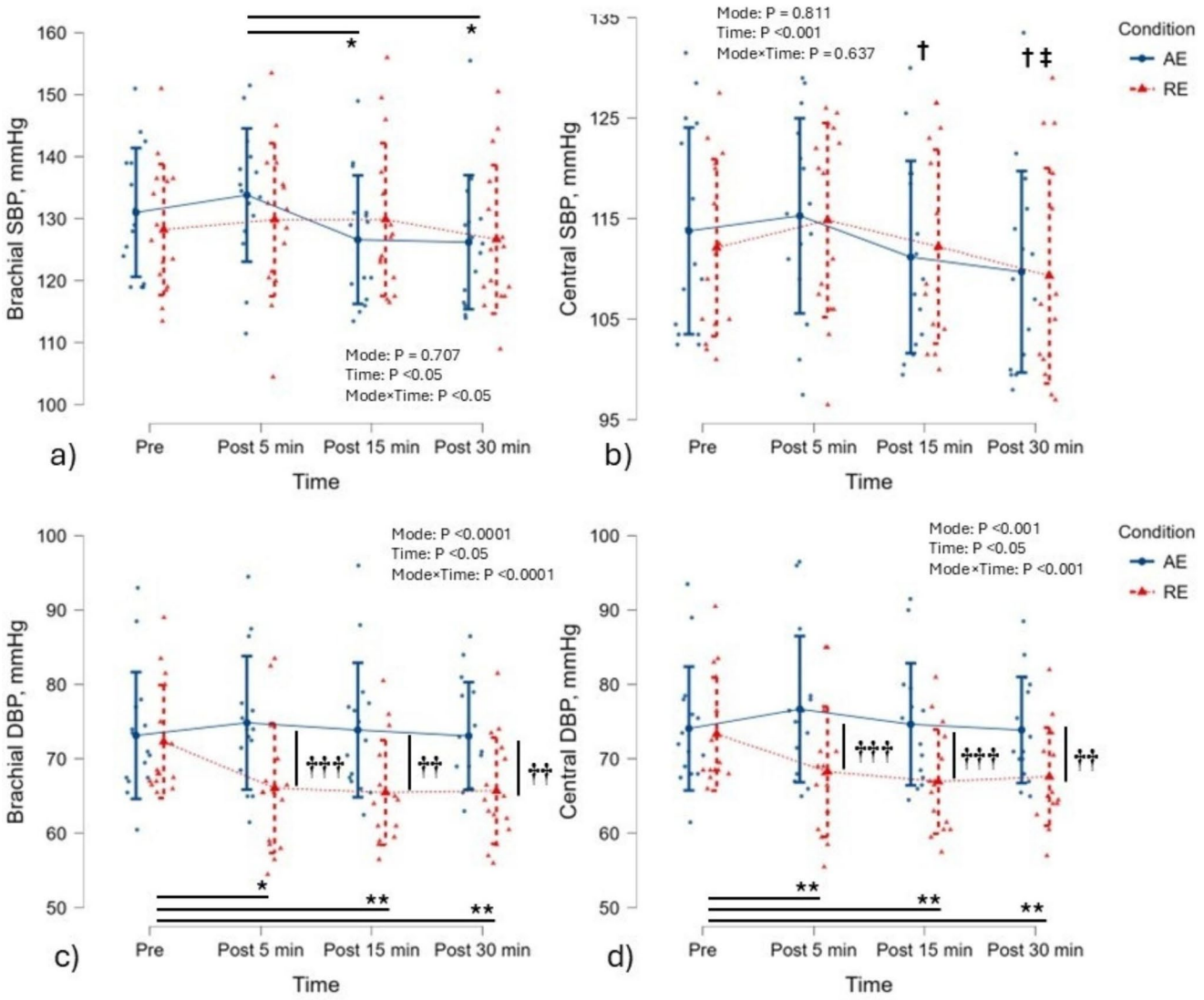

Fig. 2 Mode × time plots showing changes in **a** brachial systolic blood pressure (SBP), **b** central systolic blood pressure (SBP), **c** brachial diastolic blood pressure (DBP), and **d** central diastolic blood pressure (DBP) measured pre-exercise and at 5, 15, and 30 min post-exercise following aerobic (AE) and resistance exercise (RE). Data are presented as individual responses with group means ± standard deviation. Horizontal black lines indicate significant within-mode post-hoc comparisons, with significance denoted by *$P<0.05$, **$P<0.01$, and ***$P<0.001$. Vertical black lines indicate significant between-mode differences at corresponding time points, with significance denoted by †$P<0.05$, ††$P<0.01$, and †††$P<0.001$. For central SBP (panel b), cross symbols reflect post-hoc comparisons from the significant main effect of time, irrespective of exercise mode; ‡ indicates significantly different from pre-exercise and † indicates significantly different from 5 min post-exercise ($P<0.05$)

HR was greater at all post-RE time points relative to corresponding post-AE values (Ps < 0.001, g = 1.60–1.66).

A significant mode × time interaction was observed for LnRMSSD ($F_{3,42}$ = 98.9, $P<0.0001$), along with significant main effects of mode and time (Ps < 0.001). Post-hoc comparisons showed the LnRMSSD inversely mirrored significant HR differences post-RE (Ps < 0.05, g = −0.61 to −3.41). Contrastingly, LnRMSSD decreased at only 5 and 15 min post-AE relative to pre-AE (Ps < 0.001, g = −1.03 to −1.75), with LnRMSSD at 5 min post-AE being lower than at 15 and 30 min post-AE (Ps < 0.05, g = −0.70 to −1.28). Additionally, LnRMSSD was lower at all post-RE time points than corresponding post-AE values (Ps < 0.01, g = −1.51 to −1.72).

Sensitivity analysis

Since HRpeak was significantly higher during RE and because SEVR and LnRMSSD are HR-dependent, sensitivity analyses were performed using repeated-measures analysis of covariance (ANCOVA) models with HRpeak included as a covariate. Mode × time interactions for both SEVR and

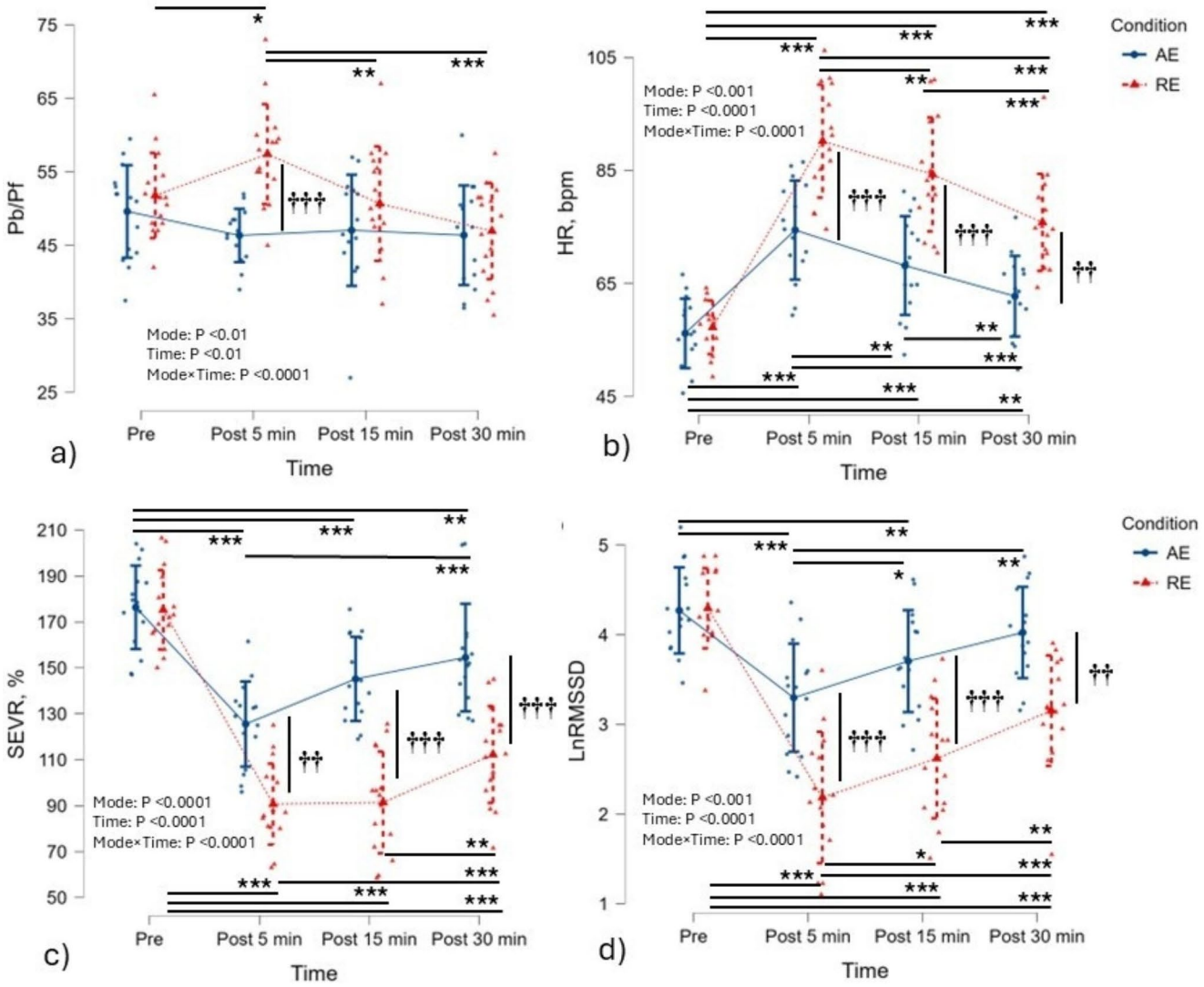

Fig. 3 Mode × time plots showing changes in **a** reflection magnitude (Pb/Pf), **b** heart rate (HR), **c** subendocardial viability ratio (SEVR), and **d** the natural log of the root mean square of successive differences (LnRMSSD) measured pre-exercise and at 5, 15, and 30 min post-exercise following aerobic (AE) and resistance exercise (RE). Data are presented as individual responses with group means ± standard deviation. Horizontal black lines indicate significant within-mode post-hoc comparisons, with significance denoted by *$P<0.05$, **$P<0.01$, and ***$P<0.001$. Vertical black lines indicate significant between-mode differences at corresponding time points, with significance denoted by †$P<0.05$, ††$P<0.01$, and †††$P<0.001$

LnRMSSD remained significant (both $P<0.0001$), while HRpeak was not independently associated with either outcome (Ps = 0.18 to 0.68).

Influence of baseline blood pressure

Given the elevated resting blood pressure observed in several participants and evidence that baseline blood pressure influences postexercise hypotension (Pescatello et al. 2019), exploratory analyses examined associations between resting and postexercise blood pressure responses. Following AE, baseline brachial SBP demonstrated a moderate inverse association with the change in brachial SBP at 5 min ($r = -0.50$, $P=0.056$), suggesting that participants with higher resting SBP tended to experience larger post-exercise SBP reductions. The strength of this association diminished progressively at 15 min ($r = -0.44$, $P=0.103$) and 30 min ($r = -0.34$, $P=0.208$). In contrast, associations were consistently weaker following RE ($r = -0.17$ to -0.30, $P=0.282$–0.547).

For DBP, baseline values were not associated with early recovery responses (i.e., change at 5 min post-exercise) following AE or RE (Ps > 0.49). However, moderate inverse associations emerged later in recovery. Following AE, baseline DBP was associated with the change in brachial DBP at 30 min ($r = -0.53$, $P=0.043$). Following RE, baseline DBP

was associated with changes in brachial DBP at both 15 min ($r = -0.53$, $P = 0.041$) and 30 min ($r = -0.55$, $P = 0.033$), indicating that participants with higher resting DBP tended to exhibit larger DBP reductions during later stages of recovery.

Discussion

This study provides the first integrated characterization of central hemodynamic and cardiac autonomic responses following acute AE versus RE in young Black men. Consistent with our hypothesis, RE produced larger and more sustained elevations in central loading and wave reflection, greater and more persistent reductions in SEVR, and lower central DBP across recovery than AE, whereas central SBP declined over time irrespective of mode. Concurrently, RE elicited greater and more prolonged elevations in HR with lower LnRMSSD, indicating delayed cardiac parasympathetic reactivation. These differences remained significant after adjustment for HRpeak during exercise. Collectively, our results indicate that RE elicits a coordinated physiological response characterized by elevated central afterload, reduced myocardial oxygen supply–demand balance, and delayed parasympathetic recovery, reflecting a greater transient cardiovascular strain during early post-exercise recovery compared with AE.

AIx75 spiked at 5 min post-RE and remained elevated through 30 min, whereas AE produced a more modest increase at 5 min that returned to baseline thereafter. These responses likely reflect a combination of increased peripheral vasoconstriction, altered reflection site timing, and larger forward and reflected wave amplitudes following RE, contributing to sustained late-systolic pressure augmentation. Concurrent increases in Pf and Pb suggest that both forward wave generation and wave reflection contributed to the elevated AIx75 following RE, indicating a combined influence of cardiac ejection dynamics and peripheral vascular properties. This pattern is consistent with the notion that RE elicits greater central afterload and wave reflection than AE in the acute recovery period, likely due to factors such as more intense sympathetic activation, sustained vascular tone changes, and compressive effects from heavy compound lifting (Wakeham et al. 2025). Results from a prior mode comparison study involving a sample of recreationally trained young men (race/ethnicity not stated) agree with our findings, showing sustained post-RE (6 exercises, 3 × 10 repetitions, 80–90% of 10-RM) elevations but transient changes after AE (30 min cycling, 70–75% of age-predicted maximum HR) (Pierce et al. 2018b). Moreover, the magnitude of increase in AIx75 was similar, as we observed mean absolute increases of ~33, 21, and 10% points (pp) at 5-, 15-, and 30-min post-RE which closely align with increases of ~30, 20, and 12 pp at 10, 20 and 30 min post-RE (Pierce et al. 2018b). In further agreement with these magnitudes, other investigations involving an acute bout of total body RE in young men reported mean absolute increases of ~19–22 pp at a single 10 min post-exercise time point (Kingsley et al. 2017; Tai et al. 2018).

Further supporting this interpretation, AP and Pb peaked at 5 min post-RE and remained significantly elevated at 15 min before partially recovering by 30 min, whereas they were unchanged after AE. Pf was unchanged post-AE but increased at all post-RE time points, while Pb/Pf transiently increased at 5 min post-RE with no changes post-AE. To our knowledge, only one prior study assessed Pf, Pb, and Pb/Pf at multiple time points post-RE (Pierce et al. 2018b). Contrasting with our more persistent RE-induced elevations in AP, Pf, and Pb, and transient increase in Pb/Pf at 5 min, Pierce et al. (2018b) reported only transient increases in Pf and Pb at 10 min post-RE and no effect on Pb/Pf across 10–60 min. This divergence likely reflects protocol differences, as we placed greater emphasis on barbell compound movements and used heavier loading (95% vs. 80–90% of 10-RM), which would impose a greater compressive and reflexive vascular load and contribute to more sustained elevations in AP, Pf, and Pb (Wakeham et al. 2025). Moreover, because AIx75 reflects the net effect of reflection timing, reflection magnitude, and cardiac ejection dynamics on late-systolic pressure augmentation, it may remain elevated despite partial recovery of individual wave-separation components (Sharman et al. 2009; Wilkinson et al. 2000). These persistent alterations in wave reflection suggest prolonged late-systolic loading during recovery, which may contribute to downstream effects on myocardial oxygen supply–demand balance.

SEVR declined sharply following RE and remained significantly depressed throughout recovery, consistent with the sustained elevations in AIx75 and wave reflection indices observed across this period. High-intensity RE appears to depress SEVR by increasing HR, which reduces the diastolic-pressure time index, and by accelerating aortic diastolic pressure decay, which further limits diastolic perfusion relative to systolic demand, and mediation analysis identifies the diastolic decay index as the pathway linking RE-related aortic stiffening to lower SEVR (Tagawa et al. 2019). This interpretation is further supported by the concurrent reductions in central DBP following RE, which may reduce the pressure gradient driving coronary perfusion during diastole. Reductions in SEVR of slightly lesser magnitude (–58 to −69 pp) relative to our findings (–84 pp at 5 and 15 min post-RE) have been reported following acute total body protocols assessed at a single recovery point within 10–20 min (Kingsley et al. 2017; Parks et al. 2020). This

discrepancy may reflect a larger exercise dose in the present study (six exercises, 3 × 10) compared with three-exercise protocols of similar set–repetition and intensity prescription, as well as the emphasis on heavy barbell compound movements that impose substantial intramuscular and intrathoracic pressure. Nevertheless, SEVR values remained above the critical threshold (~45%) associated with subendocardial ischemia (Salvi et al. 2021), indicating that while myocardial oxygen supply-demand was transiently compromised, it was never reduced to clinically concerning levels. Meanwhile, although SEVR also decreased after AE, values remained significantly higher than those observed at corresponding post-RE time points. Taken together with elevated AIx75 and reduced DBP, these findings indicate that RE promotes greater systolic loading, reduced diastolic perfusion, and a less favorable myocardial oxygen supply–demand balance during early recovery.

Both brachial and central DBP showed significant mode × time interactions, with persistent reductions throughout post-RE recovery, whereas DBP remained stable after AE. Brachial SBP remained unchanged relative to baseline across recovery for both AE and RE, whereas central SBP declined over time irrespective of mode. The preferential reduction in DBP after RE is consistent with a greater decline in peripheral vascular resistance, accelerated aortic diastolic pressure decay, and diminished aortic recoil, which reduces the diastolic pressure reservoir available for coronary perfusion (Tagawa et al. 2019). In contrast, the maintained or only modestly reduced SBP may reflect the opposing influence of elevated late-systolic augmentation (i.e., higher AIx75, AP, and Pb). These differential central versus peripheral and systolic versus diastolic responses suggest that the hemodynamic mechanisms underlying post-exercise blood pressure regulation are mode-specific. Moreover, exploratory analyses suggest that baseline blood pressure may further influence the magnitude and time course of post-exercise blood pressure responses. Like our findings, Pierce et al. (2018b) reported significant post-RE reductions only for brachial DBP throughout 10–60 min of recovery, but also found transiently increased brachial SBP at 10 min post-RE (Pierce et al. 2018b). Unlike our findings of persistent reductions, others have reported no post-RE DBP reductions within 10–20 min post-RE (Kingsley et al. 2017; Parks et al. 2020; Heffernan et al. 2007a), which may stem from differences in recovery duration, exercise protocol, or population characteristics. Collectively, our findings suggest that the post-RE reduction in DBP was not an isolated response but occurred alongside increased wave reflection and reduced SEVR, indicating coordinated alterations in vascular loading and myocardial oxygen supply-demand balance during recovery.

HR was significantly increased throughout recovery for both modes, with progressive yet incomplete restoration across time, and values consistently higher after RE than AE. LnRMSSD exhibited a reciprocal mode × time pattern, albeit with restoration to baseline values by 30 min post-AE. Faster parasympathetic reactivation following moderate intensity AE is consistent with findings from a recent systematic review demonstrating that RMSSD typically recovers to near-baseline levels within 30 min (Michael et al. 2017). Greater and more persistent cardiac parasympathetic disturbance post-RE may reflect stronger central command from greater motor unit recruitment, augmented metaboreflex activation from higher glycolytic demand and metabolite accumulation, higher intramuscular pressure with associated pressor responses, and delayed baroreflex-mediated vagal reactivation during recovery (Williamson et al. 2006; Gallagher et al. 2001; Amann et al. 2015). Although these neural mechanisms were not directly measured, they provide a plausible explanation for the sustained autonomic disturbance observed after RE. This delayed parasympathetic reactivation is consistent with the sustained central pulsatile and wave-reflection load observed post-RE, as autonomic and baroreflex recovery are closely coupled with the timing and magnitude of late systolic pressure augmentation during early recovery. Accordingly, the persistent elevation in central hemodynamic load, reductions in DBP, and impaired myocardial supply–demand balance observed following RE may be further reinforced by delayed autonomic recovery, highlighting the interdependence of vascular and autonomic responses during early post-exercise recovery. Current acute responses to RE align with a recent meta-analysis demonstrating significant RMSSD suppression between 8 and 30 min post-RE, with greater effects after higher-volume protocols similar to ours (Marasingha-Arachchige et al. 2022).

Strengths of this study include the focus on an underrepresented sample of young Black men, integration of central hemodynamics and cardiac autonomic recovery, and the use of exercise bouts that did not differ significantly in session duration or mean HR, thereby minimizing differences in overall cardiovascular workload between modes. Although peak HR was higher during RE, sensitivity analyses demonstrated that mode-specific differences in SEVR and LnRMSSD were not attributable to differences in peak chronotropic strain alone. However, several limitations warrant consideration. First, a non-exercise control condition was not included. However, prior work suggests that these outcomes are stable during comparable resting periods in young men (Pierce et al. 2018b; Christiani et al. 2021). Second, the study did not include a non-Black comparison group. Therefore, future research should determine whether previously reported racial differences in post-exercise

responses extend to RE. Third, recovery was assessed for only 30 min, capturing early but not longer-term recovery dynamics. Fourth, autonomic mechanisms were inferred from HR and HRV without direct measures of sympathetic nerve activity or baroreflex sensitivity. Fifth, energy expenditure was not directly measured, limiting our ability to determine the contribution of between-mode differences in metabolic stress. Additionally, the mode-specific associations observed between baseline blood pressure and post-exercise blood pressure responses were exploratory and warrant confirmation in larger cohorts. Finally, the sample consisted of only young Black men, which reduced physiological variability and allowed focused examination of an understudied population. Future studies should examine responses in Black women and across different age groups.

Conclusion

In young Black men, a single bout of RE elicited greater and more sustained elevations in central hemodynamic load, more pronounced depression of myocardial oxygen supply–demand balance, and delayed cardiac parasympathetic reactivation compared with AE. These findings represent the first integrated assessment of these responses in this underrepresented population and demonstrate that RE imposes a higher transient cardiovascular strain during the early post-exercise period, even when session duration and exercise HR are similar. Although SEVR remained above thresholds associated with subendocardial ischemia and both modalities are safe and evidence-based, the data suggest that exercise programming in young Black men should account for modality-specific recovery kinetics, particularly when central afterload or autonomic recovery is a consideration (e.g., in individuals with hypertension).

Acknowledgements We thank all participants for volunteering their time and commitment to this study.

Author contributions A.C.E.B., A.T.P., C.D.M., and S.L.B. contributed to data collection. A.A.F. conceived the study, developed the methodology, and provided supervision. A.C.E.B. and A.A.F. drafted the original manuscript. G.J.G. and A.A.F. contributed to data interpretation and provided critical review and editing. All authors reviewed and approved the final version of the manuscript.

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

Declarations

Conflict of interest No external funding was received for this study. The body composition analyzer was provided by InBody. G.J.G. is employed by WHOOP, Inc. and holds stock options in the company. All other authors declare no conflicts of interest.

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