

Effects of Short-Duration Cycling After Resistance Exercise on Aortic Stiffness and Next-Day Recovery in Strength-Trained Men

Joseph D. Vondrasek,^{1,2} James E. Brown,¹ Gregory J. Grosicki,^{1,3} and Andrew A. Flatt¹

¹Biodynamics and Human Performance Center, Department of Health Sciences and Kinesiology, Georgia Southern University, Savannah, Georgia; ²Cardiovascular and Applied Physiology Laboratory, Department of Health, Nutrition, and Food Sciences, Florida State University, Tallahassee, Florida; and ³Performance Science, WHOOP Inc., Boston, Massachusetts

Abstract

Vondrasek, JD, Brown, JE, Grosicki, GJ, and Flatt, AA. Effects of short-duration cycling after resistance exercise on aortic stiffness and next-day recovery in strength-trained men. *J Strength Cond Res* XX(X): 000–000, 2026—We aimed to determine whether 10 minutes of cycling after resistance exercise (RE) mitigates acute aortic stiffening and affects next-day recovery markers in trained men. Twelve men (age: 23 ± 4 years, BMI: 26.7 ± 3.4 kg/m², RE experience: 4 ± 2 years) completed identical upper-body RE sessions on separate days, with and without post-RE cycling (70% age-predicted maximal heart rate). Carotid-femoral pulse wave velocity (cfPWV) was assessed pre-RE, 5 minutes post-RE, and again 15-, 25-, and 40-minute postcycling intervention or time-matched control. Heart rate variability and maximal mean concentric barbell bench press velocity (MCV) were assessed pre-RE and 24 h post-RE. Perceived recovery and soreness were assessed 24 hours post-RE. Significance was set at $p \leq 0.05$. No condition \times time interaction ($p = 0.16$) was found for cfPWV. Irrespective of condition, 5-minute post-RE cfPWV (6.0 ± 0.7 m/s) was higher than all other time points (5.5 ± 0.6 – 5.6 ± 0.6 m/s; $ps < 0.0001$). Greater cfPWV reductions at 40 minutes post-RE, relative to immediately post-RE, correlated with lower fitness (i.e., lower relative cycling power output, [W/kg]; $r = 0.732$, $p < 0.01$) even after adjusting for increases in cfPWV 5 minutes post-RE. Heart rate variability and MCV were not different from pre-RE at 24 hours ($ps > 0.05$). Similarly, perceptual measures did not differ between conditions at 24 hours ($ps > 0.05$). A 10-minute cycling bout post-RE did not alter aortic stiffness or next-day recovery responses but was more effective in reducing cfPWV in subjects with lower aerobic fitness.

Key Words: cardiovascular, pulse wave velocity, autonomic, aerobic exercise, sports medicine

Introduction

The aorta is the largest artery in the body and is uniquely elastic because of its high density of elastin. Distensibility helps dampen pulsatile flow and regulate blood pressure (BP), protecting sensitive microcirculatory beds (3). However, aortic stiffening compromises buffering capacity, which increases pulse pressure, promotes isolated systolic hypertension, and damages target organs like the brain and kidneys (3). It also impairs stretch-sensitive baroreceptors, attenuating cardio-protective vagal outflow (32). Furthermore, aortic stiffness increases with advancing age and is an independent risk factor for cardiovascular disease (19,25). Carotid-to-femoral pulse wave velocity (cfPWV), the gold-standard measure for the noninvasive assessment of aortic stiffness (38), demonstrates substantial clinical relevance, as every 1.0 m/s increase is associated with a 15% rise in all-cause and cardiovascular mortality (39). Therefore, preserving aortic elasticity emerges as a critical target for healthy aging.

Chronic resistance training (RT) enhances bone density, glucose regulation, muscular strength, functional capacity, mental

health, and reduces BP (10,45). Physical activity guidelines (22) emphasize the importance of RT for overall health by encouraging muscle-strengthening exercise 2–3 days per week. Nevertheless, acute resistance exercise (RE) transiently increases aortic stiffness (7,15,46), and some studies report increased resting cfPWV after short-term RT interventions (26). Notably, increases in cfPWV ≥ 1.0 m/s have been reported after ≤ 12 weeks of RT (5,44). Moreover, in cross-sectional studies involving resistance-trained individuals, greater aortic stiffness associates with end-organ damage (42). This may help explain why systematic reviews and meta-analyses of cohort studies indicate that the mortality benefits of RT diminish at doses >40 – 60 minutes per week and may potentially increase risk beyond 150 minutes per week (27,33). Therefore, strategies that maintain the health benefits of RT while preventing aortic stiffening are an important topic that requires further investigation.

Aerobic exercise (AE) reduces aortic stiffness acutely (18) and chronically (28). Moreover, routinely performing 30 minutes of moderate intensity AE immediately post-RE prevents RT-induced aortic stiffening (16). However, this duration of AE may be unappealing to resistance trainers targeting muscle strength and hypertrophy because of a lack of training specificity and/or concerns about the potential for interference with recovery or chronic adaptations (30). Shorter AE protocols using a low-impact modality (e.g., cycling) limit interference effects (30) and may be less aversive to resistance trainers. In fact, 15 minutes of moderate

Address correspondence to Andrew A. Flatt, aflatt@georgiasouthern.edu.

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intensity cycling transiently reduces aortic stiffness in young men (18) and enhances cardiac-autonomic and neuromuscular recovery after rugby training (20). Thus, we hypothesized that a short bout of cycling performed post-RE would counteract acute aortic stiffening effects without negatively affecting recovery markers. To test this, we investigated the effects of a 10-minute post-RE cycling intervention on aortic stiffness and next-day recovery status in well-trained men.

Methods

Experimental Approach to the Problem

We used a counter-balanced crossover design with 5 laboratory visits. The study design is summarized in Figure 1, with details for each visit in the subsequent sections. We asked subjects to abstain from additional exercise throughout the study period and arrive for visits in a recovered state (no RE within 72 hours). On the day of each visit, we confirmed subjects had fasted and avoided consuming preworkout supplements (≥ 6 hours for visit 1; ≥ 3 hours for visits 2–5) and avoided prior alcohol (≥ 24 hours) and caffeine intake (≥ 12 hours). During visit 1, subjects provided written consent and completed exercise and training history questionnaires. Next, we measured and recorded subjects characteristics (anthropometrics, body composition, and metabolic biomarkers). Subsequently, we assessed muscular strength for the barbell bench press and barbell biceps curl and familiarized subjects with barbell velocity testing for the bench press. Finally, we familiarized subjects with the interventional fan bike cycling protocol at their target heart rate. Visits 2 and 4 included RE followed by the intervention (fan bike cycling) or control (seated rest) condition.

All aortic stiffness (cfPWV) and brachial BP measurements were taken after a 5-minute supine stabilization period. We measured cfPWV and brachial BP before RE (Pre-RE), after RE

(Post-RE_{5min}), after intervention/control, which was 15 minutes after RE (Post-RE_{15min}), 15 minutes after cycling/control, which was 25 minutes after RE (Post-RE_{25min}), and 30 minutes after cycling/control, which was 40 minutes after RE (Post-RE_{40min}). Heart rate variability (HRV) parameters were obtained at Pre-RE and Post-RE_{40min} at visits 2 and 4. During visits 3 and 5, we obtained next-day HRV (i.e., Post-RE_{24h}), and perceptual (perceived soreness and recovery) and neuromuscular (barbell velocity) recovery status.

Subjects

We recruited resistance-trained men (18–30 years old) who self-reported ≥ 2 years of RT experience (≥ 3 d/wk). Because RT-induced aortic stiffening is less common in women, we targeted men (11). Exclusion criteria were the use of tobacco products, heart-altering medications, musculoskeletal injury within the past 6 months, or any cardiovascular, metabolic, or neurological conditions. All subjects provided written and informed consent before beginning the study, and all procedures were approved by the Institutional Review Board of Georgia Southern University (H23017).

Descriptive Characteristics

Subjects completed survey questions about training history, strength-sport participation, and AE habits. We then measured height with a stadiometer, body mass with a digital scale, and body composition via dual-energy X-ray absorptiometry (DXA; GE Lunar, GE HealthCare, Chicago, IL) (14,34). The DXA machine was calibrated before each scan per the manufacturer’s instructions. A trained research technician performed all scans (JDV or JEB). Next, a lancet was used to perform a finger capillary blood draw to obtain a fasting metabolic profile (glucose,

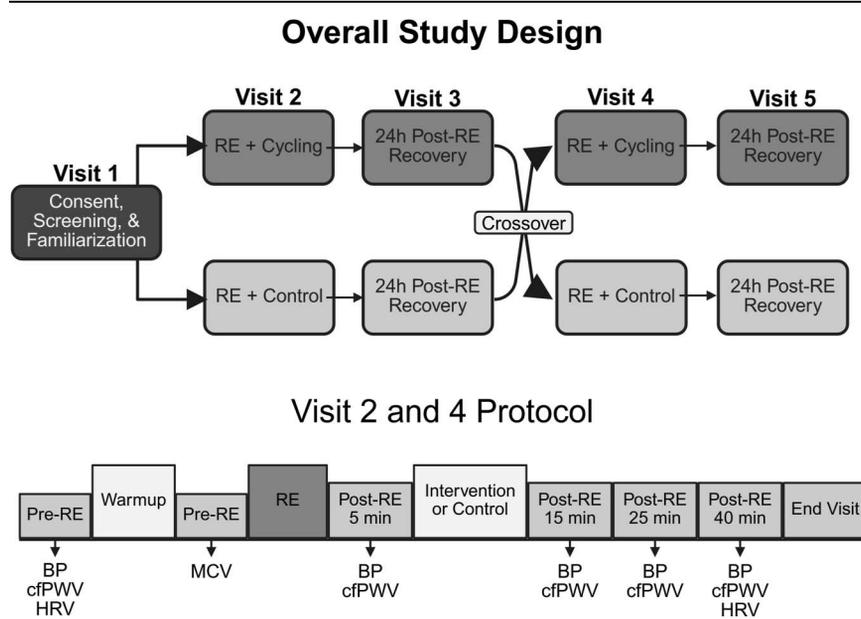


Figure 1. Experimental approach to the problem. HRV = heart rate variability; BP = blood pressure; cfPWV = carotid-to-femoral pulse wave velocity; MCV = mean concentric velocity; RE = resistance exercise; Post-RE_{5min} = immediately after RE; Post-RE_{15min} = after cycling/control, which was 15 minutes after RE; Post-RE_{25min} = 10 minutes after control or intervention, which was 25 minutes after RE; Post-RE_{40min} = 30 minutes after control or intervention, which was 40 minutes after RE; Post-RE_{24h} = 24 h after RE; PRS = perceived recovery score; PSS = perceived soreness score; Intervention = 10-minute cycling on a fan bike; control = 10 minutes of seated quiet rest.

total cholesterol, low- and high-density lipoprotein concentrations) with a Cholestech analyzer (Alere Cholestech LDX Analyzer, Waltham, MA) (1).

Baseline Strength

After blood analysis and before baseline strength testing, subjects were permitted to consume a light snack (e.g., banana, protein bar). If a subject consumed a light snack, the snack type was matched across conditions. To begin the warm-up, each subject cycled on the fan bike for 3 minutes at a self-selected pace and then was guided through dynamic stretches for the upper and lower limbs. Next, we determined each subject's 5-repetition maximum (5RM) for barbell bench press, followed by their 10-repetition maximum (10RM) for the barbell arm curl. For the bench press, subjects completed 1 warm-up set of 10 repetitions with only the barbell (20 kg). Thereafter, subjects completed 5 repetitions with 50, 75, and 100% of their self-estimated 5RM. If the 5RM was not achieved after this process, weight was added to the barbell in 1–5 kg increments until the 5RM was obtained. There were 2 minutes of rest between each warm-up set. Similar procedures were followed to determine 10RM for the barbell arm curl. If a subject could not complete a set, the RM was recorded as the last completed set.

Fan Bike Familiarization

During familiarization, we recorded the seat height that each subject found most comfortable (~25° knee flexion) (31) and determined the cycling cadence (intensity) required to achieve the subject's target heart rate during the intervention. A previous study found that post-RE cycling at 60% of maximum heart rate for 30 minutes attenuated increases in aortic stiffness during 4 months of RT that were observed in a comparison group that performed only RT (16). Thus, with the goal of a time-efficient protocol, we selected a slightly higher intensity (70% of age-predicted [220-age] maximum heart rate) to compensate for the shorter duration (10 minutes) of our AE intervention. We measured heart rate with a Polar H10 (Polar Electro Oy, Kempele, Finland) during all cycling sessions (12). The heart rate strap electrodes were moistened to improve conductivity and worn at the level of the xiphoid process. The H10 was connected via Bluetooth to the Polar Beat application on a mobile device (iPhone XR, Apple Inc. Cupertino, CA). Subjects were instructed to use their arms and legs while cycling.

Hemodynamic Assessments

At visits 2 and 4 (RE visits), we measured brachial BP and cFPWV after 5 minutes of quiet supine rest in a dimly lit, temperature-controlled room (~21 °C) without excess noise or distractions at each time point. We measured brachial BP via oscillometry with an automated brachial cuff (SphygmoCor XCEL, AtCor Medical, Naperville, IL). Measures were taken in duplicate or until 2 measures had systolic BP differing by ≤ 5 mm Hg; the 2 closest measures were averaged.

We assessed aortic stiffness with cFPWV (SphygmoCor XCEL, AtCor Medical) via applanation tonometry using the subtraction method. We placed a tonometer on the site of the strongest carotid pulse while a thigh cuff simultaneously inflated to a subsystolic pressure. Carotid-femoral pulse wave velocity was calculated as $cFPWV = D/PTT$ where D is the arterial path length and PTT is the pulse transit time. We measured arterial path

length (D) with a large board compass and a nonelastic tape measure. D was measured as the linear distance from the suprasternal notch to the top of the cuff at the superficial femoral artery, subtracting the distance from the suprasternal notch to the common carotid artery palpation site and the distance from the femoral pulse to the top of the cuff. PTT was measured with a proprietary algorithm that automatically calculated the time between the foot of the proximal pressure waveform (carotid) and the foot of the distal (femoral) pressure waveform (Butlin et al., 2013). One trained investigator (JDV) completed cFPWV measures until 2 measures differed by ≤ 0.5 m/s; the 2 closest measures were averaged (mean absolute difference: 0.13 m/s).

HRV

Cardiac-autonomic status was characterized via supine and standing HRV with spontaneous breathing, obtained immediately after hemodynamic assessment. We placed 3 electrodes in a modified lead II configuration for electrocardiographic (ECG) recording. Subjects remained supine for 3 minutes while RR intervals were recorded. Next, subjects were instructed to stand quietly beside the examination table with their arms relaxed at their sides for 3 minutes. We marked the ECG acquisition file at the time of postural change. RR interval data were transmitted from the integration belt (Bionomadix, Biopac Systems Inc., Coletta, CA) to the data acquisition system (MP 160, Biopac Systems Inc.) at a sampling rate of 1,000 Hz (40). We recorded RR interval data in AcqKnowledge software 5.0 (BIOPAC Systems Inc.) and exported files to Kubios HRV Premium software (Version 4.2.1, University of Kuopio, Kuopio, Finland) for HRV analysis. We visually inspected files for ectopic beats and performed automatic filtering via the Kubios "auto-correction" method (23). Parameters recorded for analysis included mean RR interval and the natural log of the root-mean square of successive differences (ln-RMSSD). The first min of each 3-minute recording was a stabilization period, and HRV parameters were calculated from the final 2 minutes (8). The ln-RMSSD is a time domain HRV index reflective of cardiac-parasympathetic activity that is commonly used for day-to-day monitoring in resistance trainers (9).

Neuromuscular Performance and Recovery

We assessed neuromuscular performance via a barbell velocity test using a linear position transducer (GymAware LE, Kinetic Performance Technology, Canberra, Australia) after the warm-up and before RE during visits 2 and 4 (6). The transducer tether was attached to the barbell loaded with the subjects' bench press 5RM. Up to 3 single repetition attempts were provided to determine maximal mean concentric velocity (MCV). To perform the test, we instructed subjects to "un-rack the barbell, lower the barbell under control, pause with the barbell motionless on the chest for ~1 s, and then press the barbell as hard and fast as possible." If the MCV for the second attempt was higher than the first, subjects completed a third trial. The 2 highest MCVs were averaged. This procedure was repeated at Post-RE_{24h} during visits 3 and 5.

RE

The RE session included 5 sets of 5 repetitions of the barbell bench press using 95% of the subject's 5RM with 90-second rest between sets. After 2 minutes of rest, subjects completed 5 sets of 10 repetitions of barbell arm curls using 95% of their 10RM with 90-

second rest between sets. This protocol was selected because it reliably increases cfPWV (21). If all repetitions of a set could not be completed, the weight was reduced for the next set (21). All RE sessions were supervised to ensure that exercises were performed with the correct technique.

Fan Bike Intervention or Control

The 10-minute fan bike intervention or 10-minute seated rest control period occurred immediately after Post-RE_{5min} measurements. For the intervention, subjects completed 10 minutes of stationary upper and lower limb cycling on the fan bike (Echo, Rogue Fitness, Columbus, OH). Subjects were instructed to match the cadence established during the familiarization period to achieve the target heart rate. Exercise intensity was adjusted as needed to maintain the target heart rate. We recorded the average heart rate during cycling from the Polar Beat application. Immediately after the cycling intervention, subjects rated their perceived exertion (RPE) using the modified Borg scale (0 “no exertion,” 1 “very light,” 2–3 “light,” 4–5 “moderate,” 6–7 “hard,” 8–9 “very hard,” 10 “maximal”). We recorded the average power output (W) displayed on the fan bike’s digital screen. During the control condition, subjects sat quietly in a chair with their backs supported and feet on the floor for 10 minutes.

Perceptual Recovery Markers

Subjects rated their perceived recovery (PRS; 0 to 10; higher ratings reflect greater perceived recovery) and perceived soreness (PSS; 0 to 10; higher ratings reflect greater perceived soreness) at post-RE 24 hours (35).

Statistical Analyses

We used linear mixed models to examine variation in hemodynamic parameters. Fixed effects of each model were condition (intervention vs. control), time (Pre-RE, Post-RE_{5min}, Post-RE_{15min}, Post-RE_{25min}, and Post-RE_{40min}), and a condition × time interaction; subject identification was a random effect. Tukey’s Honest Significant Difference tests were used for post-hoc analyses. The same procedures were used to examine variation in cardiac-autonomic parameters and MCV, using their corresponding time points of analysis. Normality assumptions were confirmed with Shapiro-Wilk tests on model residuals. Cohen’s *d* (*d*) effect sizes were used to quantify standardized differences for parametric pairwise comparisons. We compared perceived recovery score [PRS] and perceived soreness score [PSS] scores at Post-RE_{24h} using a Wilcoxon signed-rank test. Pearson and partial correlation analyses were used to explore potential associations between subjects characteristics (e.g., body composition, strength, aerobic fitness inferred from relative average cycling wattage) and intervention effects on cfPWV while adjusting for covariates. *p* values <0.05 were considered statistically significant. Analyses were conducted in JMP Pro 16 (SAS Institute Inc. Cary, NC). Data are reported as mean ± SD or median (interquartile range).

Results

Subjects Characteristics

Twelve resistance-trained men (4.1 ± 2.0 years of training, 4–5 d/wk) participated in this study. Four subjects competed in

bodybuilding, powerlifting, or weightlifting; 11/12 subjects reported performing habitual AE; and 7/11 typically completed their AE session on the same day as RE sessions. Types of AE included running, cycling, incline treadmill walking, jumping rope, or stair climbing. Subject characteristics are presented in Table 1.

Aortic Stiffness: RE Transiently Elevates cfPWV, With No Effect of Condition

A significant main effect of time was observed for cfPWV (*p* < 0.0001). Post-hoc analyses showed that cfPWV at Post-RE_{5min} was greater than all other time points ($\Delta = 0.38\text{--}0.50 \text{ m}\cdot\text{s}^{-1}$; *P*s < 0.0001, *d* = 0.53–0.73). No condition (*p* = 0.738, *d* = 0.04) or condition × time interaction (*p* = 0.155, *d* = 0.00–0.79) effects were observed (Figure 2A).

BP: RE Induces Postexercise Hypotension, Independent of Condition

A significant main effect of time was observed for systolic BP (*p* = 0.006). Post-hoc analyses showed that systolic BP at Post-RE_{25min} was lower than Pre-RE, Post-RE_{5min}, and Post-RE_{15min} ($\Delta = -4.4$ to -5.0 mm Hg; *P*s ≤ 0.041, *d* = 0.53–0.62). No condition (*p* = 0.476, *d* = 0.10) or condition × time interaction (*p* = 0.254, *d* = 0.01–0.75) effects were observed (Figure 2B).

A significant main effect of time was observed for diastolic BP (*p* = 0.001). Post-hoc analyses showed that diastolic BP at Post-RE_{5min} was lower than all other time points ($\Delta = -3.0$ to -4.0 mm Hg; *P*s ≤ 0.019, *d* = 0.63–0.84). No condition (*p* = 0.822, *d* = 0.08) or condition × time interaction (*p* = 0.128, *d* = 0.01–1.22) effects were observed (Figure 2C).

Table 1

Subjects characteristics.*

Subjects characteristics (<i>n</i> = 12)	Mean ± SD or median [IQR]
Age (y)	23 ± 4
Height (cm)	180 ± 7
Mass (kg)	86.7 ± 12.8
BMI (kg/m ²)	26.7 ± 3.4
Blood markers	
Total cholesterol (mg/dl)	168 ± 24 [†]
High-density lipoprotein cholesterol (mg/dl)	43 ± 11 [†]
Low-density lipoprotein cholesterol (mg/dl)	100 ± 31 [†]
Glucose (mg/dl)	91 ± 7 [†]
DXA results	
Total lean mass (kg)	66.8 ± 8.6
Total fat mass (kg)	13.9 [4.9]
Body fat percentage (%)	18.7 ± 5.7
Android fat percentage (%)	19.6 ± 8.2
Gynoid fat percentage (%)	20.3 ± 6.5
Android/gynoid fat percentage ratio	0.95 ± 0.12
RE and cycling	
Bench press 5RM (kg)	99 ± 12
Biceps curl 10RM (kg)	35 ± 4
Target heart rate (70% of age predicted maximum, bpm)	138 ± 3
10-min average cycling heart rate (bpm)	133 ± 4
Percentage of target heart rate achieved (%)	68 ± 2
Cycling power output (W)	126 ± 26
Cycling RPE	5 ± 2

*Normally distributed data are shown as mean ± SD, and non-normally distributed data are shown as median [IQR].

[†]Missing subjects data. *n* for total cholesterol was 9, 2 subjects had values of “<100 mg/dl.” *n* for HDL = 11. *n* for LDL = 8. *n* for glucose = 11. DXA = dual-energy X-ray absorptiometry; 5RM = 5-repetition maximum; 10RM = 10-repetition maximum; RPE = rating of perceived exertion.

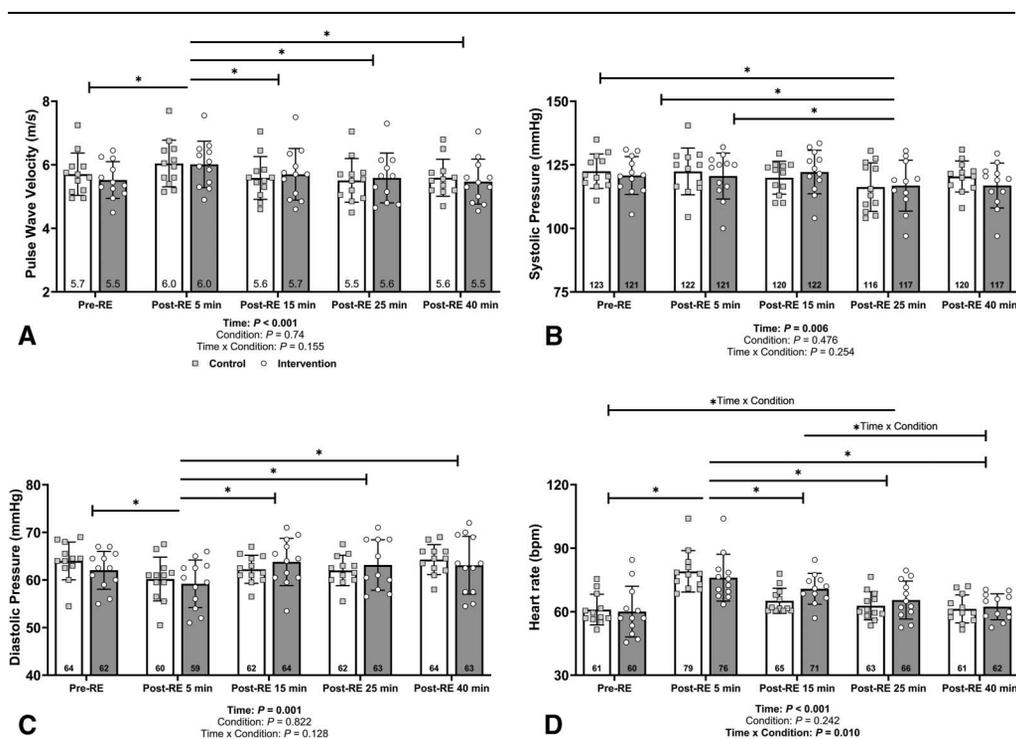


Figure 2. Time \times condition means and standard deviations for carotid-to-femoral pulse wave velocity (A), systolic blood pressure (B), diastolic blood pressure (C), and heart rate (D). Pre-RE = before the resistance exercise session (RE); Post-RE_{5min} = after the RE session; Post-RE_{15min} = after the intervention or control and 15 minutes after the RE session; Post-RE_{25min} = 15 minutes after the intervention or control and 25 minutes after the RE session; Post-RE_{40min} = 30 minutes after the intervention or control and 40 minutes after the RE session. * denotes significant difference ($p < 0.05$).

No time ($p = 0.066$, $d = 0.06$ – 0.51), condition ($p = 0.642$, $d = 0.02$), nor condition \times time interaction ($p = 0.093$, $d = 0.00$ – 0.91) effects were observed for MAP (see Figure 1, Supplemental Digital Content 1, <http://links.lww.com/JSCRA/760>).

Heart Rate: Acute Increases in Proportion to External Demand

A significant condition \times time interaction effect was observed for heart rate ($p < 0.010$). For control, heart rate at Post-RE_{5min} was significantly higher than all other time points ($\Delta = 13.9$ – 18.1 $\text{b}\cdot\text{min}^{-1}$; $P_s < 0.0001$, $d = 1.72$ – 2.13). For the intervention, heart rate at Post-RE_{5min} was significantly higher than all other time points ($\Delta = 11.3$ – 19.1 $\text{b}\cdot\text{min}^{-1}$; $p_s < 0.0001$, $d = 0.72$ – 1.87). In addition, Post-RE_{15min} was significantly higher than Post-RE_{40min} ($\Delta = 7.7$ $\text{b}\cdot\text{min}^{-1}$; $p = 0.017$, $d = 1.22$), and Post-RE_{25min} was significantly higher than baseline ($\Delta = 7.8$ $\text{b}\cdot\text{min}^{-1}$; $p = 0.021$, $d = 0.81$) (Figure 2D). No between-condition differences were observed for comparisons at the same time points ($P_s > 0.05$).

Lower Relative Cycling Power Output Is Associated With Reductions in cfPWV

Greater reductions in cfPWV at Post-RE_{40min} relative to Post-RE_{5min} were associated with lower relative cycling power output ($r = 0.732$, $p = 0.007$, Figure 3B) and greater increases in cfPWV at Post-RE_{5min} relative to Pre-RE ($r = -0.586$, $p = 0.045$). Partial correlation analysis demonstrated that for the intervention condition, lower relative cycling power output remained significantly associated with greater reductions in cfPWV at Post-RE_{40min} relative to Post-RE_{5min} ($r = 0.642$, $p = 0.033$) after controlling

for increases in cfPWV at Post-RE_{5min} relative to Pre-RE. Changes in MAP at Post-RE_{40min} relative to Post-RE_{5min} were not associated with changes in cfPWV at concurrent time points ($p > 0.05$). Pre-RE cardiovascular parameters (systolic and diastolic BP, cfPWV), body composition parameters, and strength parameters were not associated with changes in cfPWV ($p_s > 0.05$).

Supine HRV Parameters: Increased HR and Reduced HRV Post-RE

A significant condition \times time interaction was observed for supine mean RR ($p = 0.015$). For control, supine mean RR at Post-RE_{5min} was significantly lower than all other time points ($\Delta = -205.0$ – -222.5 ms; $p_s < 0.0001$, $d = 1.86$ – 2.31). For the intervention, supine mean RR at Post-RE_{5min} was significantly lower than all other time points ($\Delta = -174.3$ to -284.6 ms; $P_s < 0.0001$, $d = 1.83$ – 2.07), and supine mean RR at Post-RE_{40min} was significantly lower than Pre-RE ($\Delta = -110.3$; $p = 0.020$, $d = 0.61$) (Figure 4A).

A significant main effect of time was observed for supine lnRMSSD ($p < 0.0001$). Post-hoc analysis showed that supine lnRMSSD at Post-RE_{5min} was significantly lower than all other time points ($\Delta = -1.0$ to 1.2 ; $P_s < 0.0001$, $d = 1.86$ – 2.04). No condition ($p = 0.866$, $d = 0.00$) or condition \times time interaction ($p = 0.995$, $d = 0.00$ – 2.28) effects were observed (Figure 4B).

Standing HRV Parameters: Increased Heart Rate and Reduced HRV Post-RE

A significant main effect of time was observed for standing mean RR ($p < 0.0001$). Post-hoc analysis showed that standing mean

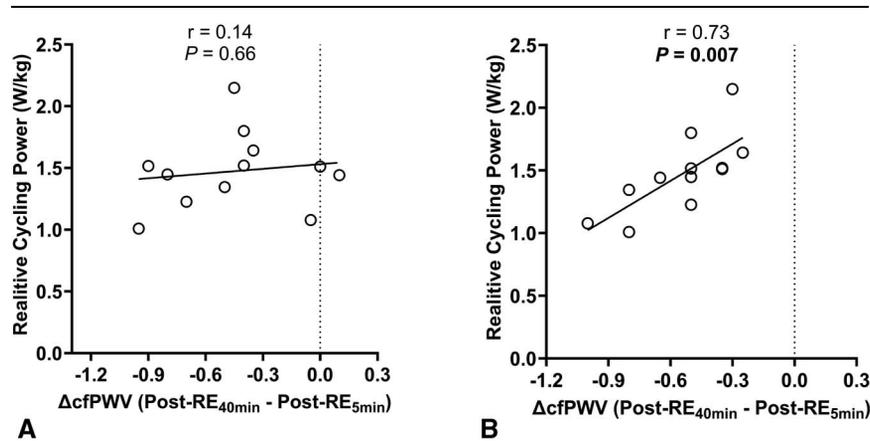


Figure 3. Scatterplots showing the association between relative cycling power output at the target heart rate and the change in carotid-to-femoral pulse wave velocity ($\Delta cfPWV$) from Post-RE_{5min} to Post-RE_{40min} during the control (A) and intervention (B) conditions. Post-RE_{5min} = immediately after the resistance exercise (RE) session. Post-RE_{40min} = 30 minutes after the intervention or control and 40 minutes after the RE session.

RR at Post-RE_{5min} was significantly lower than all other time points ($\Delta = -96.6$ to 158.8 ms; $P_s < 0.0001$, $d = 1.12$ – 1.67). In addition, standing mean RR at Post-RE_{40min} was significantly lower than Pre-RE and Post-RE_{24h} ($\Delta = -51.5$ to -62.2 ms; $P_s \leq 0.011$, $d = 0.68$ and 0.58). No condition ($p = 0.199$, $d = 0.13$) or

condition \times time interaction ($p = 0.051$, $d = 0.11$ – 1.87) effects were observed (Figure 4C).

A significant main effect of time was observed for standing ln-RMSSD ($p < 0.0001$). Post-hoc analysis showed that standing ln-RMSSD at Post-RE_{5min} was significantly lower than all other time

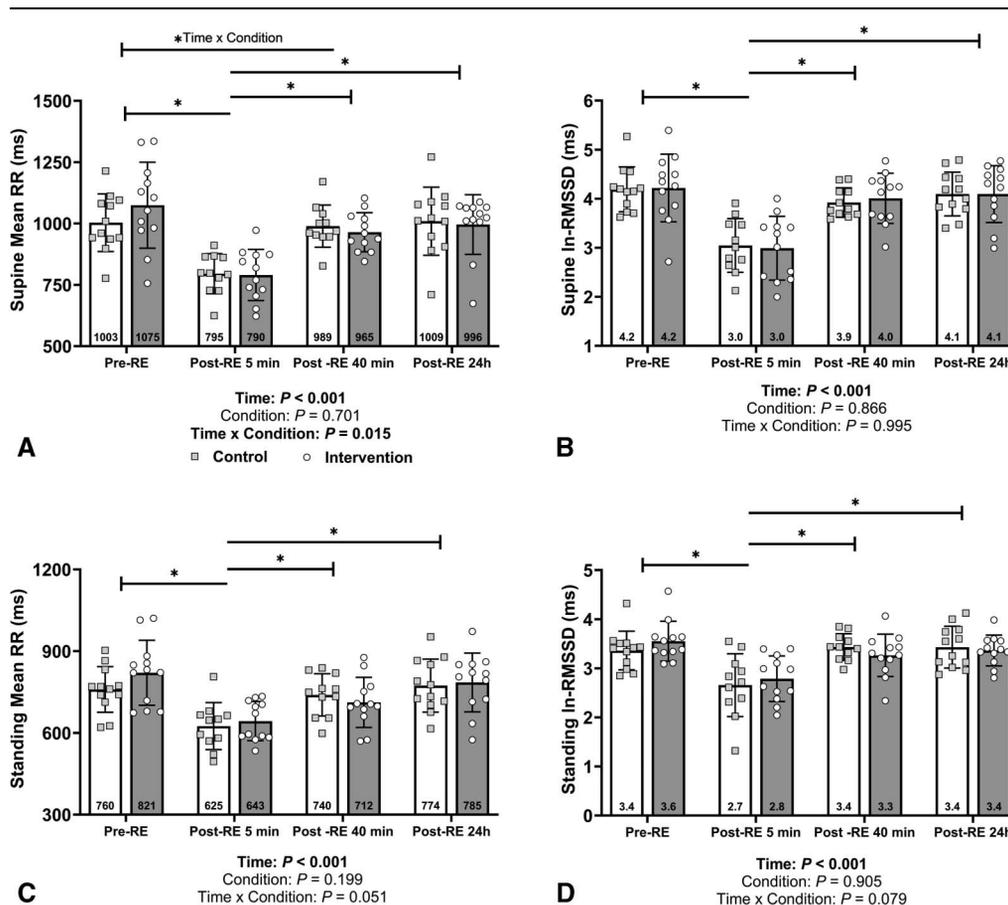


Figure 4. Time \times condition means and SD for supine mean RR (A), supine natural log of the root-mean square of successive differences (ln-RMSSD) (B), standing mean RR (C), and standing ln-RMSSD (D). Pre-RE = before the high-intensity resistance exercise session (RE); Post-RE_{5min} = after the RE session; Post-RE_{40min} = 30 minutes after the intervention or control and 40 minutes after the RE session; Post-RE_{24h} = 24 h after RE. * denotes significant difference ($p < 0.05$).

points ($\Delta = -0.62$ to -0.72 ; $P_s < 0.0001$; $d = 1.33$ – 1.53). No condition ($p = 0.905$, $d = 0.03$) or condition \times time interaction ($p = 0.079$, 0.00 – 1.75) effects were observed (Figure 4D).

Neuromuscular Recovery: No Change in MCV From Pre-RE to Post-RE_{24h}

No time ($p = 0.844$; $d = 0.04$), condition ($p = 0.627$; $d = 0.01$), or condition \times time interaction ($p = 0.775$, $d = 0.01$ – 0.05) effects were observed for MCV (Figure 5).

Subjective Recovery: No Difference in Subjective Status at Post-RE_{24h}

There was no difference in PRS ($p = 0.076$; control: 7 [2] vs. intervention: 8 [1]) or PSS ($p = 0.09$; control: 2 [2] vs. intervention: 4 [3]) at Post-RE_{24h} between conditions.

Discussion

Our primary aim was to determine whether 10 minutes of cycling performed after a bout of RE attenuates acute RE-induced increases in aortic stiffness among experienced resistance trainers. Although RE caused an immediate increase in cFPWV, subsequent values were not different from Pre-RE in either condition, revealing no effect of the intervention relative to seated rest (control). Despite no effects of cycling on cFPWV, lower relative cycling power output at the target heart rate was associated with greater cFPWV reductions after the intervention, but not the control. This indicates that aerobic fitness level may influence AE-induced destiffening effects after RE. Our secondary aim was to investigate the effect of post-RE cycling on subjective (PSS and PRS) and objective markers of recovery (MCV and ln-RMSSD). We found no significant impact of the intervention on status markers 24 h postexercise, indicating a neutral effect of performing short-duration AE after RE on next-day recovery.

Contrary to our hypothesis, the intervention did not elicit mean reductions in cFPWV beyond those observed in the control condition after RE. Although the acute increase in aortic stiffness at Post-RE_{5min} relative to Pre-RE was expected (4,7,15,17,21,46), its rapid return to baseline by Post-RE_{15min} in both conditions

was unexpected and helps explain the ineffectiveness of the intervention. Indeed, post-RE increases in aortic stiffness generally persist for 30–60 minutes based on studies predominantly involving recreationally active subjects (4,41,42). Thus, training status may affect the time course of post-RE aortic stiffness responses. This notion is supported by a recent study showing that although resting aortic stiffness (inferred from beta-stiffness index) significantly increased after a 4-week RT intervention in young untrained men, acute increases 30 minutes post-RE were attenuated from week 1 to week 4 (37). This suggests that habitual RE may reduce the duration of increased aortic stiffness after an acute bout, potentially explaining the relatively fast cFPWV return to baseline in our experienced sample. To our knowledge, only one study involving highly trained subjects examined cFPWV after an acute bout, showing significant increases 10 minutes post-RE, with no assessment at subsequent time points (17). Thus, our data provide new insight regarding RE-induced temporal changes in cFPWV, demonstrating accelerated cFPWV reductions after an acute bout relative to previous reports in more novice resistance trainers (42).

Although the intervention did not promote mean reductions in cFPWV, we found a significant association between changes in cFPWV (Post-RE_{40min} – Post-RE_{5min}) and the relative cycling power output (W/kg) required to elicit the target heart rate response during the intervention. Notably, this association persisted after adjusting for increases in cFPWV at Post-RE_{5min}. Furthermore, no significant association was observed after the control condition, and we ruled out potential confounding effects of Pre-RE cFPWV and concurrent changes in MAP ($P_s > 0.05$). Based on the established relation between W/kg at a fixed intensity and aerobic fitness (43), we interpret our findings to reflect greater aortic destiffening in less-fit individuals. Prior studies indicate that cFPWV responses to AE may be impacted by fitness level. For example, endurance-trained individuals show no significant change in cFPWV relative to baseline at 30 minutes after a 3-km run at race pace (29). In contrast, untrained individuals who performed 15 minutes of cycling at 65% peak oxygen uptake showed significantly reduced values at 30 minutes postexercise (18). Thus, our finding suggests that resistance trainers with lower aerobic capacity may derive greater acute vascular benefits from post-RE cycling.

We observed post-RT hypotensive effects for systolic and diastolic BP at Post-RE_{25min} and Post-RE_{5min}, respectively, while MAP remained stable across all time points, irrespective of condition. No change or transient reductions in BP variables have previously been observed post-RE in experienced resistance trainers when training at intensities that do not reach muscular failure (17,24). Importantly, stable MAP indicates that distension pressure does not explain increased cFPWV at Post-RE_{5min}, suggesting load-independent aortic stiffening. Rather, acute RE-induced increases in cFPWV are traditionally attributed to a combination of factors, including increased sympathetic vasomotor outflow and vascular smooth muscle (VSMC) tone, elevated heart rate, endothelial damage, reduced nitric oxide bioavailability, increased vasoconstrictive factors such as endothelin-1, and inflammation and oxidative stress (42). Furthermore, Wakeham et al. propose that acute BP spikes during RE induce viscoelastic changes in the arterial wall, contributing to transient stiffening (42). Specifically, they highlight stress relaxation hysteresis and energy dissipation from cyclic loading, which alter VSMC-extracellular matrix interactions (42). These effects typically resolve within 60 minutes post-RT as arterial wall mechanics normalize, with pressure redistributing from collagen to elastin fibers (42).

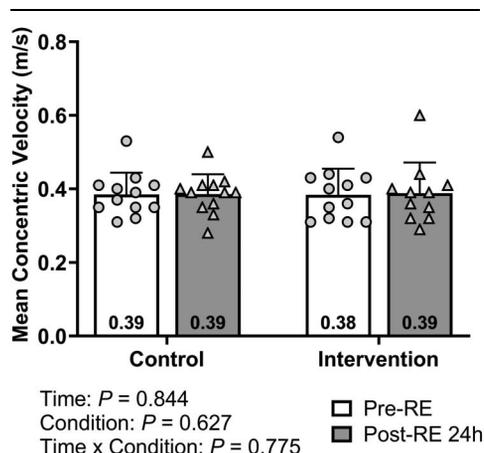


Figure 5. Time \times condition means and standard deviations for mean concentric barbell velocity during the bench press using the 5 repetition maximum load. Pre-RE = before the resistance exercise (RE); Post-RE_{24h} = 24 hours after the RE session.

Excluding one significant condition \times time interaction effect for supine mean RR, HRV responses were similar between conditions. Reduced HRV is expected immediately after RE (17) because of increased sympathetic activation and parasympathetic withdrawal, driven by factors such as elevated catecholamines, metabolic stress, and alterations in body temperature, fluid balance, and baroreflex sensitivity (36). This likely explains our findings of significantly reduced mean RR and ln-RMSSD at post-RE 5 minutes, irrespective of condition. However, supine mean RR was significantly reduced at Post-RE_{40min} relative to Pre-RE for the intervention, indicating that cycling slightly delayed heart rate recovery. Our finding of no change in next-day HRV agrees with previous work showing that parasympathetic HRV markers return to baseline by 24 hours post-RE in experienced resistance trainers (9), whereas significant reductions occur when a brief detraining period precedes the RE bout (2). Meanwhile, a previous study in elite rugby players reported a stimulatory effect of postmatch active recovery (15-minute cycling at 50% heart rate max) on autonomic function (postsubmaximal AE heart rate recovery) (20), although our 10-minute AE protocol did not elicit an ln-RMSSD rebound effect at Post-RE_{24h}.

We found no change in bench press MCV from Pre-RE to Post-RE_{24h}, irrespective of condition. Previous work in experienced resistance trainers (>1 year) found that bench press MCV (load corresponding to 1.0 m/s) was suppressed at 24 hours after RE when bench pressing 6 sets to failure with ~10 RM (9), but recovered to baseline by 24 hours when performing only 3 sets to failure with 8 RM (13). Similarly, subjective markers were not different between conditions at Post-RE_{24h}, irrespective of condition, unlike reductions observed 24 hours after a more intensive total-body bout of RE (9). Collectively, these findings indicate that 10 minutes of cycling on a fan bike did not enhance or interfere with our selected metrics of objective and subjective recovery in experienced resistance trainers.

Notable strengths of the study include the recruitment of and thorough characterization of experienced resistance trainers, consideration of next-day recovery status, and use of a practical AE intervention that may be less aversive than lengthier protocols. Several limitations should also be noted. An a priori sample size calculation was not performed because of the lack of comparable prior studies. Therefore, our sample size may have limited statistical power to detect smaller effects. Future studies with larger samples, informed by the effect sizes observed here, are needed to confirm these findings. Other notable limitations include an upper-body only protocol, use of age-predicted maximal heart rate to prescribe AE, and use of relative cycling intensity power output at the target heart rate to infer aerobic fitness. Thus, our interpretation of an association between lower aerobic fitness and greater aortic destiffening in response to the intervention requires confirmation in future research.

Practical Applications

A 10-minute bout of cycling was ineffective at countering acute RE-induced increases in cfpWV, likely because of the rapid and unanticipated cfpWV return to baseline by Post-RE_{15min} in both conditions. Thus, our findings suggest that accelerated post-RE cfpWV normalization may be an adaptation to habitual RE. Considering this observation, our findings also indicate that targeting the attenuation of acute post-RE increases in cfpWV is likely unnecessary. However, whether the intervention exerts chronic effects, such as

limiting long-term RT-induced increases in resting cfpWV, remains to be determined. Nevertheless, despite no effect of the intervention on cfpWV, it altered changes at the individual level relative to control, such that those with a lower relative cycling power output at the target heart rate exhibited greater reductions in cfpWV. This may indicate that resistance trainers with lower aerobic fitness may derive greater AE-induced destiffening effects after acute RE. However, this finding is preliminary, as the observed correlation does not establish causation. Finally, the AE intervention neither enhanced nor impaired recovery indicators, alleviating concerns about short-term AE interfering with next-day training status or performance.

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