Central and Peripheral Postexercise Blood Pressure and Vascular Responses in Young Adults with Obesity

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APPLIED SCIENCES

ABSTRACT

BUNSAWAT, K., E. C. LEFFERTS, G. GRIGORIADIS, S. O. WEE, M. M. KILIANEK, P. J. FADEL, P. S. CLIFFORD, B. FERNHALL, and T. BAYNARD. Central and Peripheral Postexercise Blood Pressure and Vascular Responses in Young Adults with Obesity. *Med. Sci. Sports Exerc.*, Vol. 53, No. 5, pp. 994–1002, 2021. **Introduction:** Adults with obesity are at an increased risk of incident hypertension. Regular aerobic exercise is recommended for the prevention and treatment of hypertension, but whether young adults with obesity exhibit impaired postexercise blood pressure (BP) and vascular responses remains unclear. **Purpose:** We tested the hypothesis that young adults with obesity exhibit attenuated postexercise hypotension (PEH) and postexercise peripheral vasodilation compared with young adults without obesity. **Methods:** Thirty-six normotensive adults without and with obesity (11 men and 7 women per group) underwent measurements of brachial and central BP, and leg blood flow (Doppler ultrasound) at baseline and at 30, 60, and 90 min after acute 1-h moderate-intensity cycling. Leg vascular conductance (LVC) was calculated as flow/mean arterial pressure. **Results:** Both groups exhibited similar brachial and central PEH (peak change from baseline, −2 and −4 mm Hg for brachial and central systolic BPs, respectively, for both groups; time effect, $P < 0.05$). Both groups also exhibited postexercise peripheral vasodilation, assessed via LVC (time effect, $P < 0.05$), but its overall magnitude was smaller in young adults with obesity (LVC change from baseline, +47% ± 37%, +29% ± 36%, and +20% ± 29%) compared with young adults without obesity (LVC change from baseline, +88% ± 58%, +59% ± 54%, and +42% ± 51%; group effect, $P < 0.05$). **Conclusions:** Although obesity did not impair PEH after acute moderate-intensity exercise, young adults with obesity exhibited smaller postexercise peripheral vasodilation compared with young adults without obesity. Collectively, these findings have identified evidence for obesity-induced alterations in the peripheral vasculature after exercise. **Key Words:** OBESITY, CENTRAL BLOOD PRESSURE, PERIPHERAL VASODILATION, ACUTE EXERCISE

Hypertension is an independent risk factor for cardiovascular mortality and morbidity (1). With the recent updates to the blood pressure (BP) guidelines from the American College of Cardiology and American Heart Association (2), nearly 50% of Americans now have hypertension (3). Importantly, adults with obesity are at an increased risk of incident hypertension (defined as a BP of 160/95 mm Hg on one occasion, a BP ≥140/90 mm Hg on two or more occasions, or use of antihypertensive medications) (4) and have a higher lifetime risk for cardiovascular mortality and morbidity compared with adults without obesity (5). Given the alarming prevalence and repercussions of hypertension, aerobic exercise has been recommended as a nonpharmacologic, lifestyle strategy for the prevention and treatment of hypertension (6). An acute bout of aerobic exercise has been reported to lower BP as much as 4–9 mm Hg in normotensive adults, a phenomenon termed "postexercise hypotension" (PEH) that may last nearly 2 h (7,8) and is accompanied by a sustained postexercise peripheral vasodilation (9). PEH after acute exercise also contributes to the long-term antihypertensive benefits of aerobic exercise training (7,10,11). Thus, investigation into the antihypertensive effects of a single bout of aerobic exercise is warranted in adults with obesity (4).

To date, few studies that have evaluated the influence of obesity alone on PEH have reported equivocal findings, with no information on postexercise peripheral vasodilation, highlighting a gap in knowledge (12–15). Although a similar magnitude of PEH has been previously reported in men who are overweight or have obesity (14), a recent study observed

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an absence of PEH in men with obesity compared with men without obesity (12). Furthermore, our group (13) and others (15) have demonstrated increased arterial stiffness after acute exercise in young adults with obesity. Arterial stiffness modulates central BP via alterations in forward and reflected wave pressures (16), and central BP is superior to brachial BP in predicting incident hypertension (17). Only one study, to date, has examined changes in central BP after acute exercise in young men with obesity (12), highlighting the need for additional studies to better understand the impact of obesity on PEH assessed in both the peripheral and central arteries.

The primary purpose of this study was to evaluate the impact of obesity on central and peripheral postexercise BP and vascular responses. We hypothesized that young adults with obesity would exhibit smaller or absent PEH for both central and brachial BPs, coupled with a smaller magnitude of postexercise peripheral vasodilation, compared with young adults without obesity. In addition, few studies have noted comparable magnitudes of PEH in both sedentary men and women without obesity, which were also accompanied by a sustained postexercise peripheral vasodilation (18,19). Given that the risk of incident hypertension is more strongly associated with obesity in women than in men (20), we performed subanalyses into the effect of obesity on PEH in men and women, separately to explore sex effects on PEH and postexercise peripheral vasodilation.

METHODS

Participants

Thirty-six young adults (18 adults without obesity (normal-weight) and 18 adults with obesity (obese)) volunteered to participate in the study. Each group had 11 men and 7 women. Young adults without and with obesity had a body mass index of 18.5–24.9 and 30.0–40.0 $kg \cdot m^{-2}$, respectively. All participants were sedentary having not engaged in regular aerobic exercise for the past 6 months (i.e., <60 min of moderate-to-vigorous physical activity per week based on a self-reported physical activity questionnaire). Exclusion criteria included any known cardiovascular, metabolic, renal, or respiratory disease. None were smokers or using any cardiovascular medications, nonsteroidal anti-inflammatory drugs, antihistamines, or multivitamin/antioxidant supplements, as indicated by a health history questionnaire. All participants provided written informed consent before participation. The institutional review board at the University of Illinois at Chicago approved all experimental procedures, which conformed to the guidelines set forth by the Declaration of Helsinki.

Study Design

Using a cross-sectional design, all participants reported to the laboratory for a screening visit and an experimental visit, separated by at least 3–5 d. For both visits, participants arrived at the laboratory in the morning after an overnight fast (12 h) and were instructed to refrain from exercise, caffeine, and alcohol (24 h). Female participants were studied during the early follicular phase of their menstrual cycle ($n = 8$) or during the placebo phase of oral contraceptives ($n = 6$). Room temperature was controlled (~22°C–24°C) for both visits.

Screening Visit: Protocol and Measurements

Upon arrival, all participants underwent assessments of anthropometrics, body composition, seated resting BP (left arm), venous blood draw, and peak oxygen consumption ($\dot{V}O_{2peak}$). Body composition and leg mass were determined using whole-body dual-energy x-ray absorptiometry (GE Lunar iDXA; GE Healthcare, Madison, WI). A venous blood draw was obtained as an experimental control to ensure similar blood glucose and lipid profile and thus no evidence of metabolic abnormalities (Alere Cholestech LDX Analyzer; Abbot, Hayward, CA). $\dot{V}O_{2peak}$ was measured using an open-circuit spirometry metabolic system (TrueOne 2400; Parvo Medics, Sandy, UT) testing during an incremental graded upright cycling exercise test performed to exhaustion (Excaliber Sport, Lode, the Netherlands) as described in detail in our previous work (21). The $\dot{V}O_{2peak}$ test was terminated when participants met three of the following four criteria: 1) rating of perceived exertion score of 17 or greater on the Borg scale (scale 6–20), 2) respiratory exchange ratio of ≥1.1, 3) no change in heart rate with a change in workload, and/or 4) volitional exhaustion (e.g., <60 rpm despite encouragement). The cycling exercise protocol was selected to support weight during locomotion in participants with obesity.

Experimental Visit: Protocol and Measurements

All measurements of resting hemodynamics, central BP, and leg blood flow were performed, with participants remaining quiet and relaxed in the supine position at baseline (pre) before acute exercise. After baseline measurements, participants completed 60 min of cycling exercise at 60% of $\dot{V}O_{2peak}$. Exercise of this intensity and duration has been reported to produce sustained PEH and postexercise peripheral vasodilation (22–24). Briefly, the participants began with a 5-min warm-up at the pedaling cadence of ≥60 rpm, while workload was gradually increased to elicit 60% of $\dot{V}O_{2peak}$. The participants were permitted to drink water *ad libitum* during acute cycling exercise. After exercise cessation, the participants returned to the supine position, after which hemodynamics and vascular measures were obtained at 30-, 60-, and 90-min postexercise. We selected these time points for the postexercise measurements, as previous studies have noted a sustained postexercise peripheral vasodilation through 90-min postexercise, whereas the PEH responses were transient and seemed to subside by 60-min postexercise (25,26).

Hemodynamics. Resting brachial BP was measured on the right arm after 10 min of quiet rest using an automated oscillometric cuff (HEM-907XL; Omron, Shimane, Japan). All measurements were made in duplicate. If values differed by >5 mm Hg, another measurement was obtained until the two values were within 5 mm Hg. Beat-to-beat BP was also continuously recorded on the left arm, which was supported

at heart level, using finger photoplethysmography (Finometer Pro, Amsterdam, the Netherlands), whereas beat-to-beat heart rate was recorded using an electrocardiogram at a sampling rate of 1000 Hz (Biopac Systems, Santa Barbara, CA) for a total of 5 min. Data were analyzed offline using WinCPRS (Absolute Aliens, Turku, Finland), and beat-to-beat BP waveforms were used to derive estimates of cardiac output and total peripheral resistance using Modelflow software, which incorporates age, sex, weight, and height (27). Modelflow-derived cardiac output and total peripheral resistance were then indexed to body surface area to derive cardiac index and total peripheral resistance index, respectively, and were reported as a change value from baseline (pre) during postexercise recovery.

Central BP. Radial pressure waveforms were collected on the right arm using applanation tonometry with a high-fidelity strain-gauge transducer (SphygmoCor; AtCor Medical, Sydney, NSW, Australia), as previously described (13). All waveforms were calibrated to brachial mean and diastolic BP. The radial pressure waveforms were further transformed algorithmically using a validated generalized transfer function to estimate central BP. Contour analysis of the radial pressure waveforms also derived: augmentation index (AIx) and augmented pressure. AIx was calculated as the percent of augmented pressure (difference between late and early systolic peaks of the waveform) to total pulse pressure, which was then normalized to a heart rate of 75 bpm (AIx@75). AIx and augmented pressure are associated with arterial stiffness (28). For quality control, all measures were made in duplicate. For measurements in which the values differed >5 mm Hg (or %), the measurement was repeated. In addition, wave separation analyses were performed on the central pressure waveforms to determine the forward and reflected wave pressures using the flow triangulation method (16).

Leg blood flow. Right common femoral artery diameter and mean blood velocity were imaged for 1 min via duplex Doppler ultrasound using a high-frequency linear array probe, ~2–3 cm proximal to the bifurcation, with an insonation angle of 60° (Hitachi-Aloka α-7, Tokyo, Japan). Dual mode was used to image common femoral artery diameter (B-mode) and Doppler velocity. The sample volume was placed in the middle of the artery with a large sampling area, but care was taken not to extend beyond the vessel wall. Images were recorded using Vascular Tools (Medical Imaging Applications, Coralville, IAUSA) during diastole and analyzed offline using automated edge-detection software (Brachial Analyzer; MIA, Coralville, IA). Mean blood velocity was analyzed using commercially available blood velocity analysis software (Cardiovascular Suite; Quipu, Pisa, Italy) as previously described (29). Leg blood flow ($mL \cdot min^{-1}$) was calculated as follows: (mean blood velocity ($cm \cdot s^{-1}$) $\times \pi \times$ [vessel diameter (cm)/2]$^2 \times 60$). Leg vascular conductance was calculated using brachial mean arterial pressure (MAP) obtained in conjunction with ultrasound recordings as follows: [leg blood flow ($mL \cdot min^{-1}$)/MAP (mm Hg)] $\times$ 100. Leg blood flow and leg vascular conductance were then normalized to leg lean mass (via iDXA).

Statistical Analysis

Statistical analyses were performed using SPSS (Version 21.0; IBM SPSS, Inc., Armonk, NY). Normality was confirmed with the Shapiro–Wilk test. Independent *t*-tests were used to identify group differences at baseline. All outcome variables were assessed with a 2 × 4 ANOVA with repeated measures (group (normal-weight vs obese) by time (preexercise and 30-, 60-, and 90-min postexercise)) to determine the PEH responses. For change values (the difference between postexercise values from preexercise values), a 2 × 3 ANOVA with repeated measures (group (normal-weight vs obese) by time (30-, 60-, and 90-min postexercise)) was performed. Subanalyses were also performed individually by sex to explore the impact of obesity on PEH in men and in women separately using a 2 × 4 ANOVA with repeated measures (group (normal-weight vs obese) by time (preexercise and 30-, 60-, and 90-min postexercise)). In case of a significant interaction, the Tukey method was used for α adjustment and *post hoc* analysis. Data are presented as mean ± SD. All *P* values were two-sided, and significance was established at $P < 0.05$.

RESULTS

Participant characteristics. Baseline descriptive characteristics are provided in Table 1. Groups were matched for age, sex, height, and resting brachial BP. However, by design, the obese group had higher weight, body mass index, waist circumference, percent body fat, android fat (visceral and subcutaneous), and android-to-gynoid ratio ($P < 0.05$). Blood lipid profile and $\dot{V}O_{2peak}$ data are provided in Table 2. No group differences were found for blood lipid profile ($P > 0.05$). Despite lower $\dot{V}O_{2peak}$ (normalized to body weight) in adults with obesity ($P < 0.05$), $\dot{V}O_{2peak}$ normalized to fat-free mass was similar between groups ($P > 0.05$).

Preexercise and postexercise brachial and central BP measures. Preexercise and postexercise brachial and central BP measures are provided in Table 3 as absolute values and in Figure 1 as change values. At preexercise, supine resting brachial and central systolic BP and MAP were not different between groups ($P > 0.05$; Table 3). However, adults with obesity exhibited higher resting supine brachial and central

TABLE 1. Descriptive characteristics in young adults without (*n* = 18, normal-weight) and with obesity (*n* = 18, obese).

	Normal-Weight	Obese	*P*
Age, yr	26 ± 5	27 ± 4	0.544
Sex, m/f	11/7	11/7	1.000
Height, cm	169.9 ± 5.7	172.2 ± 7.7	0.325
Weight, kg	65.2 ± 6.9	96.5 ± 12.7	<0.001
BMI, $kg \cdot m^{-2}$	22.6 ± 1.8	32.4 ± 2.2	<0.001
Waist circumference, cm	83.3 ± 5.8	109.4 ± 7.6	<0.001
Body fat, %	29.9 ± 5.7	41.2 ± 5.7	<0.001
Android fat, g	1294 ± 443	3485 ± 725	<0.001
Visceral fat, g	346 ± 150	1015 ± 522	<0.001
Subcutaneous fat, g	1078 ± 353	2471 ± 491	<0.001
Android-to-gynoid ratio	0.95 ± 0.22	1.20 ± 0.18	0.001
Seated brachial SBP, mm Hg	111 ± 6	110 ± 12	0.836
Seated brachial DBP, mm Hg	71 ± 6	74 ± 6	0.101

Data are mean ± SD.
BMI, body mass index; DBP, diastolic BP; m/f, male/female; SBP, systolic BP.

TABLE 2. Blood lipid profile and peak oxygen uptake in young adults without (n = 18, normal-weight) and with obesity (n = 18, obese).

	Normal-Weight	Obese	*P*
Total cholesterol, mg·dL^{-1}	177 ± 40	167 ± 35	0.441
High-density lipoprotein cholesterol, mg·dL^{-1}	59 ± 13	50 ± 15	0.067
Low-density lipoprotein cholesterol, mg·dL^{-1}	103 ± 35	95 ± 32	0.538
Triglycerides, mg·dL^{-1}	94 ± 55	100 ± 52	0.723
Glucose, mg·dL^{-1}	95 ± 10	96 ± 14	0.790
$\dot{V}O_{2peak}$, L·min^{-1}	2.07 ± 0.35	2.54 ± 0.53	0.004
$\dot{V}O_{2peak}$, mL·kg^{-1}·min^{-1}	31.8 ± 4.3	26.2 ± 4.0	<0.001
$\dot{V}O_{2peak}$, mL·FFM-kg^{-1}·min^{-1}	45.2 ± 7.0	44.3 ± 7.2	0.683
Peak work rate, W	163 ± 29	170 ± 33	0.494
Time to exhaustion, s	557 ± 172	646 ± 128	0.092

Data are mean ± SD.
FFM, fat-free mass.

diastolic BPs than did adults without obesity ($P < 0.05$). In the subanalyses (Supplemental Digital Contents 1–2, postexercise recovery responses in men and women, http://links.lww.com/MSS/C186 and http://links.lww.com/MSS/C187), the higher preexercise brachial and central diastolic BPs in the obese group seemed to be driven by men with obesity ($P < 0.05$) and not women with obesity ($P > 0.05$) compared with men and women without obesity. Furthermore, despite similar preexercise MAPs between groups (Table 3), men with obesity ($P < 0.05$), but not women with obesity ($P > 0.05$), exhibited elevated resting brachial and central MAP compared with men and women without obesity (Supplemental Digital Contents 1–2, postexercise recovery responses in men and women, http://links.lww.com/MSS/C186 and http://links.lww.com/MSS/C187).

After exercise, systolic BP and MAP (both brachial and central), expressed as absolute values, decreased similarly in both groups ($P > 0.05$; Table 3). Specifically, both systolic BP (Figs. 1A, D) and MAP (Figs. 1C, F), expressed as change values, were reduced until 60-min postexercise and returned to preexercise values by 90-min postexercise ($P < 0.05$). Diastolic BP (both brachial and central) did not change after exercise when expressed as absolute values ($P > 0.05$; Table 3). However, when expressed as change values, brachial (Fig. 1B) and central (Fig. 1E) diastolic BPs were reduced at 30-min postexercise and returned to preexercise values thereafter ($P < 0.05$). In the subanalyses (Supplemental Digital Contents 1–2, postexercise recovery responses in men and women, http://links.lww.com/MSS/C186 and http://links.lww.com/MSS/C187), only men without and with obesity exhibited reduced systolic BP and MAP (both brachial and central) after exercise ($P < 0.05$), whereas women without and with obesity did not exhibit any reductions ($P > 0.05$).

Preexercise and postexercise vascular measures. Preexercise and postexercise vascular measures are provided in Figures 2A and C as absolute values and in Figures 2B and D as change values. At preexercise, resting leg blood flow (Fig. 2A) or leg vascular conductance (Fig. 2C) was not different between groups ($P > 0.05$). Leg blood flow increased at 30-min postexercise and decreased thereafter, but remained elevated above preexercise values in both groups (Fig. 2A; $P < 0.05$). Similarly, leg vascular conductance increased at 30-min postexercise and decreased thereafter, but remained elevated above preexercise values in both groups (Fig. 2C; $P < 0.05$). When expressed as change values, the increments in leg blood flow (Fig. 2B) and leg vascular conductance (Fig. 2D) were greater overall in adults without obesity than

TABLE 3. BP, wave reflection, and other hemodynamic measures at baseline before (pre) and during postexercise recovery in normal-weight (n = 18) and obese (n = 18) participants.

			Time Postexercise					
	Group	Pre	30-min	60-min	90-min	Time	Group	Interaction
Brachial SBP, mm Hg	Normal-weight	109 ± 10	107 ± 10	108 ± 10	111 ± 10	0.046	0.906	0.544
	Obese	110 ± 10	109 ± 9	108 ± 9	110 ± 11			
Brachial DBP, mm Hg	Normal-weight	65 ± 6	64 ± 7	65 ± 9	66 ± 6	0.100	0.038	0.794
	Obese	69 ± 6*	68 ± 7	69 ± 7	71 ± 6			
Brachial MAP, mm Hg	Normal-weight	79 ± 6	79 ± 8	80 ± 8	81 ± 7	0.046	0.192	0.921
	Obese	83 ± 7	81 ± 7	82 ± 7	84 ± 7			
Central SBP, mm Hg	Normal-weight	94 ± 8	90 ± 8	91 ± 9	93 ± 8	0.001	0.333	0.874
	Obese	97 ± 9	93 ± 7	93 ± 8	95 ± 8			
Central DBP, mm Hg	Normal-weight	66 ± 6	66 ± 8	67 ± 9	67 ± 6	0.097	0.069	0.831
	Obese	70 ± 6*	69 ± 7	70 ± 7	72 ± 6			
Central MAP, mm Hg	Normal-weight	79 ± 6	77 ± 8	78 ± 9	80 ± 7	0.024	0.180	0.958
	Obese	82 ± 7	80 ± 7	81 ± 7	82 ± 7			
AIx, %	Normal-weight	7.7 ± 11.0	–4.3 ± 10.9	–3.9 ± 8.0	–2.1 ± 10.4	<0.001	0.540	0.929
	Obese	9.7 ± 10.5	–1.5 ± 10.8	–1.9 ± 10.8	–0.9 ± 12.9			
AIx@75, %	Normal-weight	1.3 ± 10.2	–2.9 ± 9.8	–4.4 ± 7.6	–3.1 ± 8.6	<0.001	0.735	0.973
	Obese	2.0 ± 11.0	–1.6 ± 11.8	–2.6 ± 12.2	–2.3 ± 14.5			
Augmented pressure, mm Hg	Normal-weight	2 ± 3	–1 ± 3	–1 ± 2	–1 ± 3	<0.001	0.618	0.942
	Obese	3 ± 3	–1 ± 3	–1 ± 3	0 ± 3			
Forward wave pressure, mm Hg	Normal-weight	25 ± 5	24 ± 4	24 ± 4	25 ± 4	0.020	0.064	0.326
	Obese	23 ± 3	22 ± 3	22 ± 4	22 ± 4			
Reflected wave pressure, mm Hg	Normal-weight	42 ± 18	31 ± 14	34 ± 14	35 ± 17	<0.001	0.610	0.507
	Obese	45 ± 15	35 ± 13	35 ± 12	37 ± 14			
Heart rate, bpm	Normal-weight	63 ± 7	80 ± 13	77 ± 12	74 ± 12	<0.001	0.362	0.770
	Obese	59 ± 7	76 ± 11	74 ± 12	73 ± 12			
Cardiac index, L·min^{-1}·m^{-2}	Normal-weight	3.3 ± 0.6	3.3 ± 0.5	3.5 ± 0.8	3.7 ± 0.8	0.012	0.003	0.063
	Obese	2.6 ± 0.5*	3.0 ± 0.7	2.8 ± 0.7	2.9 ± 0.6			
Total peripheral resistance index, mm Hg·(min·L^{-1}·m^{-2})	Normal-weight	26.4 ± 4.6	26.2 ± 3.4	25.9 ± 3.7	26.0 ± 4.0	0.137	0.000	0.141
	Obese	34.9 ± 6.8*	31.0 ± 6.9	33.7 ± 7.4	33.1 ± 6.0			

Data are mean ± SD.
DBP, diastolic BP; SBP, systolic BP.
*Different from normal-weight participants ($P < 0.05$).

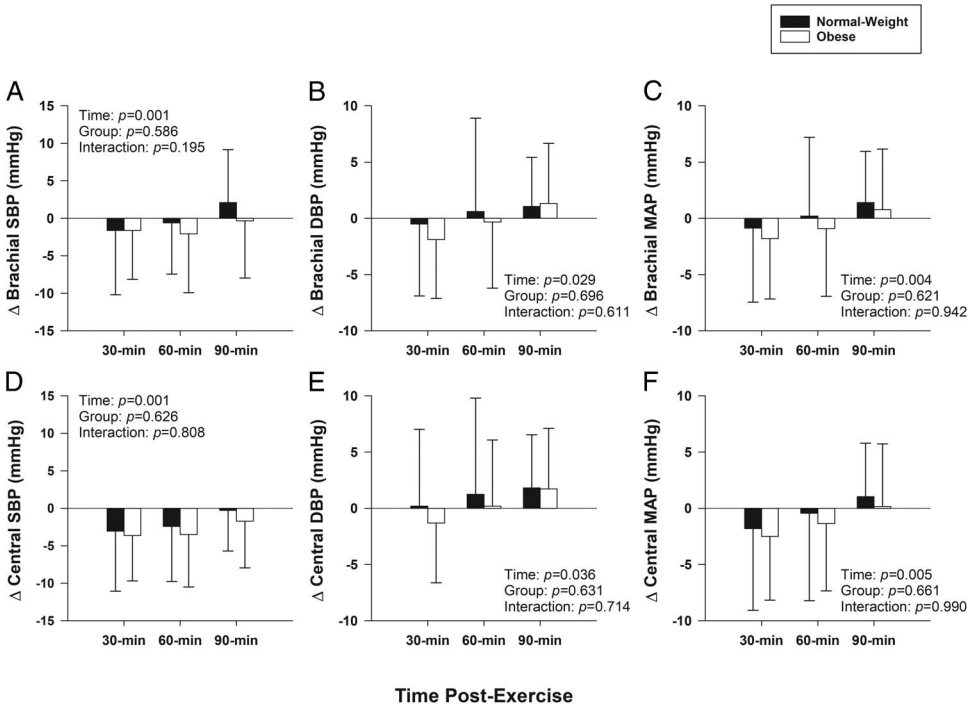

FIGURE 1—Changes in brachial (A–C) and central (D–F) BPs from baseline (pre) during postexercise recovery in young adults without (n = 18, normal-weight) and with obesity (n = 18, obese). DBP, diastolic BP; SBP, systolic BP. Data are mean ± SD.

in adults with obesity ($P < 0.05$), although leg blood flow returned toward preexercise values similarly in both groups ($P < 0.05$). The subanalyses (Supplemental Digital Contents 1–2, postexercise recovery responses in men and women, http://links.lww.com/MSS/C186 and http://links.lww.com/MSS/C187) did not reveal any group differences in leg blood flow and leg vascular conductance after exercise for either men or women ($P > 0.05$).

Preexercise and postexercise wave reflection and other hemodynamic measures. Preexercise and postexercise wave reflection and other hemodynamics are provided in Table 3 as absolute values and in Figure 3 as change values. At preexercise, AIx, AIx@75, augmented pressure, forward wave pressure, reflected wave pressure, and heart rate were not different between groups ($P > 0.05$; Table 3). Modelflow-derived estimated cardiac index and total peripheral resistance index were lower and higher, respectively, in adults with obesity than in adults without obesity ($P < 0.05$). After exercise, AIx, AIx@75, and augmented pressure decreased similarly in both groups when expressed as absolute values ($P < 0.05$; Table 3). When expressed as change values, these decrements in AIx@75 (Fig. 3A) and augmented pressure (Figs. 3B) were of similar magnitude for both groups, which was sustained until 90 min after exercise ($P < 0.05$). The reduction in AIx@75 was also accompanied by decrements in both forward wave and reflected wave pressures after exercise in both groups ($P < 0.05$; Table 3). When expressed as change values, the decrements in forward and reflected wave pressures were sustained until 90 min after exercise in both groups (Figs. 3C, D), although the decrement in reflected wave pressure seemed progressively less from 30 to 90 min after exercise ($P < 0.05$; Fig. 3D). Heart rate remained elevated similarly after exercise in both groups ($P < 0.05$; Table 3). Furthermore, Modelflow-derived estimated cardiac index increased after exercise in both groups ($P < 0.05$; Table 3), with the increase being greater in adults with obesity than in adults without obesity at 30-min postexercise ($P < 0.05$; Fig. 3E). Modelflow-derived estimated total peripheral resistance index did not change after exercise, expressed as both absolute (Table 3) and change values (Fig. 3F; $P > 0.05$).

In the subanalyses (Supplemental Digital Content 1–2, postexercise recovery responses in men and women, http://links.lww.com/MSS/C186 and http://links.lww.com/MSS/C187), both men and women, regardless of obesity status, reduced AIx, AIx@75, and augmented pressure after exercise ($P < 0.05$). Furthermore, men without and with obesity had reductions in both forward and reflected wave pressures after exercise ($P < 0.05$), whereas women without and with obesity had only reduced reflected wave pressure ($P < 0.05$), but not forward wave pressure after exercise ($P > 0.05$). Resting heart rate was slightly lower in men with obesity than in men without obesity ($P < 0.05$); however, no group differences were observed for postexercise heart rate or Modelflow-derived estimated cardiac index and total peripheral resistance index in both men and women ($P > 0.05$).

DISCUSSION

The main findings of this study were twofold. First, young adults with obesity exhibited similar brachial and central PEH compared with young adults without obesity. Second,

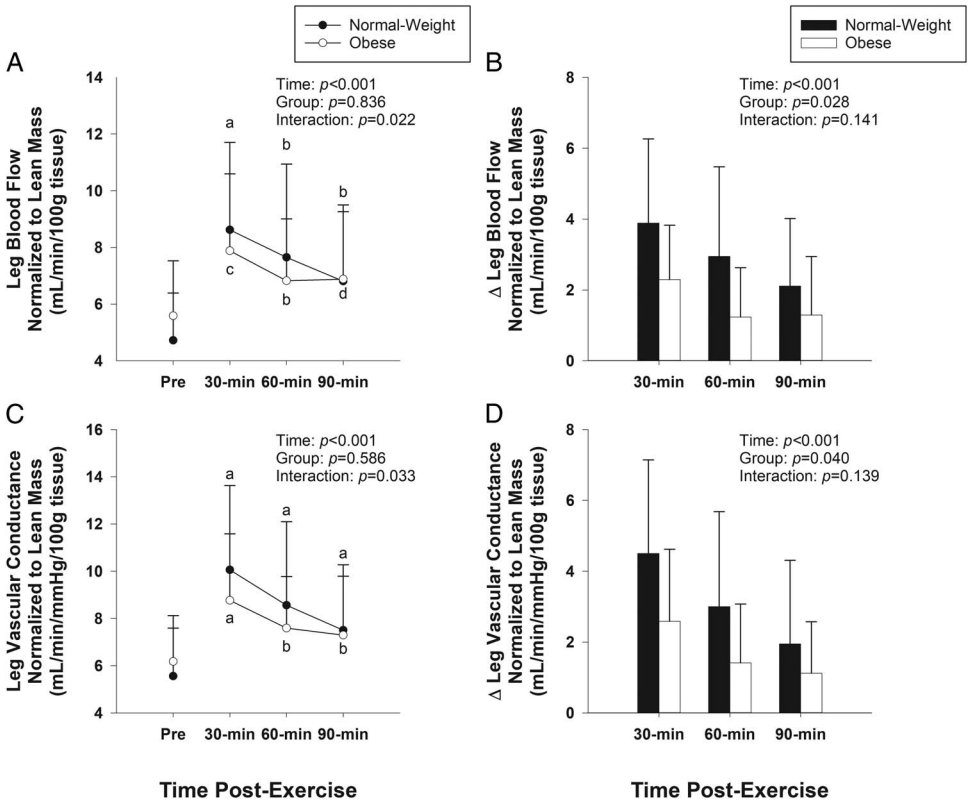

FIGURE 2—Leg blood flow (A and B) and leg vascular conductance (C and D) normalized to lean muscle mass at baseline (pre) and during postexercise recovery in both absolute (A and C) values and change values from pre (B and D) in young adults without (n = 18, normal-weight) and with obesity (n = 18, obese). AU, arbitrary units. Data are mean ± SD. [a]Different from all time points (P < 0.05). [b]Different from pre and 30-min postexercise only (P < 0.05). [c]Different from pre and 60-min postexercise only (P < 0.05). [d]Different from pre only (P < 0.05).

although postexercise peripheral vasodilation, assessed as leg vascular conductance, was present in both young adults without and with obesity, the magnitude of peripheral vasodilation was smaller in adults with obesity. In our subanalyses, brachial and central PEH were observed only in men without and with obesity, whereas women without and with obesity did not exhibit PEH, thus highlighting the potential sex differences in PEH to be explored in future studies. Taken together, despite similar brachial and central PEH in both young adults without and with obesity, the smaller magnitude of postexercise peripheral vasodilation in young adults with obesity compared with young adults without obesity provides new evidence for obesity-induced alterations in the peripheral vasculature after exercise.

Although PEH, defined as reductions in systolic and/or diastolic BP after acute exercise, has been well documented in normotensive and hypertensive populations (9,30), little is known regarding the effectiveness of acute exercise to lower BP in young adults with obesity who are at an increased risk of incident hypertension (4). In the present study, we observed similar decrements in brachial and central PEH in young adults without and with obesity through 60-min postexercise, as evidenced by peak nadirs of −2 mm Hg for brachial systolic BP, −4 mm Hg for central systolic BP, and −2 mm Hg for central MAP for both groups (Table 3, Fig. 1). Our subanalyses demonstrated that men with obesity had a smaller reduction in brachial systolic BP (peak nadir, −5 vs − 8 mm Hg) but a larger reduction in central systolic BP (peak nadir, −6 vs − 3 mm Hg) compared with men without obesity. In contrast, women without and with obesity did not exhibit PEH, which might be attributable to the lower resting brachial and central BP in women (Supplemental Digital Contents 1–2, postexercise recovery responses in men and women, http://links.lww.com/MSS/C186 and http://links.lww.com/MSS/C187). Our findings of similar PEH in young adults without and with obesity are in disagreement with previous work by Zeigler et al. (12) that has identified absent brachial and central PEH in men with obesity compared with men without obesity. In this previous study, both men without and with obesity had elevated resting brachial (126/76 vs 126/78 mm Hg) and central BPs (110/77 vs 113/80 mm Hg), but only men without obesity reduced brachial and central BPs by 3–4 mm Hg after exercise (12). The discrepant findings between our study and those of Zeigler et al. (12) are unclear but may be related to different exercise protocols. In the present study, our participants performed 60 min of leg cycling at 60% of $\dot{V}O_{2peak}$, an exercise protocol that has been consistently demonstrated to produce PEH in healthy adults (22,25,26). Conversely, 40 min of leg cycling at 65%–70% of heart rate maximum was used in the study by Zeigler et al. (12). Whether exercise duration or intensity affects the magnitude of PEH in young adults with obesity remains unclear.

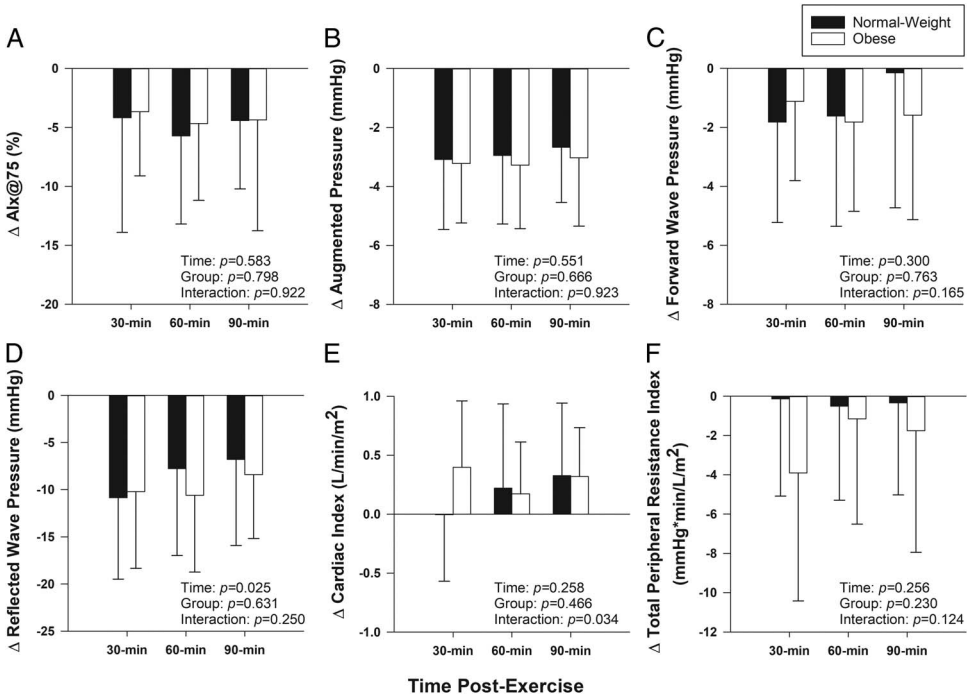

FIGURE 3—Changes in wave reflection measures (A–D) as well as Modelflow-derived estimates of cardiac index (E) and total peripheral resistance index (F) from baseline (pre) during postexercise recovery in young adults without (n = 18, normal-weight) and with obesity (n = 18, obese). Data are mean ± SD.

An important observation in the present study was that the magnitude of sustained postexercise peripheral vasodilation, assessed as leg vascular conductance, was smaller in young adults with obesity compared with young adults without obesity (Table 3, Fig. 2). The potential mechanisms underlying impaired postexercise peripheral vasodilation in young adults with obesity in the present study are unclear. In young adults without obesity, sustained postexercise peripheral vasodilation has been demonstrated to be dependent on the activation of histamine (H_1 and H_2) receptors, such that combined H_1 and H_2 receptor antagonism reduces the magnitude of postexercise peripheral vasodilation by ~80% and PEH by ~65% after 60 min of leg cycling at 60% of $\dot{V}O_{2peak}$ (22,23). Furthermore, alterations in baroreflex control of sympathetic outflow (downward baroreflex resetting) and the transduction of sympathetic activity into vascular resistance after exercise are the neural component that has also been demonstrated to contribute to postexercise peripheral vasodilation and PEH (31). Although no studies to date have investigated the mechanisms of postexercise peripheral vasodilation in human obesity, there is evidence to demonstrate impaired histamine-induced vasodilation in animals with obesity (32), as well as reduced baroreflex sensitivity (33) and elevated sympathetic outflow (34) in humans with obesity. Taken together, extending previous studies (12,13), our findings of attenuated postexercise peripheral vasodilation in young adults with obesity provide new evidence for obesity-induced alterations in the peripheral vasculature after exercise and warrant future investigation into physiologic underpinnings of postexercise peripheral vasodilation in human obesity.

It should be noted that PEH, assessed via brachial BP, is typically characterized by a reduction in total peripheral resistance that is not completely offset by an increase in cardiac output (8,35). In the present study, we also observed reductions in Modelflow-derived estimate of total peripheral resistance index along with slight elevations in Modelflow-derived estimate of cardiac index similarly in both young adults without and with obesity (Table 3; Figs. 3E, F). In contrast to our findings, Zeigler et al. (12) reported no reduction in total peripheral resistance despite slightly elevated cardiac output postexercise in young men with obesity, which might explain the lack of PEH in this previous study.

In contrast to brachial BP, central BP is the pressure to which the heart, kidneys, and major arteries are exposed (36) and is a better predictor of incident hypertension and cardiovascular mortality (17). In the present study, the reductions in central PEH were accompanied by decrements in AIx, AIx@75, and augmented pressure after exercise in both young adults without and with obesity (Table 3; Figs. 3A, B). Because both AIx and augmented pressure are associated with arterial stiffness (28), our findings suggest that central PEH is explained, in part, by acute exercise-induced reduction in arterial stiffness in both groups. Furthermore, although subanalyses demonstrated the presence of central PEH in men and not in women, regardless of obesity status, both men and women exhibited similar reductions in AIx, AIx@75, and augmented pressure after exercise (Supplemental Digital Contents 1–2, postexercise recovery responses in men and women, http://links.lww.com/MSS/C186 and http://links.lww.com/MSS/C187). Notably, AIx is a global marker of wave reflection

and therefore does not supply information on alterations in the magnitude of forward and reflected waves (37). Thus, additional wave separation analyses were undertaken in the present study, to which we observed similar reductions in forward and reflected wave pressures after exercise in both groups and that the magnitude of reduction was greater in reflected than forward wave pressures (Table 3; Figs. 3C, D). In our subanalyses, only men without and with obesity, but not women, had a reduction in forward wave pressure after exercise (Supplemental Digital Content 1–2, postexercise recovery responses in men and women, http://links.lww.com/MSS/C186 and http://links.lww.com/MSS/C187). Nevertheless, because reflected wave pressure is largely governed by small resistance arteries (37), this suggests changes in peripheral/downstream vasomotor tone, as evidenced by reductions in total peripheral resistance, after exercise. Future studies should utilize more direct assessments (i.e., pulmonary artery catheter thermodilution, microneurography) for better understanding of mechanisms underlying brachial and central PEH in obesity.

We acknowledge experimental considerations that may be perceived as limitations to this study. First, we did not directly measure arterial stiffness, assessed via carotid-femoral pulse wave velocity (13), in the present study; however, we assessed AIx and augmented pressure, both of which are related to arterial stiffness (28) and are associated with obesity (38). Second, we did not obtain BP during peak cycling exercise and cannot demonstrate if there is a relationship between peak BP during exercise and the magnitude of PEH; however, evidence suggests that preexercise BP determines the magnitude of PEH (39). Third, interpretation of our findings is limited to postexercise hemodynamic and vascular responses to acute 60-min moderate-intensity cycling exercise, which was chosen owing to its ability to consistently produce PEH and a sustained postexercise peripheral vasodilation in healthy individuals (22,25,26). Future studies are encouraged to explore the impact of obesity on postexercise hemodynamic and vascular responses after various exercise modes, intensities, and durations. Finally, we performed exploratory, subanalyses on sex differences, in which we found PEH in men, but not women, regardless of obesity status, likely owing to higher baseline BP in men, as the magnitude of PEH has been suggested to depend on preexercise BP values (39). Importantly, although the present study was not powered to examine sex differences, our initial observations may set a stage for subsequent studies with larger sample sizes to explore the independent and combined effects of obesity and sex on PEH and to examine potential underlying mechanisms. Given that change in body mass index is independently associated with a greater increase in BP in women compared with men (40), understanding the effect of obesity on PEH in men versus in women is an important area of research for future studies.

CONCLUSIONS

Although obesity did not impair PEH after acute moderate-intensity exercise, young adults with obesity exhibited smaller postexercise peripheral vasodilation compared with young adults without obesity. Collectively, these findings have identified evidence for obesity-induced alterations in the peripheral vasculature after exercise. Future studies should investigate potential mechanistic underpinnings of smaller postexercise peripheral vasodilation in human obesity.

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K. B., P. J. F., P. S. C., B. F., and T. B. conceived and designed the research; K. B., E. C. L., G. G., S. O. W., and M. M. K. performed experiments; K. B., E. S. C., G. G., S. O. W., and M. M. K. analyzed data; K. B. and T. B. drafted the manuscript. All authors edited, revised, and approved the final version of the manuscript.

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REFERENCES

1. Zhou D, Xi B, Zhao M, Wang L, Veeranki SP. Uncontrolled hypertension increases risk of all-cause and cardiovascular disease mortality in US adults: the NHANES III Linked Mortality Study. *Sci Rep*. 2018;8(1):9418.
2. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2018;71(19):e127–248.
3. Muntner P, Carey RM, Gidding S, et al. Potential U.S. population impact of the 2017 ACC/AHA high blood pressure guideline. *J Am Coll Cardiol*. 2018;71(2):109–18.
4. Shihab HM, Meoni LA, Chu AY, et al. Body mass index and risk of incident hypertension over the life course: the Johns Hopkins precursors study. *Circulation*. 2012;126(25):2983–9.
5. Khan SS, Ning H, Wilkins JT, et al. Association of body mass index with lifetime risk of cardiovascular disease and compression of morbidity. *JAMA Cardiol*. 2018;3(4):280–7.
6. Pescatello LS, MacDonald HV, Lamberti L, Johnson BT. Exercise for hypertension: a prescription update integrating existing recommendations with emerging research. *Curr Hypertens Rep*. 2015;17(11):87.
7. Liu S, Goodman J, Nolan R, Lacombe S, Thomas SG. Blood pressure responses to acute and chronic exercise are related in prehypertension. *Med Sci Sports Exerc*. 2012;44(9):1644–52.
8. MacDonald JR. Potential causes, mechanisms, and implications of post exercise hypotension. *J Hum Hypertens*. 2002;16(4):225–36.
9. Halliwill JR, Buck TM, Lacewell AN, Romero SA. Postexercise hypotension and sustained postexercise vasodilatation: what happens after we exercise? *Exp Physiol*. 2013;98(1):7–18.
10. Hecksteden A, Grutters T, Meyer T. Association between postexercise hypotension and long-term training-induced blood pressure reduction: a pilot study. *Clin J Sport Med*. 2013;23(1):58–63.
11. Wegmann M, Hecksteden A, Poppendieck W, et al. Postexercise hypotension as a predictor for long-term training-induced blood pressure reduction: a large-scale randomized controlled trial. *Clin J Sport Med*. 2018;28(6):509–15.
12. Zeigler ZS, Swan PD, Buman MP, Mookadam F, Gaesser GA, Angadi SS. Postexercise hemodynamic responses in lean and obese men. *Med Sci Sports Exerc*. 2018;50(11):2292–300.

13. Bunsawat K, Ranadive SM, Lane-Cordova AD, et al. The effect of acute maximal exercise on postexercise hemodynamics and central arterial stiffness in obese and normal-weight individuals. *Physiol Rep*. 2017;5(7):e13226.
14. Hamer M, Boutcher SH. Impact of moderate overweight and body composition on postexercise hemodynamic responses in healthy men. *J Hum Hypertens*. 2006;20(8):612–7.
15. Shim CY, Yang WI, Park S, et al. Overweight and its association with aortic pressure wave reflection after exercise. *Am J Hypertens*. 2011; 24(10):1136–42.
16. Westerhof BE, Guelen I, Westerhof N, Karemaker JM, Avolio A. Quantification of wave reflection in the human aorta from pressure alone: a proof of principle. *Hypertension*. 2006;48(4):595–601.
17. Pini R, Cavallini MC, Palmieri V, et al. Central but not brachial blood pressure predicts cardiovascular events in an unselected geriatric population: the ICARe Dicomano Study. *J Am Coll Cardiol*. 2008;51(25):2432–9.
18. Senitko AN, Charkoudian N, Halliwill JR. Influence of endurance exercise training status and gender on postexercise hypotension. *J Appl Physiol*. 2002;92(6):2368–74.
19. Lynn BM, McCord JL, Halliwill JR. Effects of the menstrual cycle and sex on postexercise hemodynamics. *Am J Physiol Regul Integr Comp Physiol*. 2007;292(3):R1260–70.
20. Fujita M, Hata A. Sex and age differences in the effect of obesity on incidence of hypertension in the Japanese population: a large historical cohort study. *J Am Soc Hypertens*. 2014;8(1):64–70.
21. Bunsawat K, Grigoriadis G, Schroeder EC, et al. Preserved ability to blunt sympathetically-mediated vasoconstriction in exercising skeletal muscle of young obese humans. *Physiol Rep*. 2019;7(8):e14068.
22. McCord JL, Halliwill JR. H1 and H2 receptors mediate postexercise hyperemia in sedentary and endurance exercise-trained men and women. *J Appl Physiol (1985)*. 2006;101(6):1693–701.
23. McCord JL, Beasley JM, Halliwill JR. H2-receptor-mediated vasodilation contributes to postexercise hypotension. *J Appl Physiol (1985)*. 2006;100(1):67–75.
24. Pellinger TK, Dumke BR, Halliwill JR. Effect of H1- and H2-histamine receptor blockade on postexercise insulin sensitivity. *Physiol Rep*. 2013;1(2):e00033.
25. Lockwood JM, Wilkins BW, Halliwill JR. H1 receptor-mediated vasodilatation contributes to postexercise hypotension. *J Physiol*. 2005; 563(Pt 2):633–42.
26. Lockwood JM, Pricher MP, Wilkins BW, Holowatz LA, Halliwill JR. Postexercise hypotension is not explained by a prostaglandin-dependent peripheral vasodilation. *J Appl Physiol*. 2005;98(2):447–53.
27. Wesseling KH, Jansen JR, Settels JJ, Schreuder JJ. Computation of aortic flow from pressure in humans using a nonlinear, three-element model. *J Appl Physiol*. 1993;74(5):2566–73.
28. Laurent S, Cockcroft J, Van Bortel L, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. *Eur Heart J*. 2006;27(21):2588–605.
29. Thomas KN, van Rij AM, Lucas SJ, Cotter JD. Lower-limb hot-water immersion acutely induces beneficial hemodynamic and cardiovascular responses in peripheral arterial disease and healthy, elderly controls. *Am J Physiol Regul Integr Comp Physiol*. 2017;312(3):R281–91.
30. Brito LC, Queiroz AC, Forjaz CL. Influence of population and exercise protocol characteristics on hemodynamic determinants of post-aerobic exercise hypotension. *Braz J Med Biol Res*. 2014;47(8):626–36.
31. Halliwill JR, Taylor JA, Eckberg DL. Impaired sympathetic vascular regulation in humans after acute dynamic exercise. *J Physiol*. 1996; 495(Pt 1):279–88.
32. Erdei N, Toth A, Pasztor ET, et al. High-fat diet-induced reduction in nitric oxide-dependent arteriolar dilation in rats: role of xanthine oxidase–derived superoxide anion. *Am J Physiol Heart Circ Physiol*. 2006;291(5):H2107–15.
33. Skrapari I, Tentolouris N, Perrea D, Bakoyiannis C, Papazafiropoulou A, Katsilambros N. Baroreflex sensitivity in obesity: relationship with cardiac autonomic nervous system activity. *Obesity*. 2007;15(7):1685–93.
34. Grassi G, Biffi A, Seravalle G, et al. Sympathetic neural overdrive in the obese and overweight state. *Hypertension*. 2019;74(2):349–58.
35. Halliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. *Exerc Sport Sci Rev*. 2001;29(2):65–70.
36. Kollias A, Lagou S, Zeniodi ME, Boubouchairopoulou N, Stergiou GS. Association of central versus brachial blood pressure with target-organ damage: systematic review and meta-analysis. *Hypertension*. 2016;67(1):183–90.
37. Vlachopoulos C, Dima I, Aznaouridis K, et al. Acute systemic inflammation increases arterial stiffness and decreases wave reflections in healthy individuals. *Circulation*. 2005;112(14):2193–200.
38. Pal S, Radavelli-Bagatini S. Association of arterial stiffness with obesity in Australian women: a pilot study. *J Clin Hypertens (Greenwich)*. 2013;15(4):304.
39. Pescatello LS, Kulikowich JM. The aftereffects of dynamic exercise on ambulatory blood pressure. *Med Sci Sports Exerc*. 2001; 33(11):1855–61.
40. Wilsgaard T, Schirmer H, Arnesen E. Impact of body weight on blood pressure with a focus on sex differences: the Tromso Study, 1986–1995. *Arch Intern Med*. 2000;160(18):2847–53.