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Hemodynamic and arterial stiffness differences between African-Americans and Caucasians after maximal exercise

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African-Americans (AA) have a much higher prevalence of hypertension, cardiovascular disease (CVD), and renal disease compared with their Caucasian-American (CA) counterparts (55). Hypertension in AA subjects is characterized by higher incidence, earlier onset, longer duration, higher prevalence, and higher rates of hypertension-related mortality and morbidity (15).

Reduced arterial compliance is associated with hypertension and coronary artery disease. Carotid to femoral pulse wave velocity (PWV) has been regarded as the gold standard method (36) because of its accessibility and reliability (16). It is associated with incident CVD, independently of traditional risk factors in various populations (35). Even in young individuals, arterial compliance is a predictor of CVD events (61), and it is associated with functional and structural characteristics of the vasculature (13).

AA men have higher resting aortic blood pressure (BP) and aortic stiffness compared with age- and fitness-matched CA men (20, 21). An acute bout of maximal aerobic exercise also elicits a differential response in peripheral arterial stiffness in AA men compared with CA men (20). However, these studies used relatively small subject numbers and included only men. The differences in arterial stiffness may be attributed to a heightened BP response in AA subjects during exercise (47, 57) and the absence of postexercise hypotension after exercise in AA subjects but not in CA subjects (41). AA subjects also have elevated central BP despite comparable brachial BP compared with CA subjects (21). However, we are not aware of any study examining central BP after exercise in AA subjects. Furthermore, postexercise BP may have important diagnostic and prognostic implications (22). Postexercise hypotension is also associated long-term adaptations in BP with exercise training (19, 30); thus, the BP response to acute exercise can provide important clinical and physiological information.

Although young women have lower CVD mortality than men, women have higher resting levels of aortic wave reflection compared with men (10, 29) despite having lower pulse pressure and similar or lower arterial stiffness (2). This has been speculated to be due to the shorter stature in women, creating earlier reflection points and thus increasing wave reflection (49). However, the higher aortic wave reflection in women has been shown to be independent of height (4); thus, factors other than shorter stature likely contribute as well. Limited research has shown that the sex difference in aortic wave reflection may be maintained after an acute bout of exercise (3). Interestingly, sympathetic nerve activity affects aortic wave reflection differentially in men and women (10), as
there is a positive association between sympathetic nerve traffic and aortic wave reflection in men but a negative association in women. Thus, considering the large increase in sympathetic nerve activity with exercise, one might expect a differential response in aortic wave reflection between men and women after exercise. Furthermore, enhanced wave reflection is mediated by heart rate (HR) in men but not in women (9), suggesting that exercise may differentially affect wave reflection in women. There is also limited evidence showing that increases in arterial stiffness during exercise are greater in hypertensive women compared with hypertensive men (39). We (46) have shown that endurance-trained men and women exhibit different mechanisms producing postexercise hypotension, with a greater role of total peripheral resistance in women, suggesting sex differences in arterial function after exercise. Nevertheless, little is known regarding the integrated arterial response to maximal exercise in young women or if this response is different compared with young men.

It is also not known whether AA women would have different arterial dynamics at rest and after maximal exercise compared with CA women or if there is a general sex by race interaction for arterial responses after exercise. Limited evidence suggests that AA men (5) and AA women (7) may exhibit differential BP responses to cold pressor tests, and this may be dependent on exercise training status. This suggests there may be a sex by race interaction in BP and possibly arterial function after acute exercise, but this has not been investigated to date.

Therefore, the purpose of this study was to investigate arterial stiffness and both central and peripheral BPs after a maximal aerobic exercise test in young, healthy AA and CA men and women. We hypothesized that both AA and CA subjects would have unchanged central arterial stiffness, whereas CA subjects, but not AA subjects, would exhibit decreased peripheral arterial stiffness after a maximal exercise bout. We further hypothesized that CA subjects, but not AA subjects, would decrease both brachial and aortic BPs after maximal exercise. We also hypothesized that women would have reduced changes in arterial stiffness compared with men.

METHODS

Participants. This study was approved by the Institutional Review Board of the University of Illinois. One hundred young (age range: 18–37 yr), healthy individuals (28 AA men, 24 AA women, 25 CA men, and 23 CA women) volunteered for this study and provided signed informed consent.

All subjects were free of CVD, metabolic, renal, or respiratory disease and were nonsmokers. Subjects did not take any medications, including over-the-counter pain/anti-inflammatory medication. All subjects were in normal sinus rhythm and had no history arrhythmias. Subjects were self-defined as AA or CA if they reported that both parents were of African descent or both parents were of Caucasian descent. All subjects were recruited from the local community or university population.

Study design. All subjects reported to the laboratory for 2 days of testing (i.e., fasting blood draws on day 1 and vascular assessment on day 2). For vascular measures on day 2, all subjects were at least 3 h postprandial and did not exercise or consume caffeine or alcohol for 24 h before being tested. Female subjects were tested for vascular assessment during the early follicular phase of their menstrual cycle. On day 2, subjects rested in the supine position for a period of 10 min in a temperature-controlled room before being tested. The sequence of measures was as follows: brachial artery oscillimetry, arterial tonometry, peak O2 uptake (V\text{O}_2\text{peak}) exercise testing, and recovery cardiovascular measurements at 15 and 30 min after exercise. These measurement periods were based on prior research showing racial differences in peripheral arterial stiffness after maximal exercise in men (20).

Anthropometrics. Height and weight were recorded as previously described (21).

Brachial artery BP assessment. After 5 min of quiet supine rest in a dimly lit room, resting systolic BP (SBP) and diastolic BP (DBP) were measured with an automated oscillometric cuff following established guidelines (31). All BP measurements were repeated, and the average of the two values was recorded and used for analysis. If values differed by ≥5 mmHg, a third measurement was obtained, and the two closest values were averaged.

Wave reflection and aortic BP. Applanation tonimetry was performed using a high-fidelity strain-gauge transducer (Sphygmocor, AtCor Medical, Sydney, NSW, Australia) on the radial artery to obtain pressure waveforms. Aortic SBP, aortic DBP, aortic mean arterial pressure (MAP), augmented pressure, augmentation index (AIx), and normalized AIx at a HR of 75 beats/min were derived from radial pressure waveforms as previously described (20).

Using a generalized validated transfer function (40), a central aortic pressure waveform was reconstructed from the radial artery pressure waveform to obtain aortic SBP and aortic DBP. This transfer function has been validated at rest and during exercise through invasive methods (48). Aortic MAP was derived from integrating the area under the central BP waveform. The reflected wave pressure amplitude (augmented pressure) was defined as the difference between peak SBP and pressure at the inflection point of the aortic waveform. AIx was calculated as the ratio of the amplitude of the pressure wave above its systolic shoulder (i.e., the difference between the early and late systolic peaks of the arterial waveform) to the total pulse pressure (pulse pressure = SBP − DBP). The result was expressed as a percentage and was used as an index of aortic pressure wave-reflection intensity. Because AIx is influenced by HR, AIx values were also normalized to a HR of 75 beats/min as previously described (20).

PWV. PWV was measured following current guidelines (54). Values from the carotid artery to the femoral artery (\text{cPWV}) and from the femoral artery to the superior dorsalis pedis artery (\text{fPWV}) were obtained as previously described (SphygmoCor, AtCor Medical) (20). A single high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX) was used to sequentially obtain pressure waveforms between I) the right common carotid artery and right femoral artery and 2) the right femoral artery and ipsilateral superior dorsalis pedis artery. Consecutive waveforms were captured for a 10-s epoch. Simultaneous ECG gating, as a timing marker, was assessed via a three-lead CM5 configuration and further used to obtain HR. The foot of the pressure wave was identified automatically, removing potential observer bias, using an algorithm that detects the initial upstroke via a line tangent to the initial systolic upstroke point of the pressure tracing and an intersecting horizontal line through the minimum point (12). This algorithm has been shown to be highly reproducible (12). Distances from the suprasternal notch to the femoral artery, carotid artery, and femoral artery sampling site to the superior dorsalis pedis artery were measured as straight lines with a tape measure. The distance from the carotid artery to the sternal notch was then subtracted from the sternal-femoral segment to correct for differences in propagation direction along the arterial path length and taken as a measure of central arterial stiffness. \text{fPWV} values were taken as indexes of peripheral arterial stiffness. \text{cPWV} values were taken as a measure of central/aortic stiffness. Integral software assessed the pulse wave quality (strength of the pulse wave signal, pulse height variation, pulse length variation, and baseline variation) and SD of mean time differences (SphygmoCor, AtCor Medical). This technique has been shown to be highly reproducible (60).
interaction; †

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cycling exercise test to exhaustion. \( \dot{V}O_2 \text{peak} \) was measured during all tests using a breath-by-breath metabolic system (Quark b2, Cosmed, Rome, Italy). Subjects completed a brief warmup consisting of cycling against no resistance for 1 min. The first workload was set at 50 W. Workload was increased by 30 W every 2 min until test termination. Subjects were asked to maintain a preferential cadence of 60–100 rpm. HR was measured using a HR monitor (Polar Electro, Woodbury, NY). Ratings of perceived exertion were also assessed once per stage. After termination of the test, the recovery protocol for leg ergometry consisted of 2 min of light cycling (50 rpm and 0 W) followed by 1 min of quiet sitting on the ergometer. Subjects were then immediately transferred to a table and assumed a supine position for the arterial measurements at 15 and 30 min postexercise.

Fasting blood chemistries. All blood draws were carried out in the morning with subjects in a fasted state for at least 12 h. Fasting glucose was assessed via an oxygen rate method using a Beckman Coulter oxygen electrode (Beckman Coulter, Villepinte, France). Total cholesterol, HDL-cholesterol, and triglycerides were measured using enzymatic techniques. LDL-cholesterol was calculated using the Friedewald formula. VLDL-cholesterol was calculated by dividing triglycerides by 5. White blood cell counts were measured using a quantitative automated hematology analyzer (Sysmex XE-2100, Sysmex, Kobe, Japan).

Renal function assessment. Given known racial differences in renal function, the estimated glomerular filtration rate (eGFR) was estimated from serum creatinine in accordance with recommendations from the Laboratory Working Group of the National Kidney Disease Education Program (34). eGFR was estimated from the Modification of Diet in Renal Disease Study formula (34).

Statistical analysis. All data are presented as means ± SE. Descriptive variables and baseline hemodynamic variables preexercise were analyzed with multivariate ANOVA to test for possible sex, race, and their interaction effects.

To evaluate the effect of maximal exercise in arterial and hemodynamic variables, absolute change values were calculated by subtracting postexercise from preexercise values. Three-way repeated-measures ANOVA was used to test for possible sex, race, time, and their interaction effects. Three-way repeated-measures analysis of covariance (ANCOVA) was used to test for possible sex, race, time, and their interaction effects after controlling for body mass index (BMI) or \( \dot{V}O_2 \text{peak} \). Significant three-way interactions were probed by two-way ANOVAs with repeated measures (race by time, sex by time, and race by sex). We also conducted these probes using ANCOVAs controlling for \( \dot{V}O_2 \text{peak} \) and BMI. Bivariate correlations between changes in arterial stiffness measurements and changes in BPs were assessed using Pearson’s correlation coefficients, respectively. Statistical significance was set at \( P < 0.05 \). SPSS 17.0 (SPSS, Chicago, IL) was used for all analyses.

RESULTS

Subject characteristics and hemodynamic variables are shown in Tables 1 and 2. There were significant interactions between race and sex in eGFR, HR, and BMI at rest (\( P < 0.05 \)). AA subjects had significantly lower weight, higher \( \dot{V}O_2 \text{peak} \), higher weight, and greater BMI and eGFR compared with CA subjects (\( P < 0.05 \)). Women had lower height, weight, \( \dot{V}O_2 \text{peak} \), brachial SBP, AIX, normalized AIX at a HR of 75 beats/min, HR, augmented pressure, cPWV, pulse wave velocity from the femoral artery to the superior dorsalis pedis artery, \( \dot{V}O_2 \text{peak} \), higher HDL-cholesterol than men (\( P < 0.05 \)). Women also had significantly higher HDL-cholesterol than men (\( P < 0.05 \); Table 1). No racial difference or sex by race interaction were found in any blood lipid levels.

Postexercise hemodynamics. Hemodynamic changes at 15 and 30 min after exercise are shown in Figs. 1–4. The change in brachial SBP was greater at 30 min postexercise than at 15

### Table 1. Subject characteristics

<table>
<thead>
<tr>
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<th>Men</th>
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<th>Women</th>
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<tbody>
<tr>
<td></td>
<td>AA</td>
<td>CA</td>
<td>AA</td>
<td>CA</td>
</tr>
<tr>
<td>Age, yr</td>
<td>25 ± 1</td>
<td>25 ± 1</td>
<td>24 ± 1</td>
<td>26 ± 1</td>
</tr>
<tr>
<td>Height, cm†‡</td>
<td>178 ± 1</td>
<td>180 ± 1</td>
<td>162 ± 1</td>
<td>165 ± 1</td>
</tr>
<tr>
<td>Weight, kg†‡</td>
<td>84 ± 3</td>
<td>83 ± 4</td>
<td>76 ± 4</td>
<td>63 ± 4</td>
</tr>
<tr>
<td>Body mass index, kg/m²†‡</td>
<td>26.7 ± 1.1</td>
<td>25.7 ± 1.1</td>
<td>28.9 ± 1.1</td>
<td>23.1 ± 1.2</td>
</tr>
<tr>
<td>Peak ( \dot{V}_O_2 ) uptake, ml/kg·min⁻¹†‡</td>
<td>35.0 ± 1.6</td>
<td>42.1 ± 1.6</td>
<td>27 ± 1.7</td>
<td>35.4 ± 1.8</td>
</tr>
<tr>
<td>eGFR, ml/min⁻¹·1.73 m⁻²†‡</td>
<td>105.3 ± 4.3</td>
<td>95.5 ± 3.7</td>
<td>115.4 ± 3.2</td>
<td>88.1 ± 4.2</td>
</tr>
<tr>
<td>Glucose, mg/dl</td>
<td>90.8 ± 4.3</td>
<td>87.8 ± 3.7</td>
<td>93.1 ± 3.2</td>
<td>83.6 ± 4.2</td>
</tr>
<tr>
<td>White blood cell count, mg/dl</td>
<td>5.9 ± 0.4</td>
<td>6.3 ± 3</td>
<td>5.7 ± 3</td>
<td>5.6 ± 0.4</td>
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<tr>
<td>Triglycerides, mg/dl†‡</td>
<td>96 ± 11</td>
<td>120 ± 9</td>
<td>71 ± 8</td>
<td>77 ± 10</td>
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<tr>
<td>Total cholesterol, mg/dl</td>
<td>160 ± 9</td>
<td>154 ± 7</td>
<td>149 ± 7</td>
<td>147 ± 8</td>
</tr>
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<td>High-density lipoprotein-cholesterol, mg/dl†‡</td>
<td>44 ± 3</td>
<td>46 ± 2</td>
<td>45 ± 2</td>
<td>57 ± 3</td>
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<tr>
<td>Very low-density lipoprotein-cholesterol, mg/dl</td>
<td>19 ± 2</td>
<td>24 ± 2</td>
<td>14 ± 2</td>
<td>15 ± 2</td>
</tr>
<tr>
<td>Low-density lipoprotein-cholesterol, mg/dl</td>
<td>97 ± 8</td>
<td>84 ± 7</td>
<td>90 ± 6</td>
<td>74 ± 8</td>
</tr>
</tbody>
</table>

Values are means ± SE. AA, African-American; CA, Caucasian-American; eGFR, estimated glomerular filtration rate. *\( P < 0.05 \), sex by race significant interaction; †\( P < 0.05 \), significant race differences; ‡\( P < 0.05 \), significant sex differences.

### Table 2. Central and peripheral hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
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<th>Women</th>
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<tbody>
<tr>
<td></td>
<td>AA</td>
<td>CA</td>
<td>AA</td>
<td>CA</td>
</tr>
<tr>
<td>Brachial SBP, mmHg‡</td>
<td>125 ± 2</td>
<td>130 ± 2</td>
<td>115 ± 2</td>
<td>114 ± 2</td>
</tr>
<tr>
<td>Brachial DBP, mmHg</td>
<td>73 ± 2</td>
<td>70 ± 2</td>
<td>72 ± 2</td>
<td>67 ± 2</td>
</tr>
<tr>
<td>Aortic SBP, mmHg</td>
<td>106 ± 2</td>
<td>107 ± 2</td>
<td>101 ± 2</td>
<td>100 ± 2</td>
</tr>
<tr>
<td>Aortic DBP, mmHg</td>
<td>73 ± 2</td>
<td>71 ± 2</td>
<td>73 ± 2</td>
<td>69 ± 2</td>
</tr>
<tr>
<td>Aortic MAP, mmHg</td>
<td>87 ± 2</td>
<td>87 ± 2</td>
<td>87 ± 2</td>
<td>83 ± 2</td>
</tr>
<tr>
<td>HR, beats/min‡‡</td>
<td>60 ± 2</td>
<td>65 ± 2</td>
<td>69 ± 2</td>
<td>62 ± 2</td>
</tr>
<tr>
<td>AIX@75, %‡</td>
<td>−4.8 ± 2.4</td>
<td>−7.6 ± 2.4</td>
<td>9.2 ± 2.5</td>
<td>4.4 ± 2.7</td>
</tr>
<tr>
<td>AIX, %‡</td>
<td>1.9 ± 2.3</td>
<td>−2.8 ± 2.4</td>
<td>12.2 ± 2.5</td>
<td>11.0 ± 2.6</td>
</tr>
<tr>
<td>Augmented pressure, mmHg‡</td>
<td>1 ± 1</td>
<td>−1 ± 1</td>
<td>4 ± 1</td>
<td>3 ± 1</td>
</tr>
<tr>
<td>cPWV, m/s‡</td>
<td>6.3 ± 0.2</td>
<td>6.1 ± 0.2</td>
<td>5.7 ± 0.2</td>
<td>5.5 ± 0.2</td>
</tr>
<tr>
<td>fPWV, m/s‡</td>
<td>8.3 ± 0.3</td>
<td>8.7 ± 0.3</td>
<td>8.3 ± 0.3</td>
<td>8.6 ± 0.3</td>
</tr>
</tbody>
</table>

Values are means ± SE. BP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean arterial pressure; AIX@75, augmentation index (AIX) normalized to a heart rate (HR) of 75 beats/min; cPWV, pulse wave velocity from the carotid artery to the femoral artery; fPWV, pulse wave velocity from the femoral artery to the superior dorsalis pedis artery; *\( P < 0.05 \), sex by race significant interaction; †\( P < 0.05 \), significant race differences; ‡\( P < 0.05 \), significant sex differences.

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min postexercise, but there were no race or sex differences and no significant interactions. Significant time by race by sex interactions were found in brachial DBP, aortic DBP, and aortic MAP \( (P < 0.05) \). The change in both brachial DBP and aortic DBP was significantly greater in CA men at 15 min after exercise compared with 30 min after exercise \( (P < 0.05) \), and it was also significantly different than AA men at 15 min after exercise \( (P < 0.05) \). There was no significant difference in the change in either brachial DBP or aortic DBP between AA and CA women. The change in aortic MAP was greater in CA men at 15 min than at 30 min \( (P < 0.05) \). There was a significant race by time interaction for the change in aortic MAP among men, showing that the change in aortic MAP from 15 to 30 min after exercise was greater in CA men compared with AA men. There was no difference between AA and CA women. However, there was a race effect for the change in aortic SBP after exercise, showing that the change in aortic SBP was greater in both CA men and women compared with AA men and women \( (P < 0.05) \). There were no significant sex effect or interaction effects for aortic SBP.

There was a significant sex effect for the change in AIX, showing that women exhibited a greater change than men \( (P < 0.05) \). There were no significant race effects or interactions. The sex effect was still significant after normalization for HR \( (P < 0.05) \). There were no significant race effects or interactions for normalized AIX at a HR of 75 beats/min. A significant time by sex effect and main effect of time, race, and sex were

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**Fig. 1.**

*Fig. 1. A* change from rest to 15 min (P15) and 30 min (P30) after exercise for brachial systolic blood pressure (bSBP). §The change in bSBP was greater at 30 min compared with 15 min after exercise \( (P < 0.05) \). There were no significant race, sex, or interaction effects. B: change from rest to 15 and 30 min after exercise for brachial diastolic blood pressure (bDBP). There was a significant three-way interaction. *Caucasian-American (CA) men exhibited a different change from African-American (AA) men \( (P < 0.05) \). §The change in bDBP was different at 30 min compared with 15 min after exercise \( (P < 0.05) \) for both AA and CA men. There were no significant differences between AA and CA women and no significant differences between 15 and 30 min after exercise for either AA or CA women. Values are means ± SE.

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**Fig. 2.**

*Fig. 2. A* change from rest to 15 and 30 min after exercise for aortic systolic blood pressure (aoSBP). †There was a significant race difference \( (P < 0.05) \). The change in aoSBP was greater in CA subjects compared with AA subjects. There were no significant time, sex, or interaction effects. B: change from rest to 15 and 30 min after exercise for aortic diastolic blood pressure (aoDBP). *CA men exhibited a different change from AA men \( (P < 0.05) \). §The change in aoDBP was different at 30 min compared with 15 min after exercise \( (P < 0.05) \) for both AA and CA men. There were no significant differences between AA and CA women and no significant differences between 15 and 30 min after exercise for either AA or CA women. C: change from rest to 15 and 30 min after exercise for aortic mean arterial pressure (aoMAP). §There was a significant time by race interaction for men, showing that the change in aoMAP from 15 to 30 min after exercise was greater in CA men compared with AA men. †There was an overall race effect \( (P < 0.05) \) showing a greater change in aoMAP in CA subjects compared with AA subjects. Values are means ± SE.
found in the change in HR. The change in HR in AA men was greater at 15 and 30 min after exercise than AA women (men: 25 ± 2 to 15 ± 2 beats/min; women: 14 ± 2 to 10 ± 2 beats/min). The change in HR was similar in CA men and CA women (men: 26 ± 2 to 18 ± 2 beats/min; women: 23 ± 2 to 16 ± 2 beats/min). CA women also exhibited a greater change in HR compared with AA women ($P < 0.05$). The change in HR was also greater at 15 min compared with 30 min after exercise in all groups ($P < 0.05$). There were no statistically significant effects for the change in cuff PWV or fPWV. Covariance for VO$_2$peak did not alter any of these findings. However, after adjustment for BMI, a significant time by race interaction was found for the change in cPWV ($P < 0.05$). The change in cPWV was significantly different in CA subjects compared with AA subjects at the 30-min time point after exercise (Fig. 4), with cPWV being below baseline in CA subjects and above baseline in AA subjects, making the difference between in the change in cPWV between AA and CA subjects (men and women combined) significant.

The change value of AIx from rest to 15 min after exercise was negatively correlated with the change value of HR ($r = \cdots$).

**Fig. 3.** A: change from rest to 15 and 30 min after exercise for the augmentation index (AIx). ‡There was a significant sex effect ($P < 0.05$), showing a greater change in women than in men. There were no race, time, or interaction effects. B: AIx normalized to a heart rate of 75 beats/min (AIx@75). ‡There was a significant sex effect ($P < 0.05$), showing a differential change in women compared with men. There were no race, time, or interaction effects. C: augmented pressure from preexercise to 15 and 30 min after exercise. There were no significant effects. Values are means ± SE.

**Fig. 4.** A: body mass index-adjusted change from rest to 15 and 30 min after exercise for the pulse wave velocity from the carotid artery to the femoral artery (cPWV). #There was a significant time by race interaction for the change in cPWV ($P < 0.05$). The change in cPWV was significantly different in CA subjects compared with AA subjects at the 30-min time point after exercise, with cPWV being below baseline in CA subjects and above baseline in AA subjects, making the difference between AA and CA (men and women combined) significant. B: body mass index-adjusted change from rest to 15 and 30 min after exercise for the pulse wave velocity from the femoral artery to the superior dorsalis pedis artery (fPWV). There were no significant effects. Values are means ± SE.
DISCUSSION

The main finding of the study was that acute maximal exercise induced differential central arterial stiffness and BP responses during exercise recovery in AA and CA subjects. An acute bout of maximal cycling exercise elicited a small increase in central stiffness in AA subjects but a small decrease in central stiffness in CA subjects, making the change in central stiffness significantly different between AA and CA subjects. Another novel finding was that the change in central BP after maximal exercise was reduced in AA subjects compared with CA subjects but that the change in central stiffness was not associated with the change in central BP. These differences cannot be attributed to resting differences in cardiovascular risk factors, as kidney function was normal, resting BP and blood lipids did not differ between CA and AA subjects, and we statistically controlled for differences in BMI. In addition, women exhibited higher aortic wave reflection and a greater change in AIx after maximal aerobic exercise compared with men.

Racial comparisons. AA subjects have been shown to exhibit cardiovascular hyperreactivity to stress with an exaggerated BP response to behavioral and physiological (7) sympathetic stimuli, as kidney function was normal, and sympathetic stimuli might be implicated in the stiffening of large arteries and thus contribute to the development and progression of hypertension and its complications.

Earlier work from our laboratory (21) has shown that resting central BP is elevated in AA subjects despite comparable brachial BP at rest. Here, we extend our previous work by showing that central SBP after exercise changed differently in AA and CA subjects despite comparable changes in brachial BP. However, we did not note increased central arterial stiffness in AA participants compared with their CA counterparts at rest, nor did we see differential responses of leg peripheral arterial stiffness after maximal aerobic exercise, as in the earlier studies (20, 21). It is possible that the discrepancy was a result of different study subjects since our subjects were all sedentary and both men and women were included in the present study. The AA population in the present study was also more overweight and less fit compared with the CA population, whereas BMI and fitness were similar between AA and CA populations in previous studies. However, since we statistically controlled for fitness and BMI, this should not influence our present findings, but this may explain differences compared with earlier investigations.

The mismatch between the changes in central and peripheral arterial stiffness may have been due to differences in BPs. Changes in central SBP were greater in CA subjects than in AA subjects, whereas the changes in brachial SBP were comparable in both groups. Previous work (20) has shown that changes in peripheral PWV match changes in vascular tone, but these were not necessarily associated with changes in peripheral BP. However, it is possible that central arterial compliance is more dependent on changes in central BP than peripheral muscular arteries since we observed that the change in central BP was in the same direction as the change in cPWV, although these changes were in opposite directions for AA subjects compared with CA subjects. Nevertheless, the change in central BP was not associated with changes in cPWV, suggesting that the change in arterial stiffness was independent of changes in central BP. Peripheral arterial stiffness is likely less dependent on BP because it may be subject to other mediators, possibly local vasoactive substances, such as nitric oxide (38), prostaglandin (14), and endothelin (53).

The AA group in our study was less fit and had higher BMI than the CA group. After adjustment for cardiorespiratory fitness levels, none of the results were altered. When we statistically adjusted for BMI, only cPWV was affected. Thirty minutes after maximal exercise, the change in cPWV was in opposite directions for CA versus AA subjects, and the difference in this change was significant. Therefore, central stiffness, but none of the other measured variables, was affected by BMI, but not fitness in our study. Differential arterial stiffness responses in AA and CA subjects may be affected by structural factors such as the composition of the arterial wall, including the contents of the extracellular matrix (37). Arteries become wider and less elastic with a reduction in arterial elastin and an increase in collagen content (25). The wall thickness of large arteries correlates well with the burden of generalized atherosclerosis and is a reliable predictor of coronary events (44). AA subjects had greater mean aortic wall thickness than CA subjects and higher age-related mean maximal aortic wall thickness (28, 45). In addition, AA subjects tend to have higher relative collagen content in the aorta than CA subjects between 30 and 69 yr of age (32). AA subjects also have a tendency to overgrow connective tissue in response to diseases (42). Although we did not measure arterial wall composition in our study, the higher content of collagen in the aortic arterial wall in AA subjects may affect elastic properties of the large arteries after exercise. It should be noted the studies revealing structural differences between AA and CA groups were population-based studies with a wide age range from teenagers to older individuals. The fact that we did not observe differences in central stiffness between AA and CA subjects at rest confirmed the use of acute exercise as physiological perturbation to evoke racial difference in elastic properties in the central arteries in young individuals free of overt CVD.

Sex comparisons. In agreement with previous studies (10, 29), women demonstrated higher baseline levels of aortic wave reflection compared with men. When normalized for HR, AIx remained higher in women. Augmented pressure was also increased in women. The majority of wave reflection as seen by the heart is attributable to the branching of the aorta at the celiac trunk and renal arteries and its terminal tapering (33). These sex differences have been attributed to women with shorter body height and an associated shorter aorta, which, in turn, results in earlier wave reflections (49). Even in height-matched elderly hypertensive subjects, women had earlier
During the early follicular phase of the menstrual cycle, which
function with aging (56). Our female subjects were all tested
(FSH) is highly correlated with arterial mechanical properties
differential response in women. Follicle-stimulating hormone
waves compared with men.
mediated vasodilatation in resistance arteries in women during
reduction in aortic AIx during high-intensity exercise may be due to differential levels
in modulating aortic waveforms in men and women, as positive
Muscle sympathetic nerve activity may play an important role
controlled for HR (normalized AIx at a HR of 75 beats/min).
However, the difference in HR between men and women is
unlikely to explain the sex difference in AIx, since there was
considering the large increase in sympathetic
consider the sex difference in AIx, since there was
Muscle sympathetic nerve activity may play an important role
in modulating aortic waveforms in men and women, as positive
correlations have been found between sympathetic activity and
in men but a negative association was found in
women (10). Considering the large increase in sympathetic
nerve activity with exercise, it may not be surprising to find
derential changes in aortic wave reflection during recovery.
However, the mechanism underlying the inverse relationship
remains unknown; therefore, we can only speculate the potent-
tial mechanisms. An increased sensitivity to β-adrenergic re-
ceptor stimulation in women may be partly responsible for the
observed differential responses (24). Greater β-adrenoceptor-
mediated vasodilatation in resistance arteries in women during
recovery from exercise may contribute to differences in central
wave patterns compared with men.

Female sex hormones may also be responsible for this
differential response in women. Follicle-stimulating hormone
(FSH) is highly correlated with arterial mechanical properties
and may be the most sensitive marker of declining arterial
function with aging (56). Our female subjects were all tested
during the early follicular phase of the menstrual cycle, which
is characterized by a rise in FSH during the first days of the
cycle. It is possible that the differences between men and
women were increased due to FSH. Finally, estrogen has direct
vasodilation effects by stimulating endothelial nitric oxide
synthase activity via activation of estrogen receptor-α (11, 18)
and potentially decreases wave reflection. However, the con-
tribution of estrogen is probably limited because the surge of
estrogen and the associated decline in Alx occurs only during
late follicular phase of the menstruation cycle (1). Future
research is warranted to examine the hormone levels and other
potential mechanisms responsible for the observed differential
responses in men and women.

Strength and limitations. The strength of the present study is
a relatively large sample size. In addition, women were tested
during the early follicular phase of their menstrual cycle to
minimize the effect of female hormones. The design of the
study also allowed us to examine baseline differences in our
four groups as well as cardiovascular responses after an acute
bout of exercise. Standard maximal aerobic exercise was used
as a stressor to elicit cardiovascular responses that may not be
evident at rest.

We did not control for socioeconomic status; however, we
recruited most of our subjects from university students, and,
therefore, they had a similar level of education. Furthermore,
this is a cross-sectional study, and cause-effect cannot be
inferred.

We did not directly measure aortic stiffness or central wave
reflection but used noninvasive validated and well-accepted
techniques. cPWV is an accepted standard measure of arterial
stiffness and has been used in most large-scale studies. Also,
central wave reflection, although derived from a transfer func-
tion, is valid both at rest and after exercise, as shown by
validation compared with invasive techniques (48). Further-
more, although we screened subjects based on health history,
we do not know if any of the subjects had latent coronary artery
disease, diastolic dysfunction, or left ventricular hypertrophy,
which may have affected our results. However, given the
young age and general health status of our subject population,
it is unlikely that these factors exhibited a significant influence
on our results. Also, none of the subjects experienced any signs
or symptoms of coronary artery disease during the maximal
e xercise test.

Conclusions. Young AA and CA subjects exhibited differ-
tential responses in central stiffness and central BP after acute
maximal exercise. Premenopausal women had greater Alx and
augmented pressure at rest and exhibited greater reduction in
aortic wave reflection after maximal aerobic exercise.

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P.S. performed experiments; H.Y. and P.-T.W. analyzed data; H.Y., K.S.H.,
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conception and design of research.


