Racial differences in central blood pressure and vascular function in young men

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Heffernan KS, Jae SY, Wilund KR, Woods JA, Fernhall B. Racial differences in central blood pressure and vascular function in young men. Am J Physiol Heart Circ Physiol 295: H2380–H2387, 2008. First published October 10, 2008; doi:10.1152/ajpheart.00902.2008.—Young African-American men have altered macrovascular and microvascular function. In this cross-sectional study, we tested the hypothesis that vascular dysfunction in young African-American men would contribute to greater central blood pressure (BP) compared with young white men. Fifty-five young (23 yr), healthy men (25 African-American and 30 white) underwent measures of vascular structure and function, including carotid artery intima-media thickness (IMT) and carotid artery β-stiffness via ultrasonography, aortic pulse wave velocity, aortic augmentation index (AIx), and wave reflection travel time (Tr) via radial artery tonometry and a generalized transfer function, and microvascular vasodilatory capacity of forearm resistance arteries with strain-gauge plethysmography. African-American men had similar brachial systolic BP (SBP) but greater aortic SBP (P < 0.05) and carotid SBP (P < 0.05). African-American men also had greater carotid IMT, greater carotid β-stiffness, greater aortic stiffness and AIx, reduced aortic Tr and reduced peak hyperemic, and total hyperemic forearm blood flow compared with white men (P < 0.05). In conclusion, young African-American men have greater central BP, despite comparable brachial BP, compared with young white men. Diffuse macrovascular and microvascular dysfunction manifesting as carotid hypertrophy, increased stiffness of central elastic arteries, heightened resistance artery constriction/blunted resistance artery dilation, and greater arterial wave reflection are present at a young age in apparently healthy African-American men, and conventional brachial BP measurement does not reflect this vascular burden.

AFRICAN-AMERICAN MEN HAVE a greater prevalence of hypertension compared with their white American counterparts (23, 27). African-American men develop high blood pressure (BP) sooner, and this may manifest as early as their second decade of life (23). Measurement of central BP has been suggested to hold greater prognostic capability than conventional brachial BP (40). In end-stage renal disease, carotid BP is a stronger predictor of all-cause mortality than brachial BP (65). Recent findings from the Strong Heart Study have confirmed that central BP is more strongly related to vascular hypertrophy, extent of atherosclerosis, and cardiovascular events than brachial BP (63). Moreover, central BP is a better predictor of coronary artery disease severity and adverse cardiovascular events than brachial pressure (14, 70). Increases in carotid BP directly contribute to increases in carotid intima-media thickness (IMT) (69), a reflection of subclinical vascular target-organ damage and an independent risk factor for cardiovascular disease (34, 55). At present, no study has examined central BP in young African-American males.

One consequence and/or potential mechanism of hypertension is detrimental alterations in vascular structure and function. Arterial compliance reflects the ability of an artery to expand and recoil in response to cardiac systole and diastole, allowing blood flow to be converted from an intermittent, pulsatile flow to a more steady and laminar flow. Increases in arterial stiffness and loss of this dampening effect may cause microvascular damage (49, 57) and hasten the atherosclerotic process, regardless of age (38, 58). Consequently, increased arterial stiffness (41, 47), reduced microvascular reactivity (32), and increased subclinical atherosclerosis (34) have been identified as risk factors for future cardiovascular events, as well as the development of hypertension (47).

African-Americans have diffuse macrovascular and microvascular dysfunction manifesting as increased stiffness (reduced compliance) of elastic central arteries, such as the carotid artery and aorta (19, 21, 30), heightened resistance artery constriction, and blunted resistance artery dilation (67). The greater vascular stiffness and microvascular dysfunction seen in African-Americans are directly related to target organ damage (12, 68). Carotid IMT is greater in African-Americans compared with white Americans (16, 43). Although vascular dysfunction is considered a function of the aging process, “premature arterial senility” (56) has been reported in normotensive African-American men as young as 21 yr of age (30, 75).

Central BP is influenced by arterial stiffness and microvascular function. Given the known vascular dysfunction in young African-American men, it is possible that central BP may be greater in African-American men, and changes in central BP may precede changes in peripheral (i.e., brachial) BP in this cohort. Therefore, the primary purpose of this study was to examine racial differences in central BP in young African-American and white men. A secondary purpose was to examine macrovascular and microvascular variables related to central hemodynamics. Several methods were employed in an attempt to provide a comprehensive view of racial differences in vascular structure and function as it relates to central and peripheral hemodynamics.
METHODS

Subjects. Fifty-five young, healthy men (25 African-American and 30 white) volunteered for this study. All subjects were free of cardiovascular, metabolic, renal, or respiratory disease, and none smoked. Subjects did not take medication of any kind, including over-the-counter pain/anti-inflammatory medication. Subjects were self-defined as African-American, if reporting that both parents were of African descent. All subjects were recruited from the local university student population. All subjects gave written consent. This study was approved by the Institutional Review Board of the University of Illinois at Urbana-Champaign.

Study design. All subjects reported to the laboratory for 2 days of testing (day 1, fasting blood and body composition assessment; day 2, vascular assessment). For vascular measures, all subjects were at least 3 h postprandial and did not consume caffeine or alcohol or exercise for 24 h before testing. Participants rested in the supine position for a period of 10 min in a temperature-controlled room before testing. The sequence of measures was as follows: brachial artery oscillometry, arterial tonometry, ultrasound imaging/Doppler measures, forearm strain-gauge plethysmography, and exercise testing.

A subset of subjects (n = 38) reported back to the laboratory for a third visit for vascular repeatability assessment. Vascular measures were carried out at the same time of day to reduce influence of diurnal variation. Values are reported below in appropriate sections.

Macrovascular structure and function. The IMT of the common carotid artery was defined as the distance between the leading edge of the lumen-intima interface to the leading edge of the media- Adventitia interface of the far wall of the carotid artery. All measurements were made at end diastole. The IMT of the common carotid artery was determined from an average of five measurements of a 10-mm segment (separated by 2-mm intervals) obtained 2 cm proximal to the carotid bifurcation. The intraclass correlation coefficient (ICC) for carotid artery IMT in our laboratory is 0.95. The ICC for carotid IMT in a subset of subjects from this investigation, collected on 2 separate days, was 0.94.

Carotid artery diameter was measured by ultrasonography (SSD-5500, Aloka, Tokyo, Japan). The cephalic portion of carotid artery was imaged in a longitudinal section, 1–2 cm proximal to the bifurcation, using a high-frequency (7.5 MHz) linear array probe. Carotid pressure was measured with applation tonometry (see below). Heart rate (HR) was derived from ECG with a single-lead CM5 configuration. β-Stiffness index (β) was calculated as a means of adjusting arterial compliance for changes in distending pressure as follows:

\[
\beta = \frac{\log P_p/P_s}{(D_i - D_o)/D_o}
\]

where \(D_i\) and \(D_o\) are the maximum (systolic) and minimum (diastolic) diameters, and \(P_1\) and \(P_0\) are the highest (systolic) and lowest (diastolic) carotid pressures, respectively. All aforementioned procedures were repeated using the brachial artery (imaged longitudinally ~3 cm proximal to the olecranon process) to assess regional peripheral muscular artery stiffness. In our laboratory, the ICC attained on 2 separate days is > 0.9. The ICC for carotid stiffness in a subset of subjects from this investigation, collected on 2 separate days, was 0.89.

Simultaneous brachial diameter and blood velocity were measured with a Doppler ultrasound technique. The angle of insonation was maintained at < 60°. The area beneath a minimum of five velocity curves was traced offline to obtain a single mean velocity-time integral (VTI). A mean brachial diameter was calculated as 1/3 \(d_{\text{maximum}} + 2/3 d_{\text{minimum}}\), where maximum and minimum diameters correspond to systolic and diastolic diameters, respectively. Mean brachial blood flow was calculated as mean VTI × (π × radius²) × HR. The pulsatility index was calculated as (\(V_s − V_d\))/mean \(V_s\), where \(V_s\) is the systolic velocity, \(V_d\) diastolic velocity, and mean \(V\) is the mean velocity.

The aortic pulse wave velocity (PWV) was calculated from the right carotid artery pressure waveform and the right femoral artery pressure waveform, as previously described (28). Brachial PWV was calculated from the right carotid artery pressure waveform and the right radial artery pressure waveform. This technique has previously been shown to be highly reproducible. In our laboratory, test-retest repeatability for resting PWV is high (28) and, when calculated on a subset of subjects for this investigation, was 0.92.

Hemodynamics. Brachial BP was measured in the supine position using an automated oscillometric cuff. All brachial BP measurements were made in duplicate, following established guidelines (61). If these values deviated by > 5 mmHg, a third measurement was conducted. The average of the two closest values was recorded and used for subsequent analysis. Reliability of BP measures in our laboratory is high (29).

Carotid artery pressure waveforms were attained using applation tonometry (Millar Instruments, Houston, TX) and calibrated against brachial mean arterial and diastolic pressure. This technique has been shown to record a pressure wave with harmonic content that does not differ from that of an intra-arterially recorded wave (37). Tonometer hold-down pressure was kept constant throughout the measurement using integral software (SphygmoCor, AtCor Medical, Sydney, Australia).

Radial artery pressure waveforms were attained in the supine position from a 10-s epoch using applation tonometry and a high-fidelity, strain-gauge transducer (Millar Instruments, Houston, TX). Using a generalized validated transfer function (60), a central aortic pressure waveform was reconstructed from the aforementioned radial artery pressure waveform (SphygmoCor, AtCor Medical) (13). Augmented pressure (AP), a measure of the contribution of wave reflections to systolic BP (SBP), was defined as the difference between central SBP and the pressure at the forward wave peak (P1). Augmentation index (Alx) provides a measure of the contribution of wave reflection pressure (i.e., AP) to SBP relative to total pulse pressure (PP). It was calculated as the ratio of 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 PP expressed as a percentage (P2 – P1/PP * 100). Because Alx is influenced by HR, Alx values were also normalized to a HR of 75 beats/min (73). Travel time of the forward pressure wave from the aorta to the peripheral reflection site and back (Tr) was determined as the time from the initial upstroke of the pressure wave to the foot of the reflection wave. PP amplification was calculated as the ratio of brachial PP to central PP (20, 31). PP amplification devoid of the influence of wave reflection (nonaugmented PP amplification) was calculated as the ratio of peripheral PP to non-AP [i.e., aortic P1 to aortic diastolic BP (DBP)] (74). All measurements were made in duplicate, and the mean value was used for subsequent analysis.

Reproducibility of measures attained from this technique has previously been shown to be high (72). In our laboratory, the ICC attained on 2 separate days is > 0.9 (28). The ICC for all variables derived from the radial pulse contour in a subset of subjects from this investigation, collected on 2 separate days, was > 0.85.

Beat-to-beat BP was recorded for a 15-min epoch using finger plethysmography (Finometer, FMS). The Modelflow method was used to derive stroke volume and cardiac output, as previously described and validated (9, 26, 35, 71).

Macrovascular function. Metabolic vasodilatory capacity of forearm resistance arteries was assessed using reactive hyperemia and strain-gauge plethysmography (EC-6, DE Hokonson, Bellevue, WA). With the subject in the supine position, and the arm supported above heart level, a mercury-in-Silastic strain gauge was placed around the forearm at the area of greatest circumference. A pediatric BP cuff was placed around the wrist. A rapidly inflating venous occlusion cuff was placed around the upper arm. After circulation was arrested in the
hand (cuff inflated to 200 mmHg), forearm blood flow (FBF) was measured by inflating the venous occlusion cuff to 40 mmHg for 7 s followed by 8-s rapid deflation. The average of five stable measures was used for analysis.

A second BP cuff was placed over the venous occlusion cuff on the upper arm and inflated to a pressure of 250 mmHg for 5 min. Thirty seconds before release of the upper arm cuff, the wrist cuff was inflated to a pressure of 200 mmHg, occluding circulation in the hand. After 5 min of occlusion, the BP cuff in the upper arm was released. This was followed immediately by rapid inflation of the venous occlusion cuff to 40 mmHg for 7 s followed by 8-s deflation. These 15-s inflation-deflation cycles were repeated for 3 min following reactive hyperemia and used to assess change in forearm volume as an index of microvascular vasodilatory capacity. Data are presented as peak hyperemic FBF and total FBF. Total FBF was calculated as area under the curve of the 3-min hyperemic FBF response using the trapezoidal integration method. The ICC for peak FBF following relative humidity in a subset of subjects from this investigation, collected on 2 separate days, was 0.90.

Maximal aerobic capacity. Cardiorespiratory fitness may also impact racial differences in vascular function (4). Therefore, peak oxygen consumption was assessed using a graded cycle ergometry protocol until volitional fatigue. Expired gases were analyzed using a Quark b2 breath-by-breath metabolic system (Cosmed, Rome, Italy).

Anthropometrics. Body composition was determined using whole body air displacement plethysmography (Bod Pod, Life Measurement, Concord, CA). This technique has been shown valid in both African-American and white populations (15). Height and weight were measured using a stadiometer (to the nearest 0.5 cm) and a beam balance platform scale, respectively. Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared.

Fasting blood chemistries. All blood draws were carried out first thing in the morning with subjects in a fasted state. Fasting glucose was assessed via an oxygen rate method using a Beckman Coulter oxygen electrode (Beckman Coulter, Villepointhe, France). Total cholesterol, HDL cholesterol, and triglycerides were measured using enzymatic techniques. LDL was calculated using the Friedewald formula. White blood cell count was measured using a quantitative automated hematology analyzer (Sysmex XE-2100, Sysmex Corp, Kobe, Japan).

Renal function assessment. Given known racial differences in renal function, glomerular filtration rate (GFR) was estimated from serum creatinine (sCR) measurements in accordance with recommendations from the Laboratory Working Group of the National Kidney Disease Education Program (50). GFR was estimated from the Modification of Diet in Renal Disease Study formula as follows: GFR [ml/min \((1.73 \, \text{m}^2)\] = 186 \times (sCR)^{-1.154} \times (\text{age}^{-0.203}) \times (1.210 \text{ if African-American}) (50).

Statistical analysis. All data are reported as means \(\pm\) SE. A priori significance was set at \(P < 0.05\). Normality of distribution was assessed using Kolmogorov-Smirnov and Shapiro-Wilk tests. If datum was not normally distributed, outcome measures were logtransformed (natural log function) to meet assumptions for parametric statistical analysis. Analysis of variance was used to assess differences in continuous outcome variables. Analysis of covariance was used to assess differences in continuous outcome variables after adjusting for potential confounders. Stepwise regression analysis was performed to examine predictors of central PP in our cohort. The dependent variables in two separate models were carotid PP and aortic PP. Independent variables were carotid \(\beta\)-stiffness, AP, reflection time, PWV, and FBF following reactive hyperemia. Although AIX is the most widely used measure of wave reflection, AIX is a composite of AP and central PP and could not be entered into the model due to issues of collinearity. \(x^2\) Tests were used to compare categorical variables (family history of hypertension, family history of diabetes). Pearson’s correlation coefficients were used to assess relationships between variables of interest. Data analysis was carried out using Statistical Package for the Social Sciences (SPSS, version 12.0.1, SPSS, Chicago, IL).

RESULTS

African-American subjects were slightly younger than white subjects (Table 1, \(P < 0.05\)). There were no group differences in height, weight, BMI, body fat, body lipids, glucose white blood cell count, GFR, peak oxygen uptake, or family history of diabetes/hypertension (Table 1).

There were no differences in stroke volume and cardiac output between African-American men and white men (Table 2, \(P > 0.05\)). HR was significantly lower in African-American compared with white men (56 \(\pm\) 2 vs. 61 \(\pm\) 2 beats/min, \(P = 0.022\)). There were no group differences in brachial SBP, brachial DBP, brachial PP, or brachial mean arterial pressure (Table 2). African-American men had significantly greater carotid SBP and aortic SBP (Table 2, \(P < 0.05\)). When covarying body fat, cardiorespiratory fitness, and HR, adjusted means were still significantly different between African-American and white men for aortic-brachial PP amplification (1.60 \(\pm\) 0.02 vs. 1.69 \(\pm\) 0.02, \(P = 0.009\)) and carotid-brachial PP amplification (1.08 \(\pm\) 0.04 vs. 1.20 \(\pm\) 0.03, \(P = 0.031\)). There were no group differences in carotid DBP, aortic DBP, aortic PP, systolic ejection duration, \(P_1\), or nonaugmented PP amplification (Tables 2 and 3).

African-American men had significantly greater AIX, AIX normalized to HR of 75 beats/min, and AP (Table 3, \(P < 0.05\)). When covarying for body fat and cardiorespiratory fitness, group differences in these variables remained significant. African-American men had faster arterial wave reflection time (Table 3, \(P < 0.05\)), and covarying for body fat and cardiorespiratory fitness did not alter group differences (adjusted means: 162 \(\pm\) 0.05). African-American men also had higher PWV (Table 3, \(P < 0.05\)), and covarying for body fat and cardiorespiratory fitness did not alter

<table>
<thead>
<tr>
<th>Table 1. Subject characteristics</th>
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<tbody>
<tr>
<td><strong>Variable</strong></td>
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<tr>
<td>Age, yr</td>
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<tr>
<td>Height, cm</td>
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<tr>
<td>Weight, kg</td>
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<td>Body mass index, kg/m²</td>
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<td>Body fat, %</td>
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<tr>
<td>Total cholesterol, mg/dl</td>
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<tr>
<td>HDL cholesterol, mg/dl</td>
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<tr>
<td>LDL cholesterol, mg/dl</td>
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<tr>
<td>Triglyceride, mg/dl</td>
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<tr>
<td>Glucose, mg/dl</td>
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<tr>
<td>WBC count, mg/dl</td>
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<tr>
<td>GFR, ml/min (\cdot)1.73 m²</td>
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<tr>
<td>Peak oxygen uptake, ml/kg min⁻¹</td>
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<tr>
<td>Family history hypertension, n/%</td>
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<tr>
<td>Family history diabetes, n/%</td>
</tr>
</tbody>
</table>

Values are means \(\pm\) SE; \(n\), no. of people. HDL, high density lipoprotein; LDL, low density lipoprotein; WBC, white blood cell; GFR, glomerular filtration rate.
Similarly, when examining predictors of carotid PP, only white men (Fig. 1, 66.8 vs. 5.3 ms, 95% confidence interval = 3.2–8.2, P = 0.033).

**DISCUSSION**

The novel findings of the present study were as follows. Compared with young white men of similar BMI, body fat, cardiorespiratory fitness, renal function, blood lipid, and glucose levels, young African-American men have 1) greater central BP, yet comparable brachial BP compared with young white men; and 2) greater augmentation of central BP from wave reflections. Moreover, this study confirmed the presence of diffuse macrovascular and microvascular dysfunction manifesting as increased central artery stiffness and reduced peripheral endothelial function in young African-American men, and this likely contributed to noted racial differences in central BP.

To our knowledge, this is the first study to examine racial differences in central BP. No differences in brachial BP were noted in young African-American men, despite greater central BP, and this is novel. Central BP holds greater prognostic value than conventional brachial BP as central pressure more aptly reflects the load encountered by the heart (i.e., ventricular-vascular coupling) (46). Thus brachial BP may neglect impor-
tient information on cardiovascular burden and response to therapy in African-American men (2, 24).

We noted greater carotid IMT in young African-American compared with the white men, and this is consistent with findings reported in older adults (6, 8, 16, 25, 43, 44). Increased carotid IMT is a risk factor for future cardiovascular events (34). Increases in carotid IMT are due to a combination of intimal hyperplasia/diffuse atherosclerosis and medial hypertrophy in response to local pulsatile mechanical load (69). Sustained elevations in local distending pressure directly stimulate smooth muscle hypertrophy and vascular wall extracellular matrix turnover (11, 69). This process occurs in the carotid but not brachial arteries (11). Interestingly, when local carotid distending pressure is reduced by pharmacological means, there are reductions in carotid wall hypertrophy (10).

Similar to previous reports (69), we noted an association between carotid SBP and carotid IMT ($r = 0.3, P < 0.05$). Thus it is possible that the greater carotid SBP in African-American men contributes to advanced IMT of the carotid artery. Our results would further suggest that this process may occur early in life, contributing to target organ damage in young, otherwise apparently healthy, African-American men.

The contour of the central BP waveform reflects the contribution of incident and reflected pressure waves. Each cardiac cycle generates an outgoing pressure wave that traverses the aorta. These pressure waves arrive at areas of impedance mismatch in the periphery and are reflected back to the left ventricle, summatting with the forward pressure wave (51). Based on distance to the effective reflecting site, reflection time, and/or amplitude, the reflected pressure wave may augment either systolic (i.e., early arrival) or diastolic (i.e., late arrival) pressure (51). Wave reflection can be influenced by several factors, including central artery stiffness, peripheral artery stiffness, microvascular function, arteriolar/vasomotor tone, height, and HR (51). Increased magnitude of wave reflection is independently associated with several negative cardiovascular health outcomes, including atherosclerotic burden (54, 62).

Consistent with previous reports, we noted greater carotid and aortic vascular stiffness (the inverse of compliance) in African-American compared with white men, despite comparable mean arterial pressure (19, 21). Thus the higher PWV in this cohort appears to be due to intrinsically higher aortic stiffness and is not due to modulation of the vascular pressure-volume curve. African-American men also had greater wave reflection magnitude and faster Tr of the reflected pressure wave. To our knowledge, this is the first study to note greater AIx in young African-American men. It would appear that wave reflections contributed marginally to central PP augmentation in young African-American men. Although racial differences in AP were noted, central aortic pressure was only augmented 1 mmHg from wave reflections in young African-American men. Arterial stiffness is considered an antecedent for systolic hypertension (5, 42), and it is likely that increased central artery stiffness contributes more to elevated central BP in younger African-American men than wave reflection. According to findings from regression analysis, carotid and aortic stiffness were better predictors of carotid PP and aortic PP, respectively, than measures of wave reflection timing and magnitude or resistance artery vasodilatory capacity.

PP varies throughout the arterial tree, increasing from central to peripheral vessels, owing to differences in vascular stiffness, diameter, and wave reflection. This phenomenon known as PP amplification decreases with age and has recently been shown to be associated with cardiovascular disease risk (31, 52). To our knowledge, this is the first study to report that PP amplification is significantly lower in African-American men. There is a linear association between PP amplification and HR (74), and arterial stiffness may exacerbate the influence of HR on PP amplification (59). We noted racial differences in resting HR. However, these differences did not contribute to racial differences in PP amplification, as statistically adjusting for HR did not alter group values. We also assessed nonaugmented PP amplification as a measure of PP amplification due to intrinsic biophysical properties of the aortobrachiocephalic system unperturbed by wave reflection (74). There were no racial differences in this parameter. Given that there were no racial differences in brachial stiffness/diameter and nonaugmented PP amplification is not influenced by HR, these findings would suggest a role for wave reflections in modulating PP amplification in African-American men. Although hav-

Table 5. Pearson correlation coefficients for select vascular and hemodynamic measures

<table>
<thead>
<tr>
<th>Variable</th>
<th>PP_carotid</th>
<th>PP_aortic</th>
<th>B_carotid</th>
<th>AP</th>
<th>PWV</th>
<th>FBF_peak</th>
<th>FBF_AUC</th>
<th>Pulsatility</th>
<th>IMT_carotid</th>
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</thead>
<tbody>
<tr>
<td>PP_aortic</td>
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<td>β_carotid</td>
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<td>0.29*</td>
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<tr>
<td>AP</td>
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<td>0.13</td>
<td>0.04</td>
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<tr>
<td>PWV</td>
<td>0.1</td>
<td>0.28*</td>
<td>0.01</td>
<td>-0.26*</td>
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<tr>
<td>FBF_peak</td>
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<td>-0.10</td>
<td>-0.09</td>
<td>-0.17</td>
<td>-0.18</td>
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<tr>
<td>FBF_AUC</td>
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<td>-0.05</td>
<td>0.62*</td>
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<tr>
<td>Pulsatility</td>
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<td>0.12</td>
<td>0.01</td>
<td>0.06</td>
<td>0.01</td>
<td>0.16</td>
<td>-0.43*</td>
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<tr>
<td>IMT_carotid</td>
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<td>-0.01</td>
<td>0.25*</td>
<td>0.20</td>
<td>0.08</td>
<td>-0.32*</td>
<td>-0.16</td>
<td>-0.15</td>
<td></td>
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PP, pulse pressure; AP, augmented pressure; FBF, forearm blood flow; AUC, area under the curve; IMT, intima-media thickness. *Significant $P < 0.05$.

Table 6. Pearson correlation coefficients for select vascular and hemodynamic measures, cardiorespiratory fitness, and body fat percentage

| Variable         | White | | African-American | |
|------------------|-------| | | | |
|                  | V\_O2\_peak | Body Fat, % | V\_O2\_peak | Body Fat, % | |
| Brachial SBP     | -0.175 | 0.221 | 0.105 | 0.250 | |
| Brachial stiffness | -0.083 | -0.116 | 0.199 | 0.133 | |
| Carotid SBP      | 0.039 | 0.067 | 0.005 | 0.196 | |
| Carotid stiffness | 0.023 | 0.067 | -0.275 | 0.417* | |
| Aortic SBP       | -0.276 | 0.371* | -0.029 | 0.365* | |
| Aortic stiffness | -0.336* | 0.342* | 0.371* | 0.365* | |

V\_O2\_peak, peak $O_2$ consumption; SBP, systolic blood pressure. *Significant $P < 0.05$. 

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The hypertensive response in the forearm microvasculature following ischemia in young African-American men was significantly attenuated, and this is consistent with previous findings. Vasodilatory capacity of resistance arteries has been shown to be attenuated in young (22 yr) African-American men, suggesting early onset of microvascular endothelial dysfunction (7). FBF is significantly attenuated in young black vs. white men following separate infusions of isoproterenol, methacholine, acetylcholine, bradykinin, and sodium nitroprusside, signifying a global impairment of endothelial-dependent and endothelial-independent microvascular vasodilation (36, 39, 64, 66, 67).

It has been suggested that increased pulsatility stemming from vascular stiffness and loss of PP amplification is transmitted to the microvasculature, causing microvascular dysfunction (i.e., rarefaction, microvascular wall thickening/eutrophic remodeling, endothelial damage. etc.) (49). Although brachial blood flow was similar between races in the present study, flow was more pulsatile in the brachial artery in African-American men, and this was a novel finding. It is possible that upstream stiffness of the carotid artery may still propulgate pulsatile flow downstream, causing microvascular damage (49). We noted an association between pulsatility index and FBF area under the curve following ischemia, supporting a link between pulsatile flow and microvascular dysfunction (49).

Low cardiorespiratory fitness and high body fat levels are associated with increased arterial stiffness in white and African-American men (3, 22), and our results confirm an association between race/ethnicity, cardiorespiratory fitness, body fat, and various measures of vascular function. However, when statistically adjusting for cardiorespiratory fitness and body fat levels, racial differences in arterial stiffness and central hemodynamics remained. We have previously shown that aortic PWV is higher in young African-American compared with white men, despite matching groups for fitness and BMI (30). Thus levels of cardiorespiratory fitness and body fat do not appear to fully explain racial differences in vascular function in young men.

Clinical perspectives. Current European Society of Hypertension/European Society of Cardiology guidelines acknowledge the clinical utility of central BP and vascular stiffness measures and support measurement of target organ damage for optimal management of hypertension (1). It has been advocated that traditional brachial BP/cuff measures do not provide a comprehensive view of cardiovascular risk and response to therapy, and reduction of central BP is more closely linked to clinical outcome than change in brachial BP (2). Different anti-hypertensive therapies have variable effects on arterial stiffness, wave reflection, and central BP (18). β-Blockers have been shown inferior to other medications in African-Americans for BP control, as well as reducing cardiovascular events in the general population (17, 53), and this may be related to the inability of β-blockers to modulate pressure from wave reflections (18). Our results support the notion that brachial BP does not reflect vascular burden, particularly in young, healthy African-American men, and alterations in central pressure may precede alterations in brachial pressure. Thus measurement of central BP, vascular stiffness, and or IMT may fill a crucial void in current management of hypertension and related sequelae in African-Americans.

Limitations. African-American and white men were not matched for age. There is a linear association between age and arterial stiffness (45, 48), yet younger African-American men still presented with diffuse vascular dysfunction. Thus group differences were not mediated by slight differences in age. This is a cross-sectional study, and direct cause and effect cannot be ascertained; only associations noted. Use of a transfer function to derive central aortic pressure from radial pressure waveforms has been debated (33). Therefore, we also chose to measure central carotid pressure to further corroborate central BP findings. Overall, central pressure was higher in African-American vs. white men, irrespective of the method employed. We did not control for socioeconomic status. However, all subjects were of similar education level, as all subjects were currently enrolled university students. We do not have information pertaining to dietary history and factors, such as sodium and/or potassium intake, that may influence interpretations of findings.

In conclusion, young African-American men have greater central BP, despite comparable brachial BP compared with young white men, and this was not mediated by HR, cardiorespiratory fitness, or body fat. Brachial BP does not reflect the level of vascular burden in young African-American men. Although having a similar cardiovascular risk factor profile as young white men, diffuse macrovascular and microvascular dysfunction is present at a young age in apparently healthy African-American men. Values seen are comparable to values often reported in older individuals or individuals with more advanced hypertensive disease. This may instigate a cycle whereby increases in central BP beget macrovascular/microvascular damage and vice versa, contributing to target organ damage.

GRANTS
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