Ethnic Differences in the Hemodynamic Mechanisms of Ambulatory Blood Pressure Regulation

Andrew Sherwood, Joel W. Hughes, and Judy McFetridge

Background: African Americans typically exhibit greater systemic vascular resistance (SVR) than do white individuals in response to laboratory challenges that raise blood pressure (BP). However, ethnic differences in ambulatory blood pressure (ABP) regulation have not been examined.

Methods: We monitored ABP in a sample of 10 African American and 10 white men and women. Ambulatory cardiac impedance monitoring was used to assess cardiac output and SVR concurrently with each ABP reading.

Results: We found that SVR was a significant predictor of daily ABP variations in African Americans ($P < .01$) but not in whites.

Conclusion: For African Americans, SVR may play a more prominent role than for whites in the regulation of ABP during routine daily activities.

Key Words: Ambulatory blood pressure, impedance cardiography, cardiac output, systemic vascular resistance, hemodynamics.
antihypertensive medication use were excluded. In all, 50% of the participants were African American and 50% were white. A total of 30% of African American participants and 40% of white participants reported having one hypertensive parent, with the remainder reporting no hypertensive parents. All participants were employed, and the median annual income range endorsed by African American and white subjects was the same ($30,000 to $44,999).

Measurements

Ambulatory BP The ABP monitoring was conducted during the waking hours of a typical workday. The AccuTracker II ABP Monitor (Suntech, Raleigh, NC) was worn from approximately 8 AM until approximately 11 PM. The AccuTracker II was programmed to take four BP measurements per hour at random intervals ranging from 12 to 18 min apart. Participants were instructed to follow their normal schedule and to complete a diary entry after each ABP reading indicating the current time, body posture (sitting, standing, reclining), location (home, work, or other), activity, and “stress” level (0 to 5). An experienced staff member reviewed all BP readings, and artifactual readings were deleted as described previously. After editing, an average of 52 ABP measurements per patient were retained for statistical analysis.

Ambulatory Impedance Monitor The ambulatory impedance monitor (AIM) is a microcomputer based, wearable bioelectric impedance monitor and signal processing system designed for 24-h ABP measurement. The AIM generates an 80 kHz, 2 mA constant sine wave AC current. The AIM computer section ensemble averages, analyzes, and stores the electrocardiogram (ECG), dZ/dt, and Zo waveforms as well as the computed cardiac function indices during each measurement sequence. A tetrapolar combination of spot and band electrodes was used, as described and validated previously. The two recording electrodes were Mylar band electrodes (Instrumentation for Medicine Inc., Greenwich, CT) placed around the base of the neck and around the thorax over the tip of the xiphoid process. Disposable ECG spot electrodes (Cleartrace, Conmed Corp., Utica, NY) were used as current electrodes, with one applied behind the right ear (over the base of the mastoid process) and the other over the lower right ribcage, 6 cm below the lower recording band electrode. The AIM was worn on a belt around the waist and was activated via a cuff-pressure sensor by the initiation of each Accutracker ABP measurement. A 30-sec ensemble average AIM data sample was acquired concurrent with every ABP measurement.

Derivation of Cardiac Function Measures The basal thoracic impedance (Zo), the first derivative of the pulsatile impedance (dZ/dt), and the ECG waveforms from the AIM impedance cardiograph were acquired (each at a 500-Hz sample rate), downloaded to a personal computer, and processed using COP_WIN software (BioImpedance Technology, Chapel Hill, NC). In accordance with recommended standards, left ventricular ejection time was computed as the time interval (msec) between the dZ/dt B-point and X-point, and SV was derived using the Kubicek equation. The SVR was derived from the each of the simultaneously recorded ABP and CO values (SVR = (mean arterial pressure (MAP)/CO)*80; where MAP = [SBP–DBP)/3]+DBP). Because impedance cardiography provides reliable assessment of changes in SV, CO, and SVR, but does not provide valid absolute measurements for these parameters, we did not index these measurements by body size.

Data Analysis

A generalized estimating equation (GEE) approach (SAS PROC GENMOD; SAS, Cary, NC) was used to test the hypotheses that SVR would predict daytime ABP and that this association would be stronger among African Americans. The GEEs are an extension of generalized linear models to repeated measures data. The GEE approach allows an evaluation of ethnic differences in SVR contribution to variations in ABP while controlling for within-subject factors such as posture and between-subject factors such as age, sex, and body mass index (BMI).

Results

Sample characteristics and average daytime levels of SBP, DBP, CO, SVR, and HR are reported in Table 1. No ethnic differences were observed in age, height, weight, or BMI. Average ambulatory daytime HR were higher among African Americans than among whites (Z [1, 872] = 2.65, P = .008). In addition, women had lower average ambulatory SBP (mean 110.9, SD 10.5) than men (mean 120.5, SD 11.1, (Z [1, 888] = 1.96, P < .05). No other sex or ethnic differences in average daytime levels of SBP, DBP, CO, SVR, and HR were observed.

Age, BMI, and posture (reclining, sitting, standing) were entered into generalized estimating equations predicting SBP and DBP. The BMI predicted ambulatory SBP (Z [1, 833] = 2.73, P < .01), and a nonsignificant trend was observed for age (P = .11). Relative to reclining posture, sitting and standing posture were associated with higher SBP and DBP (P < .01). Based on these analyses, age, BMI, and posture were included as control variables in all subsequent analyses. When CO and SVR were added to the model, ambulatory CO was a significant predictor of both ambulatory SBP (Z [1, 831] = 4.99, P < .0001) and DBP (Z [1, 831] = 3.25, P = .001). Similarly, SVR predicted ambulatory SBP (Z [1, 831] = 3.85, P < .0001) and DBP (Z [1, 831] = 4.02, P < .0001).

To test the hypothesis that the contribution of SVR to ABP would be greater among African American subjects, sex and ethnicity were added to GEE models that included SVR as well as the previously mentioned covariates (age, BMI, and posture). For SBP, a significant interaction of
ethnicity and SVR was observed (Z [1, 826] = 2.81, P = .005). For DBP, a trend was observed for SVR to interact with ethnicity (P = .14).

To interpret the interaction of ethnicity and SVR, separate GEE within African American and white subjects were used to test the effect of SVR on SBP. The SVR was a significant predictor of SBP in African Americans (Z [1, 393] = 3.37, P = .0007) but not in whites (P = .46). To illustrate this relationship, the GEE slope coefficients were used to create Fig. 1, which shows the ambulatory SBP values associated with a 1-SD change in ambulatory SVR.

Possible ethnic differences in the contribution of CO to ABP were also evaluated using generalized estimating equations. A significant interaction of ethnicity and CO was observed for SBP (Z [1, 826] = 2.74, P = .006) but not for DBP (P = .86). We found that CO was a significant predictor of SBP in whites (Z [1, 429] = 3.38, P = .0007) but not in African Americans (P = .61).

### Discussion

To our knowledge, these are the first published observations that for African American men and women, SVR may contribute more to variations in ABP than is observed in white Americans. Conversely, CO accounted for variations in ABP for white but not African Americans subjects. These findings are consistent with laboratory studies that have documented a greater SVR contribution to pressor responses elicited by laboratory stressors, including the cold pressor test and a variety of mental stress tasks. Although our sample is too small to draw firm conclusions, the current observations provide important preliminary evidence that individual differences in hemodynamic patterns of BP regulation observed in the laboratory environment may characterize BP regulation during the activities of daily life.

Vascular regulatory mechanisms that may contribute to ethnic differences in BP regulation include both functional and structural characteristics of the vasculature. Several studies have documented that African Americans tend to exhibit increased vascular α-adrenergic receptor sensitivity as compared with whites. These studies include evidence that African American men show increased pressor responses to intravenous phenylephrine, an α-adrenergic receptor agonist. Similarly, in response to phenylephrine infusion via the brachial artery, African American men have been observed to exhibit a greater reduction in forearm blood flow than do white men. In addition, Stein et al found that forearm vasodilation to intrabrachial isoproterenol was attenuated in African American compared with white men. This pattern of altered vascular responsiveness to α- and β-adrenergic receptor stimulation is consistent with evidence that challenges eliciting similar levels of sympathetic activation result in greater vasocstriction in African American compared with white individuals. Our observations of greater SVR regulation of ABP in African Americans may also be a manifestation of

### Table 1. Characteristics of study participants by ethnicity

<table>
<thead>
<tr>
<th>Variable</th>
<th>African American (N = 10)</th>
<th>White (N = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (% male)</td>
<td>50%</td>
<td>40%</td>
</tr>
<tr>
<td>Age (y)</td>
<td>34.4 ± 6.5</td>
<td>36.8 ± 5.9</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.73 ± .13</td>
<td>1.71 ± .09</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>77.7 ± 7.9</td>
<td>72.1 ± 10.7</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.9 ± 2.7</td>
<td>24.5 ± 2.8</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>118.0 ± 10.7</td>
<td>112.4 ± 12.2</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>71.8 ± 5.2</td>
<td>70.3 ± 6.9</td>
</tr>
<tr>
<td>Heart rate (beats/min)*</td>
<td>76.4 ± 11.5</td>
<td>72.6 ± 3.1</td>
</tr>
<tr>
<td>SVR (dynes·sec·cm⁻⁵)</td>
<td>1170.1 ± 286.0</td>
<td>1380.3 ± 497.0</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>6.6 ± 1.4</td>
<td>5.6 ± 1.5</td>
</tr>
</tbody>
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BMI = body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure; SVR = systemic vascular resistance; CO = cardiac output.

* P < .05.

**FIG. 1.** Change in systolic blood pressure (SBP) for 1-SD change in systemic vascular resistance (SVR) in African American compared with white subjects. Note that the slopes depicted for African American and white participants are significantly different (P = .005). The SVR predicts ambulatory SBP among African American (P = .0007) but not white participants.
SNS activation during real-life challenges acting upon a vascular adrenergic receptor system that favors vasoconstriction.

There is also evidence that African Americans may be more likely than whites to develop vascular hypertrophy.\textsuperscript{12} Vascular hypertrophy describes a geometric remodeling that occurs predominantly in precapillary arterial resistance vessels and that is characterized by a decreased lumen size and increased wall/lumen ratio.\textsuperscript{13} This geometric remodeling of the vessel leads to an accentuated pressor response to stimuli eliciting vasoconstriction.\textsuperscript{14} Vascular hypertrophy may therefore also account in part for our observations of greater SVR modulation of BP in African Americans.

Limitations of the current observations include the small sample size, the necessary computational dependence of SVR on BP, and ambulatory hemodynamic assessments that were limited to the waking hours of a typical day. With respect to the small sample size, these participants may not adequately represent the populations of interest (ie, African Americans and whites). In addition, power to detect significant differences between ethnic groups in a number of control variables was limited: for example, sample differences in age (2.4 years), BMI (1.4 kg/m), and SBP (5.6 mm Hg).

Ambulatory hemodynamic monitoring has the potential for providing insights into previously unexplored aspects of cardiovascular control during the activities of daily living. Naturalistic studies of the hemodynamic mechanisms of BP regulation may help to further our understanding of the etiology of cardiovascular disease, as well as to evaluate more carefully the efficacy of treatment. The present findings provide further evidence that SVR may typically feature more prominently in BP regulation for African Americans than in their white counterparts. Future studies using 24-h hemodynamic monitoring in larger, more representative samples may help to establish the possible role of SVR in blunted nighttime BP dipping that has been found to be characteristic of African Americans.\textsuperscript{15}

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References