Nighttime Blood Pressure Dipping: The Role of the Sympathetic Nervous System

Andrew Sherwood, Patrick R. Steffen, James A. Blumenthal, Cynthia Kuhn, and Alan L. Hinderliter

There is a marked diurnal variation in blood pressure (BP), with BP dipping to its lowest levels during nighttime sleep. A day–night dip in systolic BP (SBP) of <10% has been used to characterize individuals as nondippers, and is associated with an increased risk for cardiovascular disease. The present study examined the contribution of the sympathetic nervous system (SNS) to BP dipping in a biracial sample of 172 men and women aged 25 to 45 years. Assessments included 24-h ambulatory BP monitoring and both waking and sleeping urinary catecholamines. In addition, cardiovascular α- and β-adrenergic receptor (AR) responsiveness was determined by the doses of isoproterenol and phenylephrine required to attain an increase in heart rate of 25 points (CD25) and BP (PD25), respectively. Compared with dippers (n = 116), nondippers (n = 56) were more likely to be African American and to have a family history of hypertension as well as a higher body mass index (BMI). The nighttime fall in both norepinephrine (NE) and epinephrine (EPI) excretion rates was reduced in nondippers compared with dippers (NE dip 9.3 v 13.1 μg/mg; EPI dip 2.7 v 4.0 μg/mg; both P < .05). Nondippers also were characterized by heightened α1-AR responsiveness compared with dippers (PD25 = 252 v 321 μg, P < .05). These data suggest that the SNS may contribute to individual differences in nighttime BP dipping, and appears to account in part for blunted BP dipping in African Americans.

Key Words: Ambulatory blood pressure, sympathetic nervous system, ethnicity, adrenergic receptors.

Studies using ambulatory blood pressure (ABP) monitoring technology have shown that ABP is a better predictor of hypertensive target organ damage than blood pressure (BP) measured in the clinic. Studies with ABP have also documented that there is a marked diurnal variation in BP, with pressures typically highest at work and lowest during nighttime sleep. Work ABP was first established as a stronger predictor than clinic BP of left ventricular mass (LVM); however, more recent studies of both hypertensive and normotensive individuals indicate that nighttime ABP may be superior to work ABP as a prognostic indicator of cardiovascular morbidity and mortality. The absence of a normal drop in systolic blood pressure (SBP) from day to night has been found to be predictive of heart failure, stroke, and myocardial infarction, as well as sudden death in elderly patients with systolic hypertension. Studies have typically considered a nocturnal decline in BP of 10% or more to be normal, with such individuals categorized as “dippers,” in contrast to “nondippers” in whom a nighttime fall in BP is attenuated or absent. Nondipping has been associated with increased LVM and wall thickness in both adults and adolescents with high BP. Advancing age is associated with attenuated dipping in both men and women. Although gender differences in diurnal ABP patterns appear to be minimal, BP dipping may become blunted in women after the occurrence of menopause. African Americans are more likely to show a blunted nocturnal drop in ABP, and thus to be categorized as nondippers, than Americans of European origin. Because African Americans also have significantly higher rates of hypertension, as well as cardiovascular mortality associated with heart disease, stroke, and end-stage renal disease, it is plausible that blunted BP dipping may contribute to this ethnic difference in cardiovascular disease risk.

Although blunted BP dipping may contribute to cardiovascular disease risk, the mechanisms accounting for individual differences in BP dipping are poorly understood. The sympathetic nervous system (SNS) plays a major role...
in BP regulation and is considered to be a potential determinant of the magnitude of diurnal BP variation. Several studies have documented that norepinephrine and epinephrine levels show a diurnal variation with a decrease occurring during nighttime sleep. The present study sought to examine the contribution of the SNS to individual differences in BP dipping in African American and white men and women. Concurrently with 24-h ABP monitoring, diurnal variations in SNS activity were assessed by measuring urinary catecholamine excretion rates. Also, because cardiac and vascular adrenergic receptors (AR) mediate the hemodynamic effects of SNS activation, their functional sensitivity was examined in relation to ABP dipping.

Methods

Subjects

Subjects were 172 employed men and women, aged 25 to 45 years, who participated in the Duke Biobehavioral Investigation of Hypertension study. Recruitment was by advertisements in local newspapers. Clinic SBP >180 mm Hg or DBP >100 mm Hg, use of cardiovascular medications, and use of tobacco products were exclusion criteria. The sample comprised 96 men and women with normal BP, including 41 with high normal BP and 35 with stage 1 hypertension, as defined by the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. All assessment procedures were reviewed and approved by the Duke University Medical Center Institutional Review Board. Before their participation in the study, subjects gave informed consent.

Clinic Blood Pressure Measurement

Clinic BP were taken on three separate visits, each approximately 1 week apart. On each visit, three seated BP readings were taken, each 2 min apart, using an appropriate-sized occlusion cuff, mercury column sphygmomanometer, and stethoscope. The SBP was recorded coincident with the first occurrence of Korotkoff sounds (phase I), and DBP with their disappearance (phase V).

Ambulatory Blood Pressure Measurement

The ABP monitoring was conducted during a typical workday. The AccuTracker II ABP Monitor (Suntech AccuTracker II; Suntech Medical Instruments, Raleigh, NC) was worn for approximately 24 hours, usually starting between 8 AM and 10 AM until the same time the following morning. The AccuTracker II measures BP noninvasively, based on the auscultatory technique. It was programmed to take four BP measurements hourly at random intervals ranging from 12 to 28 min apart. Participants were instructed to follow their normal schedule and to complete a diary entry indicating posture, activity, location, and stress level (0 to 5) at each BP reading. The same procedure was followed in the evening waking hours. Activity level during waking hours was measured by a custom-built, accelerometer-based device that was worn on a belt around the waist in a subsample of 122 subjects. This device provided an index of activity, in arbitrary units/min, throughout the waking hours, but was not worn while subjects slept. Sleep was defined by diary activity ratings, which included an indication of “going to sleep.” The ABP monitor was programmed to take only two BP readings hourly during sleeping hours, customized to subjects’ sleep habits. All BP readings were reviewed and artifactual readings deleted following criteria previously described. Mean SBP and DBP values were computed based on all valid readings obtained during waking hours and during nighttime sleep.

Urinary Catecholamines

Subjects were asked to collect a 24-h urine sample during ABP monitoring. Urine samples were collected in separate containers for three different periods: 1) day (entire workday); 2) evening (end of work to bedtime); and 3) overnight (bedtime to waking, including first void of the day). Urine samples were kept cold by storage in a portable cooler throughout the 24-h sample period and returned at completion of the monitoring period.

Samples were assayed for norepinephrine, epinephrine, and creatinine. Urinary levels of norepinephrine and epinephrine were determined by high pressure liquid chromatography with electrochemical detection. Urine creatinine was determined using the Jaffe method as modified by Slot, with kits supplied by Sigma Chemical Company (St. Louis, MO). Catecholamine levels were expressed as urine concentration (µg/mL) per urine concentration of creatine (mg/mL), yielding norepinephrine and epinephrine values in micrograms per milligram of creatine for each sample; this provides catecholamine excretion indices that are corrected for individual differences in body size and urine volume. Day and evening samples were combined to provide “awake” catecholamine excretion rates, whereas the nighttime collection represented “sleep” catecholamine excretion.

Of the 172 study subjects, complete urine samples were available for 141. To guard against poor compliance with 24-h urine collection, 24-h creatine excretion was compared against normative ranges using published algorithms based on gender, ethnicity, and body size. This quality control step reduced the available study sample for catecholamine data analysis to 128 subjects.

β-Adrenergic Receptor Responsiveness

The standardized isoproterenol sensitivity test was used to evaluate β-adrenergic receptor (AR) responsiveness in terms of the chronotropic dose of isoproterenol required to increase heart rate (HR) by 25 beats/min (CD25). Progressively increasing bolus doses of isoproterenol (0.125, 0.25, 0.5, 1.0, 2.0, and 4.0 µg) were injected into an antecubital...
vein until an increase in HR of ≥25 beats/min was observed. The HR responses after each dose were computed as the three shortest, successive electrocardiographic R-R intervals after drug injection, compared with the three shortest R-R intervals at rest, preinjection. After each dose, the next higher dose was not administered for ≥5 min or until cardiovascular activity had returned to resting levels, usually within 5 to 10 min. The linear regression model of log-dose/HR response for each subject was used to determine CD25 exactly by interpolation. The CD25 measure provides an index of cardiac β-AR function that is inversely related to receptor responsiveness. The β-AR assessment procedure was performed while subjects were supine, during the afternoon of a weekday, and within 7 days of the ABP monitoring day.

\[\alpha_1\text{-Adrenergic Receptor Responsiveness}\]

The procedure used for assessing \(\alpha_1\)-AR receptor responsiveness was similar to the test for β-responsiveness described above, but using the \(\alpha_1\) agonist phenylephrine to stimulate vascular \(\alpha_1\) receptors.\(^\text{32}\) In this test, the criterion response is defined as the dose required to increase mean arterial pressure by 25 mm Hg (PD\(_{25}\)). An initial dose of 25 \(\mu g\) phenylephrine was used, with successive doses doubled until the 25 mm Hg response was exceeded or until a maximal dose of 800 \(\mu g\). Again, intervals of at least 5 min (or longer, if required for recovery of cardiovascular activity to resting levels) preceded administration of successive doses. The linear log-dose/MAP response curve was used to determine the exact PD\(_{25}\) dose. The PD\(_{25}\) index is inversely related to vascular \(\alpha_1\)-AR responsiveness. The \(\alpha\)-AR assessment procedure was performed while subjects were supine, during the afternoon of a weekday, and within 7 days of the ABP monitoring day.

\[\text{Demographic and Health Behavior Questionnaires}\]

Socioeconomic status was assessed by self-report of education and income. Family history of hypertension was based on self-report of whether biological parents had a history of high BP requiring treatment. Alcohol consumption was determined by self-report of the total number of drinks (beer, wine, or liquor) over the past week. Aerobic activity was scored using a nonstandardized scale requiring a description of all physical activities over the past 7 days. Type and duration of activity was used to assign aerobic points to each activity, based upon the scheme described by Cooper.\(^\text{33}\) For example, running 3 miles in 20 min would score 15 aerobic points, whereas walking 3 miles in 50 min would score only three aerobic points. A total aerobic exercise score was derived by adding together all aerobic points scored over the previous week.

\[\text{Data Analysis}\]

Blood pressure dipping was computed by subtracting the mean nighttime sleep ABP from the mean daytime waking ABP. For descriptive purposes, subjects were classified either as dippers (defined by a fall in SBP of ≥10% from awake to nighttime sleep) or as nondippers (defined by a fall in SBP of ≤10%). Student \(t\) tests and \(\chi^2\) tests were used to assess whether the demographic, anthropometric, and physiologic characteristics of dippers differed from those of nondippers. Similar analyses were used to test whether characteristics associated with dipping were also related to ethnicity. Finally, by treating BP dipping as a continuous, individual difference variable, hierarchical regression models were developed to examine potential explanatory factors accounting for ethnic differences in dipping. All statistical analyses were conducted using the SAS system (SAS Institute, Cary, NC) with significance set at \(P = .05\).

\[\text{Results}\]

\[\text{Dippers Versus Nondippers}\]

Of the 172 study participants, 116 showed a fall in SBP of >10% during nighttime sleep and were thus categorized as dippers, whereas the remaining 56 showed a fall of ≤10% and were categorized as nondippers. The diurnal BP variation for dippers and nondippers is illustrated in Fig. 1.

The demographic, anthropometric, health behavior, BP, and awake and asleep characteristics according to dipping status are presented in Table 1. Nondippers were more likely to be African American (\(\chi^2 = 10.59, P < .01\)) and to have a family history of hypertension (\(\chi^2 = 9.31, P < \)
.01). Nondippers also had a higher body mass index (BMI) ($t = 2.18, P < .05$) and higher nighttime-sleep ABP (SBP: $t = 7.87, P < .0001$; DBP: $t = 5.93, P < .0001$). All other characteristics, including clinic BP and awake ABP, were similar in dippers and nondippers.

### Sympathetic Nervous System Function in Dippers Versus Nondippers

Indices of sympathetic nervous system function are presented according to dipper status in Table 2. Urine catecholamine data were available only for a subset of subjects ($n = 128$), providing the basis for these analyses. Although awake and sleep catecholamine excretion rates were similar in dippers and nondippers, the catecholamine dip from daytime to nighttime was significantly attenuated in nondippers for both norepinephrine (NE dip, $t = -2.15; P < .05$) and epinephrine (EPI dip, $t = -2.11; P < .05$). For AR responsiveness, nondippers exhibited significantly greater $\alpha_1$-AR responsiveness compared with dippers, as indicated by lower PD$_{25}$ ($t = -2.10, P < .05$).

### Ethnicity and Dipping

As shown in Table 3, African Americans, as compared with whites, had similar awake ABP but higher sleep SBP ($t = 2.60, P < .05$) and DBP ($t = 2.05, P < .05$). The dip in SBP from day to night was significantly attenuated for African Americans ($t = -2.08, P < .05$), whereas the dip in DBP was not significantly different from that of whites ($t = -1.23, NS$). The factors found to be significantly different in dippers compared with nondippers (Tables 1 and 2) are presented according to ethnicity in Table 3. Compared with whites, African Americans were also found to have a smaller drop in norepinephrine from day to night (NE dip, $t = -2.85; P < .05$), and more sensitive $\alpha_1$-AR responsiveness (PD$_{25}$, $t = -3.25; P < .01$). Body mass index, family history of hypertension, and epineph-
rine did not differ significantly according to ethnicity. It is of note that ethnicity was related to differences in several other characteristics: African Americans compared with whites were more likely to be shift workers (17% v 6%, *P* < .05); slept fewer hours (5.7 v 6.5, *P* < .01); and had fewer years of education (13.4 v 16.8, *P* < .001).

**Sympathetic Mediation of Ethnic Differences in Dipping**

Hierarchical regression analysis was used to examine whether the SNS factors related to dipping (Table 2) that also differed according to ethnicity (Table 3) might contribute to the observed ethnic difference in SBP dipping. Results of the three-step hierarchical regression model are presented in Table 4. The dependent variable was SBP dipping, adjusted for awake SBP. In step 1, ethnicity was entered and found to be a significant determinant of SBP dipping (*P* < .05). In step 2, norepinephrine dip (day–night difference) was entered and also revealed to be a significant (*P* < .01) determinant of SBP dipping, but ethnicity now became a nonsignificant factor. The inclusion in step 3 of α₁-AR responsiveness did not alter the model.

**Discussion**

The present study demonstrates that the SNS contributes to individual differences in the magnitude of diurnal BP variation. The magnitude of fall in SNS activity from awake to sleep, as indexed by urinary excretion of catecholamines, corresponded with the degree of BP dipping. Thus, nondippers showed a significantly attenuated nocturnal decline in both norepinephrine and epinephrine excretion. The diurnal rhythm of norepinephrine appears to be driven in part by orthostatic influences. Therefore, we examined the proportion of waking ABP readings that were taken while subjects were standing, sitting, and supine. Dippers and nondippers did not differ in this regard, suggesting that orthostatic effects did not explain our observed differences in diurnal ABP variation between dippers and nondippers. However, we did not record whether, or how often, subjects may have gotten out of bed during the night, and therefore we cannot account for possible orthostatic influences on nighttime ABP. Recent evidence also indicates that the amount of physical activity during the day is a determinant of the magnitude of diurnal ABP variation, and consequently of dipper versus nondipper categorization.

### Table 2. Means and standard deviations by dipper status for urinary catecholamines and adrenergic receptor responsiveness

<table>
<thead>
<tr>
<th>Variable</th>
<th>Nondipper (n = 39)</th>
<th>Dipper (n = 89)</th>
<th><em>P</em></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(≤10% Drop in SBP)</td>
<td>(&gt;10% Drop in SBP)</td>
<td></td>
</tr>
<tr>
<td>Norepinephrine (NE)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Awake NE (μg/mg)</td>
<td>25.2 ± 12.0</td>
<td>28.6 ± 12.9</td>
<td>NS</td>
</tr>
<tr>
<td>Sleep NE (μg/mg)</td>
<td>16.0 ± 8.4</td>
<td>15.5 ± 7.9</td>
<td>NS</td>
</tr>
<tr>
<td>Dip NE (μg/mg)</td>
<td>9.3 ± 8.2</td>
<td>13.1 ± 9.5</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Epinephrine (EPI)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Awake EPI (μg/mg)</td>
<td>4.5 ± 2.7</td>
<td>5.2 ± 4.1</td>
<td>NS</td>
</tr>
<tr>
<td>Sleep EPI (μg/mg)</td>
<td>1.9 ± 2.7</td>
<td>1.2 ± 2.0</td>
<td>NS</td>
</tr>
<tr>
<td>Dip EPI (μg/mg)</td>
<td>2.7 ± 2.8</td>
<td>4.0 ± 4.2</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Adrenergic receptors (AR)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>α₁-AR responsiveness (PD₂₅ [μg])</td>
<td>252 ± 160</td>
<td>321 ± 164</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>β-AR responsiveness (CD₂₅ [μg])</td>
<td>2.0 ± 1.4</td>
<td>2.2 ± 1.8</td>
<td>NS</td>
</tr>
</tbody>
</table>

PD₉₀ – dose required to increase mean arterial pressure by 90 mm Hg; PD₂₅ – dose required to increase heart rate by 25 points; other abbreviations as in Table 1.

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1 Hours of sleep, shift-work, and years of education were not related to blood pressure dipping in the present sample. Nonetheless, a hierarchical regression analysis was performed in order to evaluate whether they contributed to the observed ethnic differences in SBP dipping. In step 1 of the regression model, ethnicity was entered and was a significant determinant of SBP dipping (*P* < .01). In three subsequent steps, hours of sleep, shift-worker status, and years of education were entered. None of these factors were related to dipping, and even with all four factors entered by step 4 of the model, ethnicity remained a significant determinant of SBP dipping (*P* < .01). The results of this analysis indicate that ethnic differences in SBP dipping could not be attributed to the ethnic differences observed for hours slept, shift-work, and years of education.
work. Ambulatory diary ratings of mental stress throughout the day of ABP monitoring were not related to BP dipping in the present study. These observations suggest that differences between dippers and nondippers in the diurnal variation in epinephrine release were not secondary to differences in their daily patterns of mental arousal.

A second aspect of SNS function, vascular \(\alpha_1\)-AR responsiveness, was related to BP dipping. Nondippers exhibited heightened \(\alpha_1\)-AR responsiveness compared with dippers, as indexed by a significantly lower dose of phenylephrine required to raise mean arterial pressure a criterion 25 mm Hg. In contrast, \(\beta\)-AR responsiveness was unrelated to dipping. This finding is consistent with observations made in a study of the effects of doxazosin, a long-acting \(\alpha_1\)-AR blocker, on 24-h ABP in untreated hypertensive subjects.\(^\text{16}\) In that study, doxazosin was associated with a lowering of nighttime SBP in nondippers but not in dippers. The characterization of nondippers by an attenuated diurnal pattern of SNS activation, coupled with heightened sensitivity of vascular \(\alpha_1\)-AR, suggests that SNS regulation of BP may be biased toward a more dominant role for systemic vascular resistance in nondippers. One speculative interpretation of the nondipper SNS profile would be that less SNS activation should be required to achieve the same adjustment in BP. For nondippers, the attenuated dip in SNS activity during sleep would translate into relatively greater \(\alpha_1\)-AR stimulation during the night, resulting in persistent nighttime vasoconstriction. This explanation would suggest that nondippers should be characterized by elevated sleep BP that is secondary to abnormally high systemic vascular resistance during sleep.

The present study also confirmed that African American men and women are more likely to exhibit a blunted fall in SBP compared with white men and women.\(^\text{19}\) Although a higher percentage of our African American subjects were shift workers than were white subjects, we were unable to confirm previous observations that shift work is associated with blunted dipping.\(^\text{37}\) African American subjects also tended to sleep fewer hours than white subjects, and scored lower on an index of socioeconomic status. In our sample, these factors also were unrelated to dipping, and exploratory regression analyses indicated that they did not account for ethnic differences in BP dipping. In contrast, our observations suggest that the sympathetic nervous system may in part account for ethnic differences

### Table 3. Ethnic differences in factors related to BP dipping

<table>
<thead>
<tr>
<th>Comparison Variable</th>
<th>African American ((n = 59))</th>
<th>White ((n = 69))</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Awake SBP (mm Hg)</td>
<td>126 ± 13</td>
<td>124 ± 13</td>
<td>NS</td>
</tr>
<tr>
<td>Awake DBP (mm Hg)</td>
<td>79 ± 10</td>
<td>76 ± 9</td>
<td>NS</td>
</tr>
<tr>
<td>Sleep SBP (mm Hg)</td>
<td>112 ± 14</td>
<td>106 ± 13</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Sleep DBP (mm Hg)</td>
<td>67 ± 11</td>
<td>63 ± 9</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Dip SBP (mm Hg)</td>
<td>14 ± 8</td>
<td>17 ± 7</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Dip DBP (mm Hg)</td>
<td>12 ± 6</td>
<td>13 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>Nondippers (%)</td>
<td>44%</td>
<td>21%</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Family history (% positive)</td>
<td>42%</td>
<td>37%</td>
<td>NS</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>26.1 ± 3.4</td>
<td>25.2 ± 3.2</td>
<td>NS</td>
</tr>
<tr>
<td>Dip NE (µg/mg)</td>
<td>9.5 ± 8.7</td>
<td>14.0 ± 9.3</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Dip EPI (µg/mg)</td>
<td>3.4 ± 2.8</td>
<td>3.8 ± 4.5</td>
<td>NS</td>
</tr>
<tr>
<td>(\alpha_1)-AR responsiveness [PD₂₅ (µg)]</td>
<td>249 ± 155</td>
<td>349 ± 174</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

Abbreviations as in Tables 1 and 2.

### Table 4. Hierarchical multiple regression analysis predicting systolic blood pressure dipping \((n = 114)\)

<table>
<thead>
<tr>
<th>Order of Entry</th>
<th>(b)</th>
<th>(P)</th>
<th>Adjusted (R^2)</th>
<th>Increase in Adjusted (R^2)</th>
<th>Model (F)</th>
<th>Model (p)</th>
</tr>
</thead>
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<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td>.04</td>
<td>.04</td>
<td>5.84</td>
<td>.017</td>
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<tr>
<td>Ethnicity</td>
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<tr>
<td>Step 2</td>
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<td>.11</td>
<td>.07</td>
<td>7.90</td>
<td>.0006</td>
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<tr>
<td>Ethnicity</td>
<td>−0.14</td>
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<td>Dip NE (µg/mg)</td>
<td>−0.29</td>
<td>.003</td>
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<tr>
<td>Step 3</td>
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<td></td>
<td>.12</td>
<td>.01</td>
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<tr>
<td>Ethnicity</td>
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<tr>
<td>Dip NE (µg/mg)</td>
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<td>.003</td>
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<tr>
<td>(\alpha_1)-adrenergic [PD₂₅ (µg)]</td>
<td>−0.13</td>
<td>.170</td>
<td></td>
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</tr>
</tbody>
</table>

Abbreviations as in Tables 2 and 3.
in BP dipping. The SNS characteristics of our African American subjects showed a marked similarity to those of the nondippers. Thus, in addition to blunted BP dipping, African Americans exhibited an attenuated dip in NE excretion during nighttime sleep and an enhanced vascular α₁-AR responsiveness compared with whites. Previous studies have shown that African Americans also exhibit abnormally elevated systemic vascular resistance and heightened vascular constriction in response to SNS activation compared with whites. Heightened vascular α₁-AR sensitivity is a plausible mechanism contributing to exaggerated vasoconstriction in African Americans. These observations relating to ethnicity suggest a broader model whereby vascular abnormalities may lead to blunted BP dipping by limiting the degree to which vascular relaxation may facilitate BP lowering during nighttime sleep.

References


