Published in final edited form as:
Am J Hypertens. 2011 September ; 24(9): 982–988. doi:10.1038/ajh.2011.87.

BLOOD PRESSURE DIPPING: ETHNICITY, SLEEP QUALITY AND SYMPATHETIC NERVOUS SYSTEM ACTIVITY

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Abstract

Background—Blunted blood pressure dipping is an established predictor of adverse cardiovascular outcomes. Although blunted blood pressure dipping is more common in African Americans than whites, the factors contributing to this ethnic difference are not well understood. This study examined the relationships of blood pressure dipping to ethnicity, body mass index, sleep quality, and fall in sympathetic nervous system activity during the sleep-period.

Methods—On 3 occasions, 128 participants with untreated high clinic blood pressure (130–159/85–99 mmHg) underwent assessments of 24-hour ambulatory blood pressure, sleep quality (evaluated by sleep interview, self-report, actigraphy) and sleep-period fall in sympathetic activity (measured by waking/sleep urinary catecholamine excretion).

Results—Compared to whites (n=72), African Americans (n=56) exhibited higher sleep-period systolic (p=.01) and diastolic blood pressure (p<.001), blunted systolic blood pressure dipping (p=.01), greater body mass index (p=.049) and poorer sleep quality (p=.02). Systolic blood pressure dipping was correlated with body mass index (r=−0.32, p<.001), sleep quality (r=0.30, p<.001), and sleep-period fall in sympathetic activity (r=0.30, p<.001). Multiple regression analyses indicated that these 3 factors were independent determinants of sleep-period systolic blood pressure dipping; ethnic differences in dipping were attenuated when controlling for these factors.

Conclusions—Blunted blood pressure dipping was related to higher body mass index, poorer sleep quality, and a lesser decline in sleep-period sympathetic nervous system activity. Although African American ethnicity also was associated with blunted dipping compared to whites in unadjusted analyses, this ethnic difference was diminished when body mass index, sleep quality and sympathetic activity were taken into account.

Keywords

blood pressure dipping; sleep quality; obesity; sympathetic nervous system; ethnicity

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Conflict(s) of Interest/Disclosure(s)
No conflict of interest

BACKGROUND

Blunted nighttime blood pressure (BP) dipping is related to target organ damage in patients with hypertension and is a predictor of cardiovascular morbidity and mortality.[1–2] One of the individual difference characteristics that has been most well documented in relationship to BP dipping is ethnicity, with African Americans characterized by relatively blunted BP dipping.[3–5] African American ethnicity is also characterized by disproportionately high overall cardiovascular risk,[6] and BP non-dipping in African Americans is considered to be a risk factor contributing to this phenomenon. Several potentially modifiable characteristics accounting for individual differences in BP dipping also have been identified. There is growing evidence that poor sleep quality is related to blunted sleep-period BP dipping.[7] Although there is an established causal link between sleep apnea and hypertension through sympathetic nervous system (SNS) hyper-arousal,[8–9] light and fragmented sleep in the absence of sleep apnea also is associated with elevated nighttime BP.[7] Furthermore, an attenuated fall in SNS activity from daytime waking activities to nighttime sleep is characteristic of blunted BP dipping in the absence of sleep apnea.[5, 10] Relative body weight also may be an important consideration, with one recent study finding that a BP "non-dipper" classification was more common in obese individuals compared to individuals of normal weight.[11] Given that both sleep disorders and heightened sympathetic activity may accompany obesity,[12–13] these three factors may be interrelated in their association with blunted BP dipping.

The present study was designed to examine the contributions of sleep quality, SNS activity and body weight to high nocturnal BP in a study sample comprised of African American and white men and women with high blood pressure. Based on prior work, we hypothesized that African American ethnicity, greater SNS activity, poorer sleep quality and greater body mass index (BMI) would be associated with blunted BP dipping during the sleep-period. A secondary aim was to evaluate whether these factors contributed to ethnic differences in BP dipping.

METHODS

Participants

Participants were 128 men and women between the ages of 40–60 years, including 56 African Americans and 72 white Americans. BP inclusion criteria were clinic systolic BP (SBP) 130–159 mmHg and/or diastolic BP (DBP) 85–99 mmHg (which includes the JNC 7 criteria[14] for Stage 1 hypertension and the upper half of the range defined for prehypertension). Exclusion criteria were BMI>35 kg/m^2; antihypertensive medication usage in the previous 12 months; current use of cardiovascular medications; diabetes mellitus; diagnosed sleep apnea; pacemaker; atrial fibrillation; myocardial infarction, percutaneous coronary intervention (PCI), or coronary artery bypass graft (CABG) surgery within 6 months of enrollment; heart failure; severe uncorrected primary valvular disease; uncorrected thyroid heart disease; oral contraceptive use; pregnancy; hormone replacement therapy; alcohol or drug abuse within 12 months; renal or hepatic dysfunction; dementia; inability to comply with the assessment procedures; or inability to provide informed consent. Participants were recruited from the general population of over one million that resides within a 30-mile radius of Duke University Medical Center (DUMC). Recruitment methods included newspaper and television advertisements and posting of study flyers in hypertension clinics and primary health care offices in DUMC Hospital and Clinics, University of North Carolina (UNC) Hospital, Durham Regional Hospital, and Durham Veterans Affairs Hospital. The study sample was selected from a total of 598 people who responded, were screened by phone and met eligibility criteria. The study protocol was

approved by the Institutional Review Board at DUMC. All eligible individuals provided written informed consent prior to participation in the study.

Clinic Blood Pressure Screening

Clinic BP was determined on three visits, each approximately 1-week apart. After subjects were seated for 5 minutes seated in a quiet, temperature-controlled room, four BP readings, each 2 minutes apart, were taken using a mercury sphygmomanometer and stethoscope. SBP and DBP for each visit were calculated as the means of the last three readings, and clinic BP eligibility was based upon whether the average of the three mean office BP readings met the study's inclusion criteria.

Sleep Apnea Screening

The Duke Structured Interview for Sleep Disorders[15] was used to assess for symptoms of sleep disorders (e.g. sleep apnea). Five participants were excluded because of a diagnosis of sleep apnea.

Blood Assays

Blood samples were collected in the morning after an overnight fast. Specimens were analyzed by LabCorp using automated enzymatic assays for glucose, total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), and triglycerides, kinetic assay for creatinine, and flame photometer assay for sodium. The modified Cockroft-Gault equation was used to estimate glomerular filtration rate (GFR).[16]

Ambulatory Blood Pressure Monitoring (ABPM)

24-hour ambulatory BP (ABP) was assessed on three occasions during regular weekdays (Mon–Fri), with an interval of one week between monitoring sessions. Shift workers were not evaluated during the same day-night schedule as nonshift workers, and so for descriptive accuracy we refer to participants' sleep-period, rather than nighttime. ABP was measured with the Oscar 2 monitor (Suntech Medical Inc., Raleigh, NC)[17] every 20-minutes during waking hours, and every 30-minutes during the sleep-period. Artifactual readings were deleted according to criteria described previously.[18]

Measures of Sympathetic Nervous System Activity

Participants collected a 24-hour urine sample during each ABPM session. Separate containers were designated for the waking and overnight (bedtime to waking, including first void of the day) collection periods and containers were stored in portable coolers. Urinary 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 creatinine (mg/ml), yielding norepinephrine and epinephrine values of μg per mg creatinine for each sample; this provided catecholamine excretion indices that were corrected for individual differences in body size and urine volume.[19] However, creatinine adjustment is not without its own inherent limitations,[20] and there were no modifications of diet or medications recommended before collection of samples for urinary catecholamine measurements.

Measures of Sleep Quality

Mini-Mitter Actiwatch wrist-watch style actigraphs (Mini-Mitter Co., Inc., Sunriver, OR) were used to derive objective estimates of sleep parameters including sleep efficiency (the percent of time asleep during the sleep period), the fragmentation index of the restlessness of

sleep ([minutes moving / sleep period] × 100 + [number of immobile phases that last ≤1 minute / total number of immobile phases] × 100), total wake time, total sleep time, and time in bed. The Pittsburgh Sleep Quality Index (PSQI) was used to assess subjective sleep quality.[21]

Demographic and Psychosocial Assessments

Data were collected on age, gender, height, weight, BMI, waist circumference, hip circumference, WHR, cigarette smoking, alcohol consumption and shift work. Participants also completed psychosocial measures of anxiety (State-Trait Anxiety Inventory),[22] depression (Beck Depression Inventory),[23] and perceived stress (Perceived Stress Scale).[24]

Data Reduction and Statistical Analysis

Waking and sleep periods, defined by self-report, and confirmed by actigraphy, were used to compute mean waking ABP and mean sleep-period ABP values for each of the three ABPM sessions. BP dipping was derived by subtracting mean sleep-period BP from mean waking BP. The within-subject variation in the dip in SBP between ABPM sessions averaged 6 mm Hg across study participants, with a standard deviation of 4 mm Hg; the dip in DBP showed an average variation of 5 mm Hg across sessions, with a standard deviation of 3 mm Hg. Values for the three sessions were averaged to derive a more robust measure of BP dipping for each participant, with the averaged magnitude of SBP and DBP dip in mm Hg providing continuous measures serving as the primary dependent variables in this report. For descriptive purposes nondipper was defined as a fall in sleep-period SBP of ≥ 0% to < 10% compared to waking SBP; dipper was defined as a fall in sleep-period SBP of ≥10% to <20% compared to waking SBP; extreme dipper was defined as a fall in sleep-period SBP of ≥ 20% compared to waking SBP; riser was defined as a rise in sleep-period SBP compared to waking SBP. Given the between session variability of BP, it is of note that only 68 of the 128 participants (53%) had a consistent dipping status (dipper versus nondipper) over the three ABPM sessions. White coat hypertension was defined as a clinic SBP of ≥140 and/or clinic DBP≥90 mmHg that was associated with a mean awake ambulatory SBP<135 and ambulatory DBP<85 mmHg. Waking and sleep-period catecholamine levels were averaged across the three sessions and mean sleep-period dip in norepinephrine and epinephrine were computed. A composite index of SNS dip was derived by combining norepinephrine dip and epinephrine dip according to the procedure recommended by O'Brien.[25] The PSQI score was combined with sleep efficiency and sleep fragmentation index to form a composite index of sleep quality.

Normality statistics (skew, kurtosis), histograms, boxplots and normal probability plots were examined to assess normality of the measured variables. Data are expressed as mean ± s.d. for continuous normally distributed variables or median (interquartile range) for non-normally distributed continuous variables. T-tests (for normal distributions) or the Wilcoxon rank sum test (for non-normal distributions) were used to compare ethnic differences in continuous variables. χ^2 tests were used to assess differences in categorical variables between African American and white participants. ANCOVA tests were used to evaluate ethnic differences in BP dipping, while controlling for waking BP. Pearson's or Spearman's correlations, partialling waking BP, were used to assess relationships between sleep-period BP dip and demographic/anthropometric characteristics, catecholamines and sleep quality. A two step, hierarchical regression approach was used to examine whether controlling for factors related to BP dipping affected ethnic differences in BP dipping. Covariates were selected based on a priori hypotheses and observed associations with BP dipping, with waking ABP always included as a covariate, thereby providing results that for descriptive purposes focus on BP dip (delta mm Hg) as the dependent variable, but are equivalent to evaluating the absolute magnitude of sleep-period BP (controlling for waking BP). Ethnicity

was entered into step 1 of the hierarchical regression model, followed by sleep quality, BMI and SNS activity dip in step 2. Potential interactions of ethnicity and sleep quality, ethnicity and BMI, and ethnicity and SNS dip were explored. Statistical analyses were conducted using the SAS 9.2 system (SAS Institute, Cary, NC) with significance set at p=.05.

RESULTS

Demographic Characteristics

The 128 participants included 29 African American women, 27 African American men, 23 White American women and 49 White American men, with a mean age of 46 years and a mean screening BP of 140/90 mm Hg (Table 1). No participants had a history of PCI and/or CABG. For 13 participants, SBP or DBP was outside the 130–159/85–99 mmHg range on a single screening visit. However, averaged across the three screening visits, all participants' BPs were within the eligible range. Thus, although there was some variability in BP across screening sessions, the majority of participants had measurements that were consistently within the range of eligibility. Of the 85 participants classified as stage 1 hypertensive based on average clinic BPs of ≥140/90 mmHg, 26 (31%) were found to have white coat hypertension based on a mean awake ambulatory BP <135/85 mmHg. There was a higher proportion of women amongst the African American study sample than in the white study sample (χ^2=5.14, p=.02). African Americans were also more likely to be younger (t(125.98)=−2.66, p=.01), have a greater BMI (t(126)=1.98, p=.049), and a higher clinic DBP (t(126)=3.21, p<.01). There were no ethnic differences in anxiety (t (126)=−0.20, p=.84), depression (W_S= 3762.5, z=.72, p=.47) or perceived stress (t(126) =0.56, p=.58).

ABP, SNS Activity and Sleep Quality

African Americans had higher DBP while awake (DBP: t(126)=3.17, p<.01) and both SBP and DBP were higher during the sleep- period (SBP: t(126)=2.83, p=.01; DBP: t(126)=3.65, p<.001); sleep-period SBP dip was blunted in African Americans compared to whites (F(2, 125)=4.24, p=.01). Overall, 42.9%, 50.0% and 7.1% of African Americans and 19.4%, 69.5% and 11.1% of whites were classified as nondippers, dippers and extreme dippers, respectively. No participant was classified as a riser. Compared to whites, proportionately more African Americans were classified as nondippers than dippers (χ^2=7.68, p<.01) Lower sleep efficiency (W_S= 3045.5, z=−2.72, p=.01) and a lower composite sleep quality score (t(126)=−2.47, p=.02) were also observed among African Americans (Table 2).

Demographics, SNS Activity, Sleep Quality and BP Dipping

As described in supplemental Table S1, ethnicity, BMI, waist circumference, awake epinephrine, epinephrine dip, awake norepinephrine, SNS dip, sleep efficiency, fragmentation index, PSQI, and the composite sleep index were all associated with SBP dipping (all p's<.05). DBP dipping was associated with awake epinephrine, epinephrine dip, SNS dip, sleep efficiency, fragmentation index, PSQI, and the composite sleep score (all p's<.05). White coat hypertension was unrelated to both SBP dipping (p=.34) and DBP dipping (p=.59). We found that BMI was related to the fall in sleep-period SNS activity (r= −0.20, p=.02), with higher BMI associated with a smaller fall in SNS arousal. BMI also was related to sleep quality (r=−0.31, p<.001), with greater BMI associated with poorer sleep quality. Sleep quality and diurnal SNS changes were found to be unrelated (r=.06, p=.49).

Relationships of BMI, Sleep Quality, and SNS Activity to SBP dipping

Hierarchical regression analysis was used to examine the roles of ethnicity, BMI, sleep quality, and SNS activity dip in accounting for individual differences in SBP dipping. In step 1 of the model, ethnicity was entered and confirmed as a significant determinant of SBP

dipping (p=.01; Table 3a). When BMI, sleep quality (composite index), and dip in SNS activity were entered in step 2, each of these three factors explained a portion of the combined 20.3% of variance accounted for in SBP dipping. However, by accounting for these factors, ethnic differences in SBP dipping were no longer significant (p=.09). Potential interactions of ethnicity and sleep quality, ethnicity and BMI, and ethnicity and SNS dip were assessed in separate exploratory hierarchical regression models that showed these interactions to be non-significant.

Relationship of BMI, Sleep Quality, and SNS Activity to DBP dipping

A similar hierarchical regression analysis was used to examine predictors of DBP dipping (Table 3b). Ethnicity and BMI were not significant predictors of DBP dipping. Sleep quality and SNS dip both predicted DBP dipping, with the final model accounting for 12% of the variance.

DISCUSSION

The current study demonstrates that BMI, sleep quality, and fall in sleep-period SNS activity contribute to individual differences observed in SBP dipping among men and women with high blood pressure. While previous studies have shown each of these factors to be related to BP dipping, the present study documents their importance in the context of hypertension and prehypertension, and demonstrates that while they are interrelated, they are nonetheless independent contributors to the magnitude of sleep-period BP dipping. We also confirmed previous observations that African Americans compared to whites exhibited blunted SBP dipping,[6–8] but found these ethnic differences in BP dipping were less evident (p=.09) after adjusting for BMI, sleep quality and SNS activity. For diastolic BP dipping, we observed a non-significant trend (p=.08) for ethnic differences in diastolic BP dipping, which is in accordance with our previous research in which African Americans exhibited blunted dipping compared to whites for SBP but not DBP.[5] We also note that approximately one third of the 85 participants characterized as hypertensive on the basis of clinic BP readings were subsequently revealed by their waking ambulatory BPs to be more accurately characterized as having white coat hypertension. However, white coat versus confirmed hypertension was found to be unrelated to BP dipping.

Our study confirms that poor sleep quality is associated with blunted BP dipping.[7, 26–27] Poor sleep quality has been found to be associated with pre-hypertension in healthy adolescents[28] and also with an increased prevalence of hypertension in adults.[29] In agreement with previous research,[4] African Americans in our study sample were characterized by poorer sleep quality than their white counterparts. Hughes et al.[4] tested the hypothesis that poor sleep quality may account for ethnic differences in BP dipping in young (18–25 yrs), healthy men and women, but found no support for this proposition. In addition to poor sleep quality, we also found that an attenuated fall in SNS activity during the sleep period is related to blunted BP dipping. Although not all studies have demonstrated that SNS activity contributes to the magnitude of BP dipping,[7] this finding confirms our previous report.[5] Inconsistent findings may reflect methodological differences between studies in the evaluation of SNS activity, and the possible importance of taking into account the diurnal variation in SNS activity. The SNS is believed to play a key role in BP regulation during both the day and at night. During sleep, and especially during non-rapid eye movement sleep, the baroreceptor reflex set point is shifted downward resulting in reduced sympathetic activity to the heart and vasculature, ultimately contributing to a decline in BP.[30–32] Sleep quality was found to be unrelated to the fall in sleep-period SNS activity; however, both sleep quality and SNS activity were independent predictors of SBP and DBP dipping. We also noted that there were no ethnic differences in the dip in SNS activity during the sleep-

period, and accordingly this factor is unlikely to contribute to ethnic differences in BP dipping.

Our observed relationship between BMI and BP dipping is consistent with Kotsis et al.[11] and Kagan et al.,[33] who reported increased prevalence of non-dipping among obese adults. Pathophysiological mechanisms implicating obesity in the etiology of hypertension are well documented.[34] Obesity is associated with greater SNS activity[35] and SNS hyperactivity has been proposed to be a likely mediator of obesity-related hypertension.[36] In our study sample of men and women with high blood pressure, African Americans had a higher BMI than their white counterparts. When BMI, sleep quality and SNS activity were included in our regression models, ethnic differences in BP dipping were attenuated, suggesting that they may been contributing factors. It is well established that weight loss is an effective method for lowering BP in men and women with high BP.[37–38] Our findings suggest the possibility that a further benefit of weight loss may be enhanced BP dipping, potentially achieved by improving sleep quality and augmenting the sleep-time fall in SNS activity.

Strengths of our study include a sample of African American and White American men and women with hypertension and prehypertension. Limitations of study include the observed inconsistency in the magnitude of BP dipping over the three 24-hour ABPM sessions. Consequently, BP dipping, sleep quality and SNS activity were based upon assessments averaged over these three occasions, repeated at weekly intervals, thereby providing a more reliable description of these individual difference characteristics. In addition to self-report we also used actigraphy to assess sleep quality. However, although a sleep interview[15] and the Berlin Sleep Apnea Index[39] were used to exclude participants with sleep apnea, the absence of polysomnography to more definitively rule out sleep apnea is a limitation. The cross-sectional study design restricts inferences regarding causal relationships amongst the factors under examination. The relatively small sample size may have potentially obscured other factors influencing BP dipping.

CONCLUSION

BMI, sleep quality and the magnitude of decline in SNS activity during sleep were all predictive of SBP dipping in men and women with high blood pressure. African Americans exhibited blunted SBP dipping compared to their white counterparts, and they also evidenced poorer sleep quality and higher BMI. Future research should further examine the role of BMI in relation to BP dipping, including its association with poor sleep quality and elevated SNS arousal, and the potential benefits of weight loss interventions on BP dipping.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We thank Julie Bower, Amy Franklin, and Angela Kirby for their work in supporting the conduct of this study.

Sources of Funding

This study was supported by Grant HL072390 from the National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, MD, and grant M01-RR-30 from the General Clinical Research Center program, National Center for Research Resources, National Institutes of Health.

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Table 1

Sample characteristics in African American and white participants

	Total (n=128)	AA (n=56)	W (n=72)	*p*
Age (yrs)	45.7 ± 8.4	43.5 ± 7.0	47.3 ± 9.1	**.01**
Female (%)	40.6	51.8	31.9	**.02**
BMI (kg/m^2)	28.2 ± 3.7	28.9 ± 3.8	27.6 ± 3.5	**.049**
Waist (cm)	95.8 ± 11.6	95.4 ± 12.6	96.1 ± 10.9	.77
Hip (cm)	107.9 ± 8.0	108.5 ± 9.1	107.3 ± 7.0	.42
WHR	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1	.15
Clinic SBP (mmHg)	140 ± 7	141 ± 7	140 ± 7	.16
Clinic DBP (mmHg)	90 ± 5	91 ± 5	88 ± 5	**<.01**
Glucose (mg/dL)	92 (87–100)	89 (84–96)	94 (89–101)	**<.01**
Total cholesterol (mg/dL)	196 ± 36	191 ± 35	199 ± 36	.20
HDL-C (mg/dL)	56 ± 1.7	57 ± 17	55 ± 17	.49
LDL-C (mg/dL)	116 ± 28	113 ± 28	119 ± 29	.23
Triglycerides (mg/dL)	94 (68–135)	84 (62–136)	95 (78–131)	.12
Serum Sodium (mmol/l)	140 ± 3	140 ±3	140 ± 3	.28
Serum Creatinine (mg/dL)	0.9 ± 0.2	0.9 ± 0.2	0.9 ± 0.1	.75
GFR (ml/min)	118 ± 29	120 ± 29	117 ± 29	.49
Shift worker (%)	9.6	14.6	5.7	.10
Smoker (%)	9.4	14.3	5.6	.09
# alcoholic beverages in the past week	3.0 ± 5.0	2.2 ± 4.3	3.7 ± 5.4	.08

For continuous variables, data are mean ± s.d. or median (interquartile range) for normally distributed and non-normally distributed data respectively. For categorical variables, data are relative frequencies (percentages).

AA = African American; BMI = body mass index; Total cholesterol = total cholesterol; DBP = diastolic blood pressure; GFR = glomerular filtration rate; HDL-C = high density lipoprotein cholesterol; HTN = hypertension; LDL-C = low density lipoprotein cholesterol; SBP = systolic blood pressure; W = White American; WHR = waist hip ratio.

Table 2

Ambulatory blood pressures, SNS activity and sleep quality in African American and white participants

	Total (n= 128)	AA (n = 56)	W (n= 72)	*p*
24-hr ABP				
Awake SBP (mmHg)	137 ± 11	139 ± 9	135.7 ± 12	.11
Awake DBP (mmHg)	84 ± 8	86 ± 7	82 ± 8	**<.01**
Sleep SBP (mmHg)	119 ± 12	122 ± 10	117 ± 13	**.01**
Sleep DBP (mmHg)	69 ± 9	72 ± 8	66 ± 10	**<.001**
Dip SBP (mmHg)	18 ± 7	16 ± 7	19 ± 6	**.01**
Dip DBP (mmHg)	15 ± 5	14 ± 5	16 ± 5	.08
Nondipper* (%)	32.8	46.2	21.9	**<.01**
Catecholamines				
Awake EPI (μg/mg)	3.4 (2.2–4.8)	3.4 (2.3–4.8)	3.4 (2.1–5.0)	.88
Awake NE (μg/mg)	23.6 (17.3–32.3)	23.0 (16.8–17.7)	24.9 (18.0–33.4)	.38
Sleep EPI (μg/mg)	1.0 (0.7–1.6)	1.1 (0.7–1.8)	0.9 (0.6–1.6)	.25
Sleep NE (μg/mg)	13.9 (10.0–17.7)	13.3 (9.9–17.7)	13.9 (10.6–17.7)	.56
Dip EPI (μg/mg)	2.2 (1.2–3.5)	2.1 (1.4–3.2)	2.3 (1.0–3.8)	.68
Dip NE (μg/mg)	9.3 (5.0–15.6)	8.7 (5.2–14.1)	11.0 (4.9–16.7)	.48
SNS Dip	129.0 ± 65.8	124.9 ± 58.4	132.20 ± 71.19	.53
Sleep Measures				
Sleep Efficiency	83.2 (75.9–87.4)	78.4 (72.7–86.2)	84.6 (79.0–87.8)	**.01**
Fragmentation Index	29.3 (23.4–36.9)	31.8 (24.4–38.4)	28.4 (21.7–34.5)	.13
Time in Bed (hr)	7.5 ± 1.2	7.3 ± 1.4	7.6 ± 1.0	.16
Global PSQI	6.2 ± 3.8	6.7 ± 4.0	5.8 ± 3.6	.17
Sleep Quality	193.5 ± 86.8	172.4 ± 90.3	209.9 ± 80.9	**.02**

For continuous variables, data are mean ± s.d. or median (interquartile range) for normally distributed and non-normally distributed data respectively. For categorical variables, data are relative frequencies (percentages).

AA = African American; ABP = ambulatory blood pressure; DBP = diastolic blood pressure; EPI = creatinine corrected epinephrine; NE = creatinine corrected norepinephrine; PSQI = Pittsburg Sleep Quality Index; SBP = systolic blood pressure; Sleep Quality = Composite Score of sleep efficiency, fragmentation index and the global PSQI; SNS = sympathetic nervous system; SNS Dip = composite measure of nighttime dip in urinary catecholamines; W = White American

Nondipper was defined as a fall in sleep-period SBP of ≥ 0% to < 10% compared to waking SBP.

*nondipper vs dipper

Table 3

Hierarchical regression models evaluating the association of BP dipping with ethnicity, BMI, sleep quality and SNS activity dip (n = 128).

a) Systolic Blood pressure Dipping

	B	β	*P* value	95% CI for *B*	
				Lower	Upper
Step 1 (dependent variable: SBP dipping) adjusted R^2 = .05					
Ethnicity	−3.07	−0.23	.01	−5.43	−0.72
Step 2 (dependent variable: SBP dipping) adjusted R^2 = .20					
Ethnicity	−1.90	−0.14	.09	−4.12	0.31
BMI	−0.36	−0.19	.03	−0.67	−0.04
Sleep Quality	0.02	0.20	.02	0.002	0.03
SNS Dip	0.02	0.24	<.01	0.01	0.04

b) Diastolic Blood pressure Dipping

	B	β	*P* value	95% CI for *B*	
				Lower	Upper
Step 1 (dependent variable: DBP dipping) adjusted R^2 = .01					
Ethnicity	−1.58	−0.16	.08	−3.35	0.20
Step 2 (dependent variable: SBP dipping) adjusted R^2 = .12					
Ethnicity	−0.81	−0.08	.35	−2.53	0.91
BMI	0.05	−0.03	.70	−0.28	0.19
Sleep Quality	0.01	0.23	.01	0.003	0.02
SNS Dip	0.02	0.26	<.01	0.006	0.03

B = non-standardized regression coefficient; β = standardized regression coefficient; BMI = body mass index; CI = confidence interval; Sleep Quality = composite measure of sleep efficiency, fragmentation index and the global PSQI; SNS Dip = composite measure of nighttime dip in urinary catecholamines.