ORIGINAL ARTICLE

Racial Differences in Abnormal Ambulatory Blood Pressure Monitoring Measures: Results From the Coronary Artery Risk Development in Young Adults (CARDIA) Study

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BACKGROUND
Several ambulatory blood pressure monitoring (ABPM) measures have been associated with increased cardiovascular disease risk independent of clinic blood pressure (BP). African Americans have higher clinic BP compared with Whites but few data are available on racial differences in ABPM measures.

METHODS
We compared ABPM measures between African American (n = 178) and White (n = 103) participants at the Year 5 Coronary Artery Risk Development in Young Adults study visit. BP was measured during a study visit and the second and third measurements were averaged. ABPM was conducted over the following 24 hours.

RESULTS
Mean ± SD age of participants was 29.8 ± 3.8 years and 30.8 ± 3.5 years for African Americans and Whites, respectively. Mean daytime systolic BP (SBP) was 3.90 (SD 1.18) mm Hg higher among African Americans compared with Whites (P < 0.001) after age–gender adjustment and 1.71 (SD 1.03) mm Hg higher after multivariable adjustment including mean clinic SBP (P = 0.10). After multivariable adjustment including mean clinic SBP, nighttime SBP was 4.83 (SD 1.11) mm Hg higher among African Americans compared with Whites (P < 0.001). After multivariable adjustment, the African Americans were more likely than Whites to have nocturnal hypertension (prevalence ratio: 2.44, 95% CI: 0.99–6.05) and nondipping (prevalence ratio: 2.50, 95% CI: 1.39–4.48). The prevalence of masked hypertension among African Americans and Whites was 4.4% and 2.1%, respectively, (P = 0.49) and white coat hypertension was 3.3% and 3.9%, respectively (P = 0.99). Twenty-four hour BP variability on ABPM was higher among African Americans compared with Whites.

CONCLUSIONS
These data suggest racial differences in several ABPM measures exist.

Keywords: ambulatory blood pressure; blood pressure; disparities; hypertension; race.

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Ambulatory blood pressure monitoring (ABPM) provides several distinct measures that cannot be determined by clinic blood pressure (BP) alone.[1] These measures include "true" or mean BP, nocturnal BP, and 24-hour BP variability. Additionally, several phenotypes can be defined by either the presence of a clinic-ambulatory hypertension mismatch (i.e., masked hypertension and white coat hypertension) or comparison of day and night BP (e.g., nondipping pattern). Higher mean ambulatory BP and separately masked hypertension maintain strong associations with an increased risk for cardiovascular disease (CVD) outcomes, whereas white coat hypertension typically has a benign prognosis.[2,3] Recent studies have also reported associations of higher 24-hour BP variability and, separately, abnormal diurnal BP patterns (e.g., nighttime BP nondipping and/or nocturnal hypertension) with an increased risk of CVD and all-cause mortality.[4–6]

African Americans have higher clinic BP and an increased risk for CVD and all-cause mortality than Whites.[7,8] Additionally, African Americans are more likely than Whites to have abnormal autonomic function, abnormal baroreflex function, and altered sodium excretion.[9–11] These biological mechanisms are implicated not only in higher mean BP, but also in increased BP variability and abnormal diurnal BP rhythm.[12–14] Therefore, compared with Whites, African Americans may have higher mean ambulatory BP, a higher prevalence of masked hypertension, increased BP variability, and abnormal diurnal rhythm on ABPM. As prior population-based studies of ABPM have had scarce minority representation, there are limited data on racial differences

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in ABPM measures and the prevalence of abnormal BP phenotypes.

The aim of the current study was to examine racial differences in BP measures defined by 24-hour ABPM between Whites and African Americans. Specifically, using data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, we compared mean levels of daytime and nighttime systolic BP (SBP) and diastolic BP (DBP) and 24-hour BP variability on ABPM between Whites and African Americans. Additionally, racial differences in the prevalence of sustained, masked, white coat, and nocturnal hypertension and BP nondipping were determined.

METHODS

CARDIA is a prospective cohort study sponsored by the National Heart, Lung, and Blood Institute. Detailed methods for CARDIA have been published previously.[15] In brief, the cohort was recruited at four field centers in the United States (Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA). CARDIA enrolled 5,115 African American and White men and women, aged 18–30 years, in 1985 and 1986. Of relevance to the current analysis, ABPM was conducted in participants from the Birmingham, AL field center at the Year 5 study visit in 1990–91. Overall, 400 of the 939 CARDIA participants enrolled at the Birmingham center who attended the Year 5 study visit were randomly selected to participate in the ABPM substudy. The ABPM substudy was completed by 316 participants. The primary reason for nonparticipation was burden of the ABPM after the study visit and jobs that made ABPM difficult to complete (e.g., truck drivers).

Data collection

Data on age, gender, and self-reported race were collected by questionnaire at the baseline CARDIA study visit and confirmed at the Year 5 study visit. Educational status and current smoking at the time of the Year 5 study exam were determined through self-report. Physical activity at the Year 5 study visit was assessed using the interview-administered Physical Activity History Questionnaire.[16] Physical activity is reported in exercise units with 300 units approximating the American College of Sports Medicine recommendations for the amount of exercise needed to support weight loss. Body mass index (BMI) was calculated based on measured height and weight from the Year 5 study visit. Blood was collected at the Year 5 study visit and stored at −70 °C until measurements were performed. Both fasting glucose and fasting lipid profile (triglycerides, total cholesterol, and high-density lipoprotein (HDL) cholesterol) were measured using a standard laboratory technique. The presence of diabetes was defined as fasting blood glucose ≥126 mg/dl or use of insulin and/or oral hypoglycemic agents.

Clinic BP measurements

Clinic SBP and DBP were measured at the Year 5 CARDIA examination following standardized protocols by trained and certified staff. Briefly, BP was measured on the right arm with a Hawksley random-zero sphygmomanometer (W. A. Baum Co., Copiague, NY). The appropriate BP cuff size was determined by measuring the participant's arm circumference at the midpoint between the acromion and olecranon. Participants were asked to sit quietly for at least 5 minutes, in a comfortable posture, with feet flat on the floor prior to their BP measurement. All readings were taken in the seated position with the participant's back supported and with their right arm positioned at heart level. Three clinic BP measurements were separated by at least 30 seconds and measurements were recorded to the nearest even number. For the current analysis, we calculated clinic SBP and DBP using the mean of the second and third BP measurements.

ABPM measurements

ABPM was conducted over a 24-hour period following the study visit using a Suntech Accutracker II (Suntech Medical, Morrisville, NC) and an appropriately sized cuff.[17] SBP and DBP were measured every 20 minutes between 6:00 AM and 10:00 PM and every 30 minutes from 10:00 PM to 6:00 AM. Values outside of preset limits for SBP and DBP (≥220 or ≤80 mm Hg for SBP and ≥130 or ≤40 mm Hg for DBP) were rejected and repeat BP measurements were taken. In addition, a change of 50 mm Hg in systolic pressure, of 40 mm Hg in diastolic pressure, or of 50 mm Hg in pulse pressure between readings also triggered a rejection and a new reading. Daytime was defined as the period between 10:00 AM and 8:00 PM, nighttime was defined as the period between midnight and 06:00 AM, and morning was defined as the period from 08:00 AM to 10:00 AM.[18] To be eligible for the current study, we required participants to have 10+ valid daytime readings and 5+ valid nighttime readings. These criteria are consistent with those applied in the International Database of Ambulatory Blood Pressure in relation to Cardiovascular Outcome (IDACO), a very large international database consisting of several prospective population-based samples.[18] Poor sleep quality during the ABPM period was defined by the participant reporting not being able to fall asleep, not being able to sleep at all, or being awakened five or more times.

Outcome definitions

Mean SBP and DBP during daytime and nighttime, separately, were calculated. Elevated daytime BP was defined as mean SBP ≥135 mm Hg or mean DBP ≥85 mm Hg and elevated clinic BP was defined by mean SBP ≥140 mm Hg or mean DBP ≥90 mm Hg.[19] Sustained hypertension was defined as elevated daytime and clinic BP. Masked hypertension was defined as elevated daytime BP without elevated clinic BP and white coat hypertension was defined as elevated clinic BP without elevated daytime BP. Nocturnal hypertension was defined as a mean nighttime SBP ≥120 mm Hg or DBP ≥70 mm Hg.[20] BP dipping was defined by the ratio of mean nighttime-to-daytime SBP.[1] Nondipping pattern was defined as a ratio ≥0.90. Morning surge was calculated as the difference between the mean SBP during the morning hours

and nighttime trough SBP.[21] Trough SBP was defined as the mean of three SBP measurements: the lowest nighttime SBP and the measurements immediately preceding and following this measurement.

Two measures of BP variability were calculated: day–night SD (SD_{dn}) and average real variability (ARV). The SD for daytime measurements and, separately for nighttime measurements, was calculated and the SD_{dn} was calculated as the weighted mean of these SDs.[22] This approach is considered advantageous compared with calculating a single SD over 24 hours because it eliminates the influence of the day–night change in BP. ARV was calculated as the average absolute difference between consecutive readings over the 24-hour ABPM period.[23] The ARV accounts for the order of BP measurements over the ABP monitoring period.

Statistical analysis

Participant characteristics and clinic SBP, DBP, and pulse pressure were calculated for Whites and African Americans and compared using *t*-tests and chi-square tests, as appropriate. Additionally, ABPM phenotypes including mean daytime, nighttime, and morning SBP and DBP, SD_{dn} and ARV of SBP and DBP, dipping percent, and morning surge were calculated by race and compared using *t*-tests. Racial differences in these phenotypes were determined after multivariable adjustment using linear regression models. Initial models included adjustment for age and gender (Model 1). Subsequent models also included adjustment for education, physical activity, current smoking, BMI, height, heart rate, diabetes, and total and HDL cholesterol (Model 2). In a third level of adjustment, (i) mean daytime and nighttime SBP were also adjusted for mean clinic SBP, (ii) mean daytime and nighttime DBP were also adjusted for mean clinic DBP, (iii) SD_{dn} and ARV of SBP were adjusted for daytime SBP, and (iv) SD_{dn} and ARV of DBP were adjusted for daytime DBP. For the outcomes of nighttime SBP and DBP, dipping percent, morning surge, and SD_{dn} and ARV, a fourth level of adjustment also included poor sleep quality during ABPM.

The prevalence of sustained, masked, white coat, and nocturnal hypertension and BP nondipping were each calculated for Whites and African Americans and the prevalence ratio for these phenotypes in African Americans vs. Whites was determined using negative binomial regression models with adjustment as described for Models 1 and 2 above. A third model including additional adjustment for poor sleep quality during ABPM was also done for nocturnal hypertension and BP nondipping. Prevalence ratios are recommended *in lieu* of odds ratios for cross-sectional studies with common outcomes.[24] *P* values <0.05 were considered statistically significant. Analyses were conducted using SAS Version 9.3 (SAS Institute, Cary, NC).

RESULTS

Of the 316 CARDIA participants who completed ABPM, 281 participants (89%; 178 African Americans and 103 Whites) had 10+ daytime BP readings and 5+ nighttime BP readings and were included in our analyses. Characteristics of participants included and not included in the current

Table 1. Characteristics of White and African American CARDIA participants with ABPM data at the Year 5 study visit

	Whites (*n* = 103)	African Americans (*n* = 178)	*P* value
Age, years	30.8 (3.5)	29.8 (3.8)	0.02
Women, %	48.5	56.7	0.18
Formal education, years	14.3 (2.4)	13.8 (2.0)	0.04
Physical activity, exercise units	302.1 (237.2)	326.0 (283.4)	0.45
Current smoking, %	29.1	28.1	0.19
Body mass index, kg/m^2	25.7 (5.4)	27.0 (5.2)	0.046
Height, cm	170.4 (8.4)	170.0 (9.9)	0.73
Heart rate, beats per minute	79.1 (10.0)	82.1 (9.9)	0.02
Diabetes, %	1.9	2.8	0.65
Total cholesterol, mg/dl	184.1 (33.3)	176.4 (33.2)	0.06
HDL cholesterol, mg/dl	47.1 (12.8)	53.0 (14.1)	<0.001
Clinic blood pressure			
Systolic blood pressure, mm Hg	107.3 (10.5)	110.2 (10.6)	0.03
Diastolic blood pressure, mm Hg	71.6 (10.6)	74.0 (9.3)	0.06
Pulse pressure, mm Hg	35.3 (4.4)	34.8 (4.2)	0.27
Taking antihypertensive medication, %	0.0	1.7	0.30
Poor sleep quality during ABPM, %	20.4	13.5	0.13

Numbers are mean (SD) or percentage.

Abbreviations: ABPM, ambulatory blood pressure monitoring; CARDIA, Coronary Artery Risk Development in Young Adults; HDL, high-density lipoprotein.

study are presented in Supplementary Table 1. The mean age of Whites and African Americans was 30.8 (SD 3.5) and 29.8 (SD 3.8) years, respectively (Table 1). Compared with Whites, African Americans had a lower mean level of education and higher mean BMI, heart rate, and HDL cholesterol. Additionally, compared with their White counterparts, African Americans had higher mean clinic SBP. No Whites and 1.7% of African Americans (n = 3) were taking antihypertensive medication.

Mean daytime SBP was higher among African Americans compared with Whites (Table 2). This association was present after age–gender adjustment and further adjustment for education, physical activity, current smoking, BMI, height, heart rate, diabetes, and total and HDL cholesterol. After adjustment for clinic SBP, daytime SBP was 1.71 (SD 1.03) mm Hg higher among African Americans compared with Whites (P = 0.10). African American–White differences in daytime DBP were not statistically significant before or after multivariable adjustment. Nighttime SBP and DBP were higher among African Americans compared with Whites. After multivariable adjustment including clinic BP and poor sleep quality during ABPM, nighttime SBP and DBP were 4.83 (SD 1.11) mm Hg and 2.29 (SD 0.87) higher, respectively, among African Americans compared with Whites.

Among African Americans and Whites, the prevalence of sustained hypertension was 3.4% and 1.9%, respectively,

Table 2. Mean daytime and nighttime systolic and diastolic blood pressure for White and African American CARDIA participants at the Year 5 study visit

	Whites (n = 103)	African Americans (n = 178)	P value
Daytime			
Systolic blood pressure, mm Hg			
Mean (SD)	116.3 (9.7)	119.6 (10.5)	0.01
Adjusted difference			
Model 1	0 (ref.)	3.90 (1.18)	0.001
Model 2	0 (ref.)	3.09 (1.23)	0.01
Model 3a	0 (ref.)	1.71 (1.03)	0.10
Diastolic blood pressure, mm Hg			
Mean (SD)	69.4 (6.8)	70.9 (7.0)	0.09
Adjusted difference			
Model 1	0 (ref.)	2.07 (0.85)	0.02
Model 2	0 (ref.)	1.41 (0.87)	0.11
Model 3b	0 (ref.)	0.44 (0.77)	0.56
Nighttime			
Systolic blood pressure, mm Hg			
Mean (SD)	101.7 (10.0)	108.1 (11.0)	<0.001
Adjusted difference			
Model 1	0 (ref.)	7.11 (1.23)	<0.001
Model 2	0 (ref.)	5.97 (1.24)	<0.001
Model 3a	0 (ref.)	4.79 (1.11)	<0.001
Model 3a + poor sleep quality[a]	0 (ref.)	4.83 (1.11)	<0.001
Diastolic blood pressure, mm Hg			
Mean (SD)	56.9 (6.7)	60.2 (7.8)	<0.001
Adjusted difference			
Model 1	0 (ref.)	3.86 (0.90)	<0.001
Model 2a	0 (ref.)	3.19 (0.94)	<0.001
Model 3b	0 (ref.)	2.32 (0.86)	0.01
Model 3b + poor sleep quality[a]	0 (ref.)	2.29 (0.87)	0.01

Numbers are mean (SD) or percentage. Model 1 includes adjustment for age and gender. Model 2 includes adjustment for age, gender, education, physical activity, current smoking, body mass index, height, heart rate, diabetes mellitus, total cholesterol, and high-density lipoprotein cholesterol. Model 3a includes variables in Model 2 with additional adjustment for mean clinic systolic blood pressure. Model 3b includes variables in Model 2 with additional adjustment for mean clinic diastolic blood pressure.

Abbreviation: CARDIA, Coronary Artery Risk Development in Young Adults.

[a]Poor sleep quality during ambulatory blood pressure monitoring.

and masked hypertension was 4.4% and 2.1%, respectively (Table 3). The prevalence ratios of sustained and masked hypertension for African Americans compared with Whites were not statistically significant after multivariable adjustment. The prevalence of white coat hypertension was 3.3% for African Americans and 3.9% among Whites and differences were not statistically significant before or after multivariable adjustment. Overall, 16.9% of African Americans and 8.7% of Whites had nocturnal hypertension ($P = 0.06$). The African American–White prevalence ratio for nocturnal hypertension was 2.52 (95% CI: 1.12–5.68) after age–gender adjustment and 2.44 (95% CI: 0.99–6.05) after further adjustment for education, physical activity, current smoking, BMI, height, heart rate, diabetes, total and HDL cholesterol, and poor sleep quality during ABPM.

The nighttime dip in SBP (i.e., relative decline in SBP) on ABPM was 0.90 (SD 0.06) in African Americans compared with 0.87 (SD 0.06) in Whites (Table 4; $P < 0.001$). African Americans had less dipping than Whites with 48.3% of African Americans and 27.2% of Whites being nondippers ($P < 0.001$). African Americans were more likely than Whites to be nondippers after age–gender and multivariable adjustment. Morning surge was smaller in African Americans (15.2 (SD 11.8) mm Hg) compared with Whites (18.4 (SD 10.0) mm Hg; $P = 0.02$). After age–gender and multivariable adjustment, the difference in morning surge was not statistically significant.

SD_{dn} of SBP was higher among African Americans compared with Whites (Table 5; $P < 0.001$). This association remained statistically significant after initial adjustment (Models 1 and 2) but was no longer statistically significant after further adjustment for daytime SBP. ARV of SBP was higher for Africans Americans compared with Whites before and after multivariable adjustment. Additionally, SD_{dn} and ARV of DBP were higher in African Americans compared with Whites before and after multivariable adjustment.

DISCUSSION

In the present study, levels of several ABPM measurements and prevalence of abnormal phenotypes were higher among African American compared with White young adults. Mean nighttime SBP and measures of BP variability were each higher, and nocturnal hypertension and nondipping were more common among African Americans compared with Whites. Although not statistically significant, African Americans had higher daytime SBP and were two times more likely than Whites to have masked hypertension.

Table 3. Prevalence and prevalence ratios for sustained, masked, white coat, and nocturnal hypertension comparing African American and White CARDIA participants

	White (*n* = 103)	African American (*n* = 178)	*P* value
Sustained hypertension			
Prevalence, %	1.9	3.4	0.72
Prevalence ratio			
Model 1	1 (ref.)	2.21 (0.43,11.45)	0.35
Model 2	1 (ref.)	3.19 (0.45, 22.82)	0.25
Masked hypertension			
Prevalence, %	2.1	4.4	0.49
Prevalence ratio			
Model 1	1 (ref.)	2.15 (0.43, 10.78)	0.35
Model 2	1 (ref.)	1.48 (0.25, 8.75)	0.67
White coat hypertension			
Prevalence, %	3.9	3.3	0.99
Prevalence ratio			
Model 1	1 (ref.)	1.11 (0.30, 4.16)	0.88
Model 2	1 (ref.)	1.55 (0.31, 7.73)	0.59
Nocturnal hypertension			
Prevalence, %	8.7	16.9	0.06
Prevalence ratio			
Model 1	1 (ref.)	2.52 (1.12, 5.68)	0.03
Model 2	1 (ref.)	2.39 (0.97, 5.88)	0.06
Model 2 + poor sleep quality[a]	1 (ref.)	2.44 (0.99, 6.05)	0.054

Model 1 includes adjustment for age and gender. Model 2 includes additional adjustment for education, physical activity, current smoking, body mass index, height, heart rate, diabetes mellitus, total cholesterol, and high-density lipoprotein cholesterol.

Abbreviation: CARDIA, Coronary Artery Risk Development in Young Adults.

[a]Poor sleep quality during ambulatory blood pressure monitoring.

Table 4. Dipping and nondipping pattern and morning surge comparing African American and White CARDIA participants

	White (*n* = 103)	African American (*n* = 178)	*P* value
Dipping, %			
Mean (SD)	0.87 (0.06)	0.90 (0.06)	<0.001
Adjusted difference			
Model 1	0 (ref.)	0.03 (0.01)	<0.001
Model 2	0 (ref.)	0.03 (0.01)	<0.001
Model 2 + poor sleep quality[a]	0 (ref.)	0.03 (0.01)	<0.001
Nondipping pattern			
Prevalence	27.2	48.3	<0.001
Prevalence ratio			
Model 1	1 (ref.)	2.70 (1.58, 4.62)	<0.001
Model 2	1 (ref.)	2.52 (1.40, 4.51)	0.002
Model 2 + poor sleep quality[a]	0 (ref.)	2.50 (1.39 – 4.48)	0.002
Morning surge, mm Hg			
Mean (SD)	18.4 (10.0)	15.2 (11.8)	0.02
Adjusted difference			
Model 1	0 (ref.)	-2.26 (1.40)	0.06
Model 2	0 (ref.)	-2.26 (1.52)	0.14
Model 2 + poor sleep quality[a]	0 (ref.)	-2.40 (1.52)	0.12

Model 1 includes adjustment for age and gender. Model 2 includes additional adjustment for education, physical activity, current smoking, body mass index, height, heart rate, diabetes mellitus, total cholesterol and high-density lipoprotein cholesterol.

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In contrast, morning surge was smaller among African Americans. The data from the current study suggest that substantial racial differences on ABPM, particularly nighttime SBP, nocturnal hypertension, and BP variability, may exist between African Americans and Whites.

Several years ago, the concept of "true" or mean BP was introduced and conceptualized as the mean level of BP over a long period of time.[25] While clinic BP is often regarded as a proxy for an individual's true BP, several studies have found substantial discrepancies between clinic BP and true BP with only a moderate correlation existing between mean clinic BP and daytime BP ($r < 0.50$).[26] Importantly, several studies have found mean BP on ABPM maintains a strong association with CVD events independent of clinic BP.[27] In the current analysis, African Americans had higher clinic BP compared with Whites. Although not statistically significant after adjustment for clinic BP, daytime SBP was 1.71 mm Hg higher in African Americans than Whites.

Mean nighttime SBP and DBP were significantly higher among African Americans compared with Whites even after full multivariable adjustment including clinic BP. These data are consistent with prior studies that have reported higher SBP and DBP levels and a higher prevalence of nocturnal hypertension and nondipping for African Americans compared with Whites.[28,29] In a meta-analysis investigating racial differences in nighttime BP, expressed using standardized mean differences, African Americans had higher mean nighttime SBP and DBP compared with Whites.[30] This meta-analysis also reported less dipping among African Americans compared with Whites. However, few studies included in the meta-analysis were population-based, limiting the generalizability of the findings. A prior study reported a higher prevalence of nondipping among Black Hispanics (69.4%) compared with White Hispanics (52.7%).[31] The current study extends prior research with a larger sample size and examination of ABPM measures in a population-based sample.

Among the different ABPM measures we studied, morning surge was the only one that was smaller for African Americans compared with Whites. The smaller morning surge among African Americans may be a direct result from the substantially higher nighttime BP among African Americans. Also, compared with prior studies, the magnitude of the morning surge was small for both Whites and African Americans.[21,32] In a prior analysis of IDACO, the risk for CVD was increased only for participants with morning surge ≥37 mm Hg vs. <37 mm Hg. The relatively low morning surge in the current analysis may reflect the young age of participants. Small artery disease, endothelial dysfunction, arterial stiffness, and orthostatic hypertension are each more common at older age and have been associated with higher morning surge.[33,34] Whether middle-aged and older African Americans have higher morning surge than Whites should be evaluated in future studies.

Beyond diurnal patterns in BP, BP variability across a 24-hour period has emerged as an ABPM measure with

Table 5. Ambulatory blood pressure variability for African American and White CARDIA participants

	Whites (*n* = 103)	African Americans (*n* = 178)	*P* value
Systolic blood pressure			
Day–night SD, mm Hg			
Mean, mm Hg	9.9 (2.0)	10.9 (2.7)	<0.001
Model 1	0 (ref.)	1.12 (0.29)	<0.001
Model 2	0 (ref.)	0.73 (0.31)	0.02
Model 3a	0 (ref.)	0.53 (0.30)	0.08
Model 3a + poor sleep quality[a]	0 (ref.)	0.55 (0.30)	0.07
Average real variability, mm Hg			
Mean, mm Hg	9.0 (2.0)	10.5 (2.9)	<0.001
Model 1	0 (ref.)	1.58 (0.32)	<0.001
Model 2	0 (ref.)	1.29 (0.34)	<0.001
Model 3a	0 (ref.)	1.14 (0.34)	<0.001
Model 3a + poor sleep quality[a]	0 (ref.)	1.16 (0.34)	<0.001
Diastolic blood pressure			
Day–night SD, mm Hg			
Mean, mm Hg	9.3 (1.8)	10.5 (2.2)	<0.001
Model 1	0 (ref.)	1.19 (0.26)	<0.001
Model 2	0 (ref.)	0.91 (0.27)	<0.001
Model 3b	0 (ref.)	0.80 (0.26)	0.003
Model 3b + poor sleep quality[a]	0 (ref.)	0.80 (0.26)	0.003
Average real variability, mm Hg			
Mean, mm Hg	8.5 (2.1)	10.1 (3.1)	<0.001
Model 1	0 (ref.)	1.65 (0.35)	<0.001
Model 2	0 (ref.)	1.34 (0.37)	<0.001
Model 3b	0 (ref.)	1.24 (0.37)	<0.001
Model 3b + poor sleep quality[a]	0 (ref.)	1.23 (0.37)	<0.001

Model 1 includes adjustment for age and gender. Model 2 includes additional adjustment for education, physical activity, current smoking, body mass index, height, heart rate, diabetes mellitus, total cholesterol and high-density lipoprotein cholesterol. Model 3a includes variables in Model 2 with additional adjustment for mean systolic blood pressure on daytime ambulatory blood pressure monitoring. Model 3b includes variables in Model 2 with additional adjustment for mean diastolic blood pressure on daytime ambulatory blood pressure monitoring.

Abbreviation: CARDIA, Coronary Artery Risk Development in Young Adults.

[a]Poor sleep quality during ambulatory blood pressure monitoring.

prognostic importance for CVD risk.[4] In a meta-analysis of 8,938 participants from 11 studies, ARV of SBP was associated with all-cause and cardiovascular mortality, CVD incidence, and stroke but not cardiac or coronary events.[4] Also, SD_{dn} was associated with all-cause mortality. However, this meta-analysis did not have any data on African Americans. In the current study, African Americans had a significantly higher ARV of SBP and SD_{dn} and ARV of DBP compared with Whites even after multivariable adjustment.

It has been hypothesized that a higher prevalence of several abnormal ABPM measures among African Americans compared with Whites may explain the excess CVD risk they experience.[32] Nondipping patterns, nocturnal hypertension, and higher BP variability, each of which are associated with increased CVD risk, were higher among African Americans compared with Whites.[4–6] ABPM was only performed in a subset of CARDIA participants, precluding assessing their association with CVD outcomes. Future studies are needed to better understand whether the racial differences in ABPM phenotypes identified in the current study contribute to the higher risk for CVD experienced by African Americans compared with Whites.

There are several potential mechanisms underlying the higher levels of ambulatory BP measures and prevalence of abnormal ABPM phenotypes in African Americans compared with Whites. Sodium sensitivity, sleep apnea, poor sleep quality, and socioeconomic factors have each been associated with abnormal ABPM phenotypes and are more common in African Americans compared with Whites. Additionally, African Americans are more likely than Whites to have abnormal autonomic function, abnormal baroreflex function, and altered sodium

excretion.[9,10,35–37] Also, insulin resistance was reported to modify the association between the 24-hour sodium/potassium ratio and several ABP measures, including nocturnal BP, in a study of individuals of black African descent.[38] BP on ABPM may be adversely affected by psychological factors including higher levels of stress, anxiety, and discrimination which are experienced disproportionally by African Americans. The association between psychological factors and ABP measures may be cyclical. In a prior study, being labeled as hypertensive was associated with greater depressive symptoms and poorer mental health among African Americans but no association was present in Whites.[39] In a study by Spruill *et al.*,[28] not being married and having less education explained 36% of the African American–White difference in dipping. Future studies, with larger sample sizes, are needed to determine the explanatory factors underlying racial differences in abnormal ABPM phenotypes.

Strengths of the CARDIA study include the large population-based sample of African Americans and Whites, collection of ABPM following a standardized protocol, and extensive data collection that allowed us to adjust for several potential confounders. Additionally, the current analysis included African American and White participants aged 23–35 years at the time of their ABPM. Hypertension has its origins in childhood and young adulthood and we were able to examine differences in ABPM between African Americans and Whites prior to the development of hypertension or vascular changes such as endothelial dysfunction or arterial stiffness, consistent with the onset of hypertension. Despite the strengths of the study, the current results need to be interpreted in the context of known and potential limitations. Only one ABPM was available and the reproducibility of some measures is modest.[40,41] Although CARDIA enrolled several thousand participants, a subsample was invited to complete the ABPM and data were available for only 281 participants at one site. Possibly due to the modest sample size of participants with ABPM data, several results were not statistically significant. Also, given the modest sample size, we were not able to evaluate factors explaining the racial differences in the ABPM phenotypes.

In conclusion, in a young adult population-based sample, compared with Whites, African Americans were more likely to have several abnormal ABPM measures and phenotypes that are associated with an increased CVD risk and mortality. Nighttime SBP and DBP, nocturnal hypertension, nondipping, and BP variability were each higher or more prevalent among African Americans. Larger population-based studies are needed to confirm our results and investigate whether abnormal ABPM phenotypes explain the higher risk for CVD among African Americans compared with Whites.

SUPPLEMENTARY MATERIAL

Supplementary materials are available at *American Journal of Hypertension* (http://ajh.oxfordjournals.org).

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DISCLOSURE

A.J.V. has served on the medical advisory board for Suntech Medical (Morrisville, NC), manufacturer of a brand of ambulatory blood pressure monitor. No other authors have conflicts of interest to report.

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