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Diurnal blood pressure pattern and development of prehypertension or hypertension in young adults: the CARDIA study

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Abstract

Nondippers (people whose sleep systolic blood pressure (SBP) fails to decrease >10% from daytime SBP) have increased risk of cardiovascular disease. The prevalence of nondipping in younger adults has not been well-studied, nor has its value for predicting hypertension. We examined the prevalence of nondipping in a sub-study of the Coronary Artery Risk Development in Young Adults (CARDIA) Study. We used Cox regression to estimate the hazard ratio (HR) conferred by nondipping for incident prehypertension or hypertension (preHTN/HTN) over 15 years. Of the 264 non-hypertensive participants at baseline, 118 (45%) were nondippers. Blacks were more likely than Whites to be nondippers (52% vs 33%, $p=0.004$). The incidence rate of preHTN/HTN was 29.2/1000 person-years among dippers and 36.2/1000 person-years among nondippers. Compared to those in the lowest quartile of nighttime to daytime SBP, those in the highest quartile were more likely to develop preHTN/HTN (HR 1.61; $p=0.06$), but this relationship was attenuated after adjustment (HR 1.34; $p=0.27$). Our results demonstrate that nondipping is common in young, nonhypertensive adults, and is more common in Blacks than Whites. Nondipping might predate a meaningful clinically detected increase in BP in some people, but more research in larger study samples is needed.

Keywords

diurnal blood pressure; blood pressure dipping; hypertension

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Introduction

Over the last two decades, through the use of 24-hour ambulatory blood pressure monitoring, the importance of the diurnal blood pressure (BP) pattern has been more clearly elucidated.¹⁻⁷ Normally during sleep, BP decreases (“dips”) more than 10% from daytime baseline BP. Individuals who do not exhibit this >10% decrease have been referred to as nondippers. In patients with hypertension, nondipping has been shown to be associated with target organ damage and cardiovascular events.¹⁻⁶ In the general population, nondipping may be a risk factor for cardiovascular disease mortality independent of the overall BP during a 24-hour period.⁶ In one study of normotensive individuals (defined as clinic BP <140/90 mm Hg and 24-hour ambulatory BP average <125/80 mm Hg), the nondipping pattern was shown to be associated with cardiac hypertrophy and remodeling.⁷

Not only does the nondipping pattern appear to be associated with an increased cardiovascular risk, but it is also common. Nondipping was recently shown to be present in approximately 40% of untreated hypertensives and more than 50% of treated hypertensives.⁸ The nondipping pattern also occurs in approximately 30% of older normotensive adults.⁷ However, the prevalence of the nondipping pattern in younger adults who are not hypertensive has not been well studied. Also not known is whether nondipping in young normotensive adults is predictive of future hypertension.

In physiological terms, the sympathetic nervous system plays an important role in the pathogenesis of hypertension and of cardiovascular disease.⁹ Autonomic imbalances (increased sympathetic tone and decreased parasympathetic tone) lead to increased heart rate, vasoconstriction, and increased BP. Elevated BP, in turn, leads to an alteration in arterial and aortic baroreceptor function, and loss of sympathetic inhibition is one of the mechanisms responsible for failure of BP to dip during sleep.¹⁰ It is possible that nondipping is an early change in a person’s BP pattern, and the loss of nocturnal BP reduction may predate the onset of clinical hypertension.

The objectives of this study were (1) to estimate the prevalence of the nondipping BP pattern among young, non-hypertensive Black and White adults, and (2) to examine whether a nondipping BP pattern among young adults is associated with the development of prehypertension or hypertension. We hypothesized that young adults with a nondipping pattern would be more likely to develop prehypertension or hypertension than those with a normal dipping pattern.

Methods

Overall Design

The Coronary Artery Risk Development in Young Adults Study (CARDIA) is an ongoing, prospective, epidemiologic study, conducted at four sites throughout the United States. A detailed description of the study design and recruitment methods can be found in an earlier publication.¹¹ The data presented in this paper are from a subgroup of individuals who participated in an ambulatory BP monitoring substudy in 1990 (year 5 of CARDIA) and had ongoing follow-up visits. The CARDIA study and this substudy were approved by the appropriate Institutional Review Boards (IRBs), and informed consent was obtained from each study participant. The plans for this secondary analysis were also submitted to the University of North Carolina’s Office of Human Research Ethics and deemed exempt from further review.

Study Participants

As previously described,¹² for feasibility reasons, ambulatory BP monitoring measurements were performed at only one of the CARDIA study sites (Birmingham, AL). A list of 100 people randomly chosen from each of the four race/gender subgroups (Black and White men and women) was generated at the CARDIA Coordinating Center. After excluding individuals whose jobs would make ambulatory BP monitoring difficult (e.g., truck drivers, delivery people), a total of 316 people (147 men and 169 women; 112 Whites and 204 Blacks) participated in the ambulatory BP monitoring sub-study.

Ambulatory Blood Pressure Monitoring

Ambulatory blood pressure was monitored over a 24 hour period using a Suntech Accutracker II (Suntech Medical, Morrisville, NC), using an appropriately sized cuff which was inflated approximately every 20 minutes.¹³ To reduce anticipation effects, the inflation schedule was variable. If any value outside preset limits (systolic ≥ 220 or ≤ 80 mm Hg; diastolic ≥ 130 or ≤ 40 mm Hg) was detected during a recording, that measurement was rejected and another measurement was immediately made. 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 also triggered a rejection and a new reading. Daytime was defined as the period between 0700 and 2359, and we required a minimum of 14 adequate measurements. Nighttime was defined as the period between 0000 and 0659, and we required a minimum of six adequate measurements. Blood pressure dipping at baseline was quantified as the ratio of mean nighttime systolic BP to mean daytime systolic BP. Nondipping pattern was defined as a ratio ≥ 0.90 .

Clinic Blood Pressure Measurements

Prior to study visits, participants were asked to refrain from smoking and heavy physical activity. At each visit, after five minutes of rest, three seated right arm BP measurements were recorded at one-minute intervals. Using an appropriately sized cuff, the first-phase and fifth-phase Korotkoff sounds were taken as the systolic and diastolic blood pressures, respectively, using a random zero sphygmomanometer. Individuals with hypertension at baseline (defined as on BP-lowering meds at CARDIA year 5) or CARDIA visit 5 clinic BP average (based on second and third readings) of either systolic ≥ 140 mm Hg or diastolic ≥ 90 mm Hg were excluded from this analysis, leaving 264 “at-risk” people in the final study sample. At the 15-year follow-up visit (CARDIA year 20), clinic BP was measured using a digital oscillometric monitor (Omron HEM-907XL; Online Fitness, Santa Monica, California) rather than a random zero sphygmomanometer. A calibration study was performed at year 20 in approximately 900 participants, and we used the formulae calibrated year 20 systolic blood pressure = $3.74 + 0.96 * \text{observed Omron systolic blood pressure}$ and calibrated year 20 diastolic blood pressure = $1.30 + 0.97 * \text{observed Omron systolic blood pressure}$.

Covariates

Education was determined by self report of highest grade level achieved. Body mass index (BMI) was calculated based on measured height and weight at the baseline visit. Diabetes mellitus was defined as a fasting glucose level ≥ 126 mg/dL or use of hypoglycemic agents at baseline (CARDIA year 5). Family history of hypertension and smoking status were determined by self-report. Alcohol consumption was a continuous variable (ethanol intake, mL/day). Physical activity was assessed by the CARDIA physical activity questionnaire, which measured the self-reported frequency of participation in 13 different categories of recreational sports and exercise in the past 12 months.¹⁴ Physical activity scores were computed by multiplying the frequency of participation by the intensity of activity and

reported as “exercise units.” Energy expenditure could not be computed because duration of activity was not queried.

Analysis

Our exposure variable was nondipping pattern based on ambulatory BP monitoring performed in 1990. Our outcome was clinical worsening of BP category—from optimal BP to incident prehypertension or hypertension, or from prehypertension to incident hypertension. Hypertension was defined as a clinic visit average (of second and third readings) systolic BP ≥ 140 mmHg or average diastolic BP ≥ 90 mmHg or interval initiation of an antihypertensive medication. Prehypertension was defined as a clinic visit BP average (of second and third readings) systolic BP ≥ 120 mm Hg or average diastolic BP ≥ 80 mm Hg with neither reading $\geq 140/90$ mm Hg. Time of follow-up was 15 years. In the survival analyses, a participant was censored when a change in BP category was detected (optimal BP, defined as systolic BP < 120 mm Hg and diastolic BP < 80 mm Hg, to prehypertension, prehypertension to hypertension, or optimal BP to hypertension). A categorization change from optimal BP to hypertension was counted as a change from optimal to hypertension whether or not the person had BP in the prehypertension range in the interim.

We calculated the crude hazard ratio and then performed a series of Cox regression analyses. In the first model, we adjusted only for race. In a second model, we adjusted for race and BMI. In a fully adjusted model, we included baseline clinic systolic BP as well as age, sex, race/ethnicity, BMI, diabetes mellitus, physical activity, family history of hypertension, alcohol consumption, and cigarette smoking.¹⁵ We also tested the significance of an interaction term of race and dipping status. We repeated the regression analyses using the ratio of nocturnal to daytime systolic BP as a categorical exposure based on quartiles as well as continuously.

Results

Characteristics of Sample

The mean age of participants at baseline was 30 years (Table 1). Forty five percent of participants were male, and 63% were Black. Few had known diabetes, but half were overweight or obese. Among this study sample, 28% were current smokers at baseline. Overall, there were 118 people (45%) with a nondipping BP pattern. This percentage included 11 people whose BP was actually higher at night—so-called “reverse dippers”. A total of 201 participants had optimal clinic BP at baseline. The remaining 63 participants had baseline clinic BP in the prehypertension range. Among those with optimal BP, 45% were nondippers, and among those with prehypertension, 44% were nondippers. Mean clinic BP among dippers and nondippers was similar at baseline, as was mean 24 hour average BP (Table 2). As expected, the mean nighttime BP was lower among the dippers (102/57 mm Hg vs 109/62 mm Hg). The associated BP changes from day to night reflect these differences. Interestingly, daytime BP average was slightly lower among nondippers (115/69 mm Hg vs 118/70 mm Hg).

Associations with Nondipping Status

Of all characteristics examined, Black race and obesity were the only factors associated with nondipping status (Table 3). Among White participants, 33% were nondippers/reverse dippers, while among Blacks, 52% were nondippers/reverse dippers ($p=0.004$). Among normal weight or overweight participants, 41% in each category were nondippers/reverse dippers, but among obese participants, 57% were nondippers/reverse dippers ($p=0.07$).

Incident Change in BP Category

Over the 15 years of follow-up, there were 128 incident cases of clinical worsening of BP category (81 incident cases of hypertension and 47 incident cases of prehypertension) (Table 4). The incidence rate of change in clinical BP category was 29.2 per 1000 person-years among the dippers and 36.2 per 1000 person-years among the nondippers/reverse dippers for a hazard ratio of 1.33 ($p=0.10$). When adjusted for race, the association was mitigated (HR 1.22; $p=0.27$). Additional adjustment for BMI further reduced the hazard ratio, but full adjustment with all covariates had little effect. The interaction of race and dipping status was not significant ($p=0.37$).

The ratio of baseline nighttime to daytime systolic BP ratio ranged from 0.736 to 1.19. Compared to those in the lowest quartile of nighttime to daytime systolic BP, those in the highest quartile had a hazard ratio of 1.61 ($p=0.06$) for change in clinical BP category (Table 5). When adjusted for race, the association persisted in the hypothesized direction, but the magnitude of the association was reduced, and the near-statistical significance of highest to lowest quartile was mitigated (HR 1.43; $p=0.17$). Further adjustment for BMI also mitigated the association. A fully adjusted model differed little from the model adjusted only for race and BMI. In a Cox regression model examining baseline nighttime to daytime systolic BP ratio as a continuous variable, an increase of 0.05 in the ratio was associated with a 5% increase (fully adjusted for covariates) in change in clinical BP category, but this was not significant (HR 1.05; $p=0.48$).

Discussion

Among a sample of young adults with non-hypertensive blood pressure at baseline, we found that the nondipping pattern was common, present in 33% of Whites and 52% of Blacks. We also found that over a follow-up period of 15 years, nocturnal BP dipping status was positively, but not significantly (after adjustment) predictive of incident prehypertension or hypertension. The first implication of our findings is that many young otherwise healthy adults, though not hypertensive, may have a diurnal pattern of BP that places them at increased risk of target organ damage and increased cardiovascular disease mortality. The second implication is that nondipping in younger adults might predate hypertension, suggesting an early (pre-clinical) autonomic imbalance as a contributor. However, this finding needs confirmation in larger studies.

Since older age has repeatedly been a factor associated with the nondipping status,⁶⁻⁸ we were somewhat surprised to find a high prevalence of the nondipping status in these young adults with non-hypertensive blood pressure. Previous studies have examined the prevalence of the nondipping pattern in various subgroups. Most such studies have been either in older people or in people with known hypertension. For example, one study of 183 unmedicated hypertensive patients showed the nondipping pattern to be present in 43% [16]. In a large international database containing ambulatory BP data on 7458 adults (mean age 57 years), nearly half of whom were taking BP-lowering drugs, 29% of participants were nondippers/reverse dippers.¹⁷ In a small study of older, normotensive Japanese adults, 34% were nondippers.⁷ A similar percentage (36%) was seen in a study of 1542 adults with a mean age of 61 years and representative of the general population of older adults in Japan.⁶ In more a recent cross-sectional study of 8383 untreated and 34,563 treated hypertensives, the nondipping pattern was present in 41% and 53%, respectively.⁸ In the HARVEST study, among never-treated borderline to mild young hypertensives ($n=1062$), nocturnal BP nondipping was present in 31%.¹⁸ One study of a British population published in 1998 found that among 265 healthy, normotensive volunteers who underwent office and ambulatory BP measurements, 17% were nondippers.¹⁹ Compared to the participants in our

study, those in the British study were slightly older with a mean age of 39 years. Most importantly, however, all of the participants in that study were White.

In our study, Black race was the one factor significantly associated with nondipping status. Similar findings have been reported previously.²⁰⁻²¹ In a study of 171 somewhat older (mean age of 53 years) Black and White normotensive and mildly hypertensive adults, 54% of Blacks and 39% of Whites were nondippers.²⁰ The observation of greater likelihood of nondipping among Blacks, even at earlier ages as shown in our study, is important and may partially explain the greater severity of hypertension-related target organ damage and resultant cardiovascular and renal disease in Blacks compared to Whites.^{22,23}

We hypothesized that young non-hypertensive adults with a nondipping pattern would be more likely to develop a change in their clinical BP category, and our findings are suggestive that this is the case. Although two-sided p-values were not significant, the findings are in the hypothesized direction, and one-sided p-values would have reached statistical significance in the crude analyses. These interesting results should therefore not be readily dismissed as due to chance, and further studies are warranted. There are few other studies to date that have examined this possibility. In the HARVEST study, mentioned above, nocturnal BP nondipping was a strong predictor of sustained clinic hypertension after a mean of 68 months of follow-up.¹⁸ An important difference from our study was that the participants in the HARVEST study already had elevated clinic BP at baseline, and many began antihypertensive treatment over the course of follow-up.

In making the case of the potential clinical significance of our findings, we have assumed that the definition and prognosis of the nondipping pattern is the same in young, healthy, normotensive adults as in middle-aged, older, and/or hypertensive adults. An alternative assessment of our findings is that people who have optimal or near-optimal daytime BP do not exhibit a 10% nocturnal dip. In other words, it may be “normal” for people with optimal (or “low”) daytime BP not to dip as much at night. Our interesting finding that average daytime BP among nondippers was actually lower than that of dippers while 24-hour averages were very similar between the two groups further supports this additional new hypothesis. One study previously demonstrated that even in normotensive individuals, the adjusted risk of cardiovascular mortality among nondippers is over twice that of dippers.⁶ However, average daytime BP in that study sample was 124/73 mm Hg compared to the average of 117/70 mm Hg in this study.

While our study had a reasonably long follow-up period over which to detect changes in clinical BP status, it was limited by a relatively small sample size and therefore may have been underpowered to detect a significant difference in prehypertension and hypertension rates. We must also acknowledge the possibility of misclassification bias in the measurement of nondipping status. Classifying patients into dippers or nondippers based on one ambulatory BP monitoring may be misleading and related to the quality of sleep the person experiences during the session.²⁴ However, if misclassification bias did occur in this study, it would be nondifferential and cause the magnitude of association we found to actually be smaller than the true association. One study that used three ambulatory BP monitoring sessions to classify participants found that while associations with dipping status based on the initial session were somewhat weaker than associations based on all three, the patterns were similar.²⁰

Conclusions

The nondipping BP pattern is common in young adults with normal BP (prehypertension as well as optimal BP). This finding has clinical significance because research to date has

shown that nondipping is associated with cardiovascular target organ damage and mortality independent of elevated BP. Blacks, even at younger ages and when normotensive, have a higher prevalence than Whites of nondipping, a finding that may partially explain their greater hypertension-related morbidity. Nondipping might predate clinical hypertension in some people, but further studies with larger sample sizes are needed to clarify this hypothesis. More importantly, if it is shown in future studies that reduction of nocturnal BP in nondippers delays hypertension onset or leads to reduced target organ damage and cardiovascular disease events, more routine use of 24-hour ambulatory BP monitoring for screening would need to be considered.

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Table 1

Characteristics of Sample at Baseline (CARDIA Year 5) (N=264)

Age, mean (SD), y	30.0 (3.7)
Age categories, %	
23-29 y	45.5
30-36 y	54.5
Male, %	45.1
Race categories, %	
White	36.7
Black	63.3
Education, mean (SD), y	14.0 (2.1)
BMI, mean (SD)	26.3 (5.2)
BMI categories, %	
Normal (<25 kg/m ²)	44.7
Overweight (25-29 kg/m ²)	30.7
Obese (≥ 30 kg/m ²)	24.6
Known diabetes, %	2.0
Family history of hypertension, %	58.3
Smoker, %	
Never smoker	62.5
Former smoker	9.5
Current smoker	28.0
Alcohol consumption, mL/day mean (SD)	11.2 (22.9)
Physical activity, Kcal/Day, mean (SD)	320.0 (266.9)

BMI, body mass index

Table 2

Blood Pressure Characteristics of Participants at Baseline (CARDIA year 5)

Characteristic	Dippers (N=146)	Non Dippers (N=118)	p-value
	Mean±SD	Mean±SD	
Clinic BP			
SBP	108.0 (8.7)	106.9 (9.6)	0.31
DBP	71.6 (9.0)	71.8 (8.2)	0.83
Ambulatory BP			
24-h average SBP	114.6 (9.2)	113.9 (8.9)	0.59
24-h average DBP	67.3 (6.2)	67.1 (5.7)	0.77
Average daytime SBP	118.3 (9.6)	115.2 (8.9)	0.008
Average daytime DBP	70.4 (6.7)	68.7 (5.8)	0.03
Average nighttime SBP	101.7 (8.7)	109.3 (10.1)	<0.0001
Average nighttime DBP	56.8 (6.1)	62.0 (7.3)	<0.0001
Change in SBP, day - night	16.6 (3.9)	5.9 (5.1)	<0.0001
Change in DBP, day - night	13.6 (4.6)	6.7 (5.6)	<0.0001

Table 3

Characteristics Associated with Nondipping Status in Young Adults (CARDIA Year 5)

	n/N	Percent	Mean (SD)	p-value
Age categories				0.51
2329 y	51/120	42.5		
3036 y	67/144	46.5		
Sex				0.65
Male	55/119	46.2		
Female	63/145	43.5		
Race				0.004
White	32/97	33.0		
Black	86/167	51.5		
Education level				0.89
< High school	5/12	41.7		
High school graduate	37/79	46.8		
At least some college	76/173	43.9		
BMI categories				0.07
Normal (<25 kg/m ²)	48/118	40.7		
Overweight (25-29 kg/m ²)	33/81	40.7		
Obese (≥ 30 kg/m ²)	37/65	56.9		
Known diabetes				0.49
Yes	3/5	60.0		
No	115/259	44.4		
Family history of hypertension				0.97
Yes	69/154	44.8		
No	49/110	44.6		
Smoker				0.22
Never smoker	69/165	41.8		
Former/current smoker	49/99	49.5		
Alcohol consumption, mL/day				0.65
Nondippers (n=118)	-	-	11.9 (23.9)	
Dippers (n=146)	-	-	10.6 (22.0)	
Physical activity, Kcal/day				0.82
Nondippers (n=118)	-	-	316.0 (241.0)	
Dippers (n=146)	-	-	323.3 (286.9)	

BMI, body mass index; Dipper = mean nighttime:mean daytime systolic BP ratio of <0.90; Nondipper = mean nighttime:mean daytime systolic BP ratio ≥0.90

Table 4

Incidences of Pre-Hypertension or Hypertension by Baseline Nocturnal BP Pattern (N=264)

	Dippers	Non-/Reverse Dippers	p-value
Crude			
No. of cases/Total	64/146	64/118	
Rate (No. per 1000 person-years)	29.2	36.2	
HR (95% CI)	1.0	1.33	0.10
Race-adjusted			
HR (95% CI)	1.0	1.22	0.27
Adjusted for race and BMI			
HR ((95% CI)	1.0	1.13	0.50
Fully adjusted **			
HR (95% CI)	1.0	1.15	0.45

HR, hazard ratio

Incident prehypertension was defined as clinic BP $\geq 120/80$ mm Hg. Incident hypertension was defined as clinic BP $\geq 140/90$ mm Hg or taking antihypertensive medication at any of CARDIA years 7, 10, 15 or 20. Those taking BP meds or with a BP $\geq 140/90$ mm Hg at CARDIA year 5 (CARDIA Year 5 is baseline for this analysis) were excluded. An "event" occurred if a change in BP category was detected (optimal BP to prehypertension, prehypertension to hypertension, or optimal BP to hypertension).

** Adjusted for age, sex, race, baseline clinic systolic BP, BMI, diabetes, family history of hypertension, smoking, alcohol intake, an physical activity

Table 5

Incidence of Pre-Hypertension or Hypertension by Quartile of Baseline Mean Nighttime to Daytime Systolic BP Ratio (N=264)

	HR	p-value
Crude		
Trend*	1.12	0.10
Q1	1.00 (ref)	
Q2	1.20	0.50
Q3	1.38	0.20
Q4	1.61	0.06
Race-adjusted		
Trend	1.08	0.29
Q1	1.00 (ref)	
Q2	1.14	0.63
Q3	1.24	0.42
Q4	1.43	0.71
Adjusted for race and BMI		
Trend	1.04	0.63
Q1	1.00 (ref)	
Q2	1.01	0.96
Q3	1.09	0.75
Q4	1.26	0.38
Multivariable-adjusted**		
Trend	1.05	0.48
Q1	1.00 (ref)	
Q2	1.09	0.76
Q3	1.13	0.65
Q4	1.34	0.27

HR, hazard ratio; ref, referent

An "event" occurred if a change in BP category was detected (optimal BP to prehypertension, prehypertension to hypertension, or optimal BP to hypertension).

Quartiles of mean nocturnal systolic BP/mean diurnal systolic BP ratio: **Q1:** 0.7360-0.8621 (n=66); **Q2:** 0.8624-0.8926 (n=66); **Q3:** 0.8934-0.9385 (n=66); **Q4:** 0.9388-1.1855 (n=66)

* P for trend based on continuous ratio variable

** Adjusted for age, sex, race, BMI, diabetes, family history of hypertension, smoking, alcohol intake, physical activity, and baseline clinic systolic BP