

Association of Usual Sleep Duration With Hypertension: The Sleep Heart Health Study

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Study Objectives: Limited experimental data suggest that sleep restriction acutely elevates blood pressure; however, little is known about the relationship between usual sleep duration and hypertension. This study assesses the relationship between usual sleep duration and hypertension in a community-based cohort.

Design: Cross-sectional observational study.

Setting: The Sleep Heart Health Study, a community-based prospective study of the cardiovascular consequences of sleep-disordered breathing.

Participants: Two thousand eight hundred thirteen men and 3097 women, aged 40 to 100 years.

Interventions: None.

Measurements and Results: Usual weekday and weekend sleep durations were obtained by questionnaire, and their weighted average were categorized as less than 6, 6 to less than 7, 7 to less than 8, 8 to less than 9, and 9 or more hours per night. Hypertension was defined as a systolic blood pressure of 140 mm Hg or greater, a diastolic blood pressure of 90 mm Hg or greater, or use of medication to treat hypertension. The relationship between sleep duration and hypertension was examined using categorical logistic regression with adjustment for age, sex, race, apnea-

hypopnea index, and body mass index. Compared to subjects sleeping 7 to less than 8 hours per night, those sleeping less than 6 and between 6 and 7 hours per night had adjusted odds ratios for hypertension of 1.66 (95% confidence interval 1.35-2.04) and 1.19 (1.02-1.39), respectively, whereas those sleeping between 8 and 9 and 9 or more hours per night had adjusted odds ratios for hypertension of 1.19 (1.04-1.37) and 1.30 (1.04-1.62), respectively ($p < .0001$ for association of sleep duration with hypertension). These associations persisted when analyses were further adjusted for caffeine and alcohol consumption, current smoking, insomnia symptoms, depression symptoms, sleep efficiency, and prevalent diabetes mellitus or cardiovascular disease.

Conclusions: Usual sleep duration above or below the median of 7 to less than 8 hours per night is associated with an increased prevalence of hypertension, particularly at the extreme of less than 6 hours per night.

Keywords: Hypertension, sleep deprivation, epidemiology

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INTRODUCTION

TYPICAL DAILY SLEEP DURATION HAS BEEN DECLINING AMONG ADULTS IN THE UNITED STATES FOR MORE THAN A GENERATION, WITH MEDIAN SLEEP duration falling from 8 hours per night in the 1950s to 7 hours per night in recent years, with more than one-third now sleeping fewer than 7 hours per night.^{1,2} Much of this reduction in sleep duration reflects voluntary sleep restriction, with nearly half of individuals reporting that they restrict sleep in order to watch television, use the Internet, or work.³ Several studies have found higher rates of mortality or coronary heart disease with both long (>8 hours/night) and short (<7 hours/night) usual sleep durations.⁴⁻⁹ Short-term experimental sleep restriction for as little as 1 night has been reported to increase blood pressure in both healthy¹⁰⁻¹² and hyper-

tensive¹³ subjects. Because hypertension carries a high risk for cardiovascular disease, an effect of short sleep duration on hypertension might increase the risk of cardiovascular disease and mortality. In the present study, we examined the relationship between self-reported usual sleep duration and prevalent hypertension in a large, community-based sample of middle-aged and older adults.

METHODS

Study Sample

Subjects were participants in the Sleep Heart Health Study (SHHS), a community-based, prospective cohort study of the cardiovascular consequences of obstructive sleep apnea/hypopnea (OSAH).¹⁴ Although not the primary aim of the SHHS, the present analysis takes advantage of data on sleep duration, hypertension, and relevant covariates collected as part of the baseline SHHS examination. All SHHS participants completed a Sleep Habits Questionnaire (SHQ) and underwent overnight polysomnography between 1995 and 1998. Medication use was ascertained at the time of polysomnography, and blood pressure was measured at the time of polysomnography or, in fewer than 5% of subjects, at a recently preceding clinic visit. Subjects in the present analysis are SHHS participants for whom hypertension status was determined within 1 year of ascertainment of usual sleep duration, who also had complete data for age, sex, race, body mass index (BMI), and apnea-hypopnea index (AHI). Of the 6441 SHHS participants, 531 (8.2%) were excluded for missing data (listed hierarchically: 124 missing sleep duration, 21 missing hypertension

Disclosure Statement

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status, 284 with the timing of hypertension assessment unknown or more than 1 year before or after the assessment of sleep duration, and 102 missing BMI). Excluded subjects were somewhat older ($66.5 \pm \text{SD } 13.4$ vs 63.1 ± 10.7 years), had lower mean BMI (27.8 ± 5.1 vs 28.6 ± 5.4 kg/m²), were more likely to be women (57% vs 52%), and were more likely to identify themselves as a member of a minority race or ethnicity (34% vs 22%). Inclusion of the 284 subjects excluded on the basis of the interval between assessments of sleep duration and hypertension had no meaningful impact on the main analysis. The protocol was approved by the Institutional Review Board of each participating center, and signed informed consent was provided by each subject.

Sleep Habits Questionnaire

Usual sleep duration on weekdays was defined as the response to the question, "How many hours of sleep do you usually get at night (or your main sleep period) on weekdays or workdays?" Responses were integer values. A similar question was used to define usual weekend sleep duration. The stability of these measures over time in SHHS participants has been reported.¹⁵ Usual daily sleep duration was calculated as a weighted average of weekday and weekend sleep durations, using the formula: $([\{\text{usual weekday sleep duration}\} \times 5] + [\{\text{usual weekend sleep duration}\} \times 2]) / 7$. In the 4117 subjects whose entire sleep period was recorded during home polysomnography, mean polysomnographic total sleep time was shorter than self-reported usual sleep duration (5.9 vs 7.1 hours). The 2 measures were weakly, although significantly, correlated ($r = 0.17$, $p < .0001$).

Symptoms of insomnia were obtained from responses on a 5-point Likert scale to the items "Have trouble falling asleep," "Wake up during the night and have difficulty getting back to sleep," "Wake up too early in the morning and be unable to get back to sleep," and "Take sleeping pills or other medication to help you sleep." Response options were Never, Rarely (1/month or less), Sometimes (2-4/month), Often (5-15/month), and Almost Always (16-30/month). For analysis, these variables were collapsed into 2 categories: Infrequent, comprising the responses Never, Rarely, and Sometimes; and Frequent, comprising the responses Often and Almost Always. Insomnia was operationally defined as a "frequent" response to any of these 4 questions.

Hypertension

Blood pressure was measured at an evening visit to the participant's home. After at least 5 minutes of rest, 3 seated blood-pressure measurements were made at 5-minute intervals with the subject seated, using a mercury gauge sphygmomanometer and recording systolic (SBP) and diastolic (DBP) blood pressure to the nearest 2 mm Hg, according to a standardized protocol that has been previously reported.¹⁶ For this analysis, SBP and DBP were obtained by taking the mean of the second and third blood-pressure measurements. Use of antihypertensive medications was obtained via a standardized health interview administered on the evening of the home visit. Hypertension was defined as SBP ≥ 140 or DBP ≥ 90 mm Hg or current use of antihypertensive medication.¹⁷

Other Measures

Waist girth (measured at the level of the umbilicus) and height

were ascertained by each parent cohort at a regularly scheduled clinic examination. BMI was calculated using height measured in the clinic and weight measured at the time of the home visit or, in some cases, at a clinic visit if scheduled within 1 month of the home visit ($n=334$). Usual daily alcohol consumption was also ascertained by each parent cohort. Unattended, in-home polysomnography was performed on the night of the home visit, and records were centrally scored. The AHI was defined as the number of apneas plus hypopneas, each associated with at least a 4% decrease in oxyhemoglobin saturation, per hour of sleep.¹⁸ A standardized health interview administered prior to polysomnography was used to obtain data on smoking habits; daily caffeine consumption; physician-diagnosed angina, myocardial infarction, stroke, or heart failure; and history of coronary revascularization procedures. Resting heart rate was recorded at the time of polysomnography set-up. Symptoms of depression were obtained from 2 questions on the Medical Outcomes Study 36-item short-form health survey¹⁹: "During the past 4 weeks, how much of the time..." (1) "Have you felt so down in the dumps that nothing could cheer you up?" and (2) "Have you felt downhearted and blue?" Responses from the 6-point Likert scale were collapsed into 2 categories: "none," "a little," or "some" of the time versus "all," "most," or "a good bit" of the time. Current medication use and blood pressure were ascertained by reviewing actual medication containers and coding each medication into standardized classes, as previously described.²⁰

Statistical Analysis

Unadjusted differences in continuous and categorical variables across sleep-duration categories were assessed for significance using single-factor analysis of variance or contingency table analysis, as appropriate. General categorical logistic regression analysis was implemented using PROC CATMOD in SAS (SAS version 8.1, SAS Institute, Cary, NC) to assess the relation of usual sleep duration to hypertension, adjusting for relevant covariates. Covariates included in the main model were age, BMI, and AHI as continuous measures and sex and race as categorical variables. Waist girth was also considered but was not included in the main model because it was not a significant predictor when BMI was included and had no meaningful impact on the outcome of interest. Additional analyses assessed the impact of caffeine and alcohol consumption, smoking status, depressive symptoms, insomnia symptoms, and prevalent cardiovascular disease on the association of sleep duration with hypertension. Secondary analyses stratified on sex, age, AHI, and presence of insomnia symptoms were performed to assess the consistency of results across the study sample and to assess the contribution of insomnia to the observed effects.

RESULTS

Among the 5910 subjects (2813 men, 3097 women), the mean age was 63.1 years (SD 10.7, range 40-100), BMI was 28.6 kg/m² (SD 5.4), and AHI was 8.8 (SD 12.4; median 4.4). Blood pressure was measured a median of 43 days after the assessment of usual sleep duration (interquartile range 1 to 88 days). Hypertension was identified on the basis of measured blood pressure or use of antihypertensive medication in 52.2% of subjects, 75.2% of whom were on antihypertensive medication. Median reported sleep duration was 7.0 hours per night. A usual sleep duration of less than

Table 1—Characteristics of the Study Sample^a

	Reported Usual Sleep Duration, h/night					p Value
	< 6	6 to < 7	7 to < 8	8 to < 9	≥ 9	
Number of subjects ^b	545	1204	2022	1689	450	
Age, y	63.8 (11.0)	62.4 (10.8)	61.9 (10.4)	63.8 (10.4)	67.2 (11.0)	< .001
BMI, kg/m ²	29.1 (6.5)	28.8 (5.5)	28.4 (5.1)	28.4 (5.3)	28.6 (5.6)	.02
AHI, events/hour	10.1 (14.5)	8.7 (12.2)	8.3 (11.6)	8.8 (12.4)	9.6 (13.3)	.02
Women, %	54.7	51.2	51.4	53.3	54.2	.43
Minority race, %	30.1	27.1	19.6	19.7	22.9	< .001
Alcohol consumption ≥ 2 drinks/day, %	4.5	5.4	6.0	7.2	9.3	.02
Caffeine consumption, cups/day	3.1 (3.3)	3.1 (3.3)	3.0 (3.2)	2.9 (2.9)	3.1 (3.1)	.24
Current smoking, %	12.2	12.2	10.4	11.4	12.0	.53
Any cardiovascular disease, %	23.3	17.4	14.7	17.7	21.4	< .001
Often or always..., %						
Have trouble falling asleep	42.9	20.7	11.1	8.7	7.2	< .001
Wake up too early in the morning	52.4	26.9	13.5	8.1	6.1	< .001
Wake up and cannot return to sleep	55.4	27.0	15.5	12.3	10.9	< .001
Take a pill to help sleep	11.0	8.0	6.8	6.6	7.0	.01
Any insomnia symptom	68.8	43.6	28.3	21.9	20.1	< .001
Often feel "down"	6.2	3.2	1.9	2.6	3.3	< .001
Often feel "blue"	8.5	5.7	3.6	3.1	4.9	< .001
Resting heart rate, beats/min	73.5 (11.6)	73.7 (11.4)	73.6 (11.4)	73.7 (11.0)	74.7 (11.3)	.37
Systolic blood pressure, mm Hg	132.2 (18.7)	130.5 (18.5)	128.4 (18.0)	130.5 (19.1)	133.0 (20.2)	< .001
Diastolic blood pressure, mm Hg	75.2 (11.3)	75.0 (10.3)	74.3 (10.6)	73.9 (11.4)	73.0 (10.4)	.002
Use of antihypertensive medication, %	48.3	39.5	34.5	39.9	46.2	< .001

^aData are presented as mean (SD) or percentages. Significance tests for the unadjusted difference across categories of sleep duration are based on the χ^2 for contingency table analysis of categorical variables and analysis of variance for continuous variables. Due to the highly skewed distribution of apnea-hypopnea index (AHI), the difference in $\ln(\text{AHI} + 1)$ was tested.

^bDue to missing data, the number of subjects with data for alcohol consumption is 5197, caffeine 5848, smoking 5888, cardiovascular disease 5643, insomnia symptom 5850, and depressive symptoms 4367.

7 hours per night was reported by 29.6% of subjects, including 9.2% sleeping less than 6 hours per night. A usual sleep duration of 8 or more hours was reported by 36.2% of subjects, including 7.6% sleeping 9 or more hours per night. Subjects at the extremes of sleep duration were slightly older and heavier, more likely to be minorities, and had a higher mean AHI than subjects sleeping 7 to < 8 hours per night (Table 1). They were also more likely to have depressive symptoms or prevalent cardiovascular disease. Daily consumption of 2 or more alcoholic beverages per day was reported more commonly among individuals who also reported longer sleep durations, whereas the prevalence of insomnia symptoms was higher in subjects with shorter usual sleep duration. There was no significant difference in sex, caffeine consumption, or current smoking status across sleep-duration categories. Among subjects with hypertension, the percentage using antihypertensive medications was similar in all sleep-duration categories (range 74% to 78%, $p = .66$ for difference across categories).

Compared with sleep durations of 7 to < 8 hours per night, self-reported usual sleep durations of less than 7 hours per night or 8 or more hours per night were associated with higher adjusted odds ratio (OR) for hypertension (Table 2). As expected, older age, higher AHI, and minority race/ethnicity were associated with higher adjusted OR for hypertension. After considering these covariates, sex was not significantly associated with hypertension. Adjustment for these variables modestly attenuated the observed association of sleep duration with hypertension (Table 2). Although higher BMI was a strong predictor of the presence of hypertension, adjustment for BMI had no impact on the observed association of sleep duration with hypertension (Table 2). There was also little

change in the magnitude of the association of sleep duration with hypertension when the models were additionally adjusted for waist girth; prevalent diabetes mellitus, coronary artery disease, heart failure, stroke, or any cardiovascular disease; current cigarette smoking; usual daily alcohol consumption; presence of insomnia symptoms; or presence of depressive symptoms. When caffeine consumption was included in the models, there was a significant inverse correlation of usual daily caffeine consumption with hypertension (adjusted OR 0.95 [95% CI 0.93 – 0.97] for each additional cup of coffee), although adjustment for caffeine consumption did not diminish the magnitude of the association of sleep duration with hypertension. Sleep efficiency was available from polysomnography for 3368 subjects in whom the entire period from lights out to final awakening was recorded. Mean sleep efficiency was highest in subjects with a reported usual sleep duration of 7 to less than 8 hours (83.1%), falling to 80.2% in those reporting a usual sleep duration less than 6 hours and to 78.1% in those reporting a usual sleep duration of 9 or more hours. When this variable was included in the models, sleep efficiency was inversely associated with the risk of hypertension (adjusted OR 0.80 [95% CI 0.74–0.87] for a 10% increase in sleep efficiency) but had little impact on the association of reported sleep duration with hypertension. Additional analyses demonstrated that the association of sleep duration with hypertension was not significantly different between men and women, younger (age < 63) and older subjects, those with an AHI less than 5 and those with an AHI of 5 or higher, and those with or without insomnia symptoms.

Table 2—Odds ratios (95% Confidence Intervals)* for Hypertension by Reported Usual Sleep Duration

Usual sleep duration, h/night	Model 1 Unadjusted	Model 2 Adjusted for age, sex, race, and AHI	Model 3 Adjusted for all covariates in Model 2 plus BMI
	p < .0001	p < .0001	P < .0001
< 6	1.86 (1.54 – 2.26)	1.67 (1.36 – 2.05)	1.66 (1.35 – 2.04)
6 to < 7	1.25 (1.08 – 1.44)	1.20 (1.03 – 1.39)	1.19 (1.02 – 1.39)
7 to < 8	1.0 (referent)	1.0 (referent)	1.0 (referent)
8 to < 9	1.31 (1.15 – 1.49)	1.19 (1.04 – 1.36)	1.19 (1.04 – 1.37)
≥9	1.75 (1.42 – 2.15)	1.31 (1.05 – 1.63)	1.30 (1.04 – 1.62)

*Odds ratios are for the presence of hypertension, from categorical logistic regression models using 7 to < 8 hours of sleep per night as the referent category. P values reflect the overall significance level of the effect of sleep duration on hypertension, based on the likelihood ratio χ^2 with 4 degrees of freedom. AHI refers to apnea-hypopnea index.

DISCUSSION

The present study provides epidemiologic evidence that both short and long habitual sleep durations are associated with prevalent hypertension in community-dwelling middle-aged and older adults. The association of sleep duration with hypertension persisted after adjustment for factors believed a priori to be potential confounders of the association between sleep duration and hypertension, including age, sex, race, obesity, and AHI. Although BMI is an imperfect measure of adiposity, adjustment for BMI caused no attenuation of the association of sleep duration with hypertension, implying that residual confounding by adiposity is unlikely. Secondary analyses also indicate that the association of sleep duration with hypertension was not confounded by caffeine or alcohol consumption or cigarette smoking, which might influence sleep habits. Almost three fourths of subjects with hypertension were being treated with antihypertensive medication, raising the concern that an effect of medication on sleep duration might underlie the association of sleep duration with hypertension; however, because the likelihood that a subject with hypertension was taking antihypertensive medications did not differ by sleep duration, confounding by medication use is unlikely. These findings extend to a community-based sample the observation from experimental studies that acute sleep restriction is associated with an increase in blood pressure. They suggest that levels of habitual sleep restriction that are common in the adult population may contribute to the high population prevalence of hypertension.

There have been several studies of the effect of acute sleep restriction on blood pressure. In a group of Japanese technical workers, a night of sleep restriction to a mean of 3.6 hours due to working overtime was associated with a 6-mm Hg increase in mean SBP and a 3-mm Hg increase in mean DBP, compared with a night of 8 hours sleep, although the possible effects of work stress per se must be considered.²¹ Lusardi and colleagues found that a single night of experimental sleep restriction to 4 hours of sleep in the home setting resulted in a 4- to 7-mm Hg increase in mean morning SBP in normotensive and hypertensive subjects, respectively, with smaller increases in mean morning DBP.^{10,13} Others have found a similar increase in blood pressure in healthy subjects following a single night of total sleep deprivation.¹¹ A progressive increase in blood pressure across 88 hours of total sleep deprivation has also been reported¹² but should be interpreted with caution because there was no control group and blood pressure continued to increase during the recovery day. The same group reported increases of 22 mm Hg in mean SBP and 17 mm

Hg in mean DBP across 10 days of partial sleep deprivation to 4 hours per night in 4 healthy subjects, but this was not significantly different from the 10-mm Hg increase in mean SBP and 13-mm Hg increase in mean DBP observed in the 5 control subjects.¹² The study had little statistical power to exclude even such large effects of sleep deprivation, although it is possible that other aspects of the protocol were responsible for the observed increase in blood pressure. Another group reported considerably smaller and statistically nonsignificant increases in morning mean SBP and DBP after 4 nights of sleep restriction to 4 hours per night.²²

The biologic mechanisms underlying an association of short sleep duration and hypertension are uncertain. Sleep deprivation has been reported to cause an increase in sympathetic nervous system activity,^{13, 21, 23} which may cause sustained hypertension, although the importance of this mechanism in the apparent hypertensive response to sleep deprivation has been questioned.¹¹ Sleep deprivation also alters activity of the hypothalamic-pituitary-adrenal axis, with short-term partial sleep deprivation causing a shorter quiescent period of cortisol secretion and slower clearance of free cortisol,^{23,24} and the resultant elevated cortisol levels may increase blood pressure. Primary insomnia is associated with increased activity of the hypothalamic-pituitary-adrenal axis,^{25,26} and patients suffering from insomnia often underestimate their actual sleep duration.²⁷ However, the observed association of short sleep duration with hypertension in the current study remained significant after adjustment for insomnia or excluding subjects with insomnia, implying that voluntary sleep restriction at levels common in the population may lead to hypertension. Exercise has been reported both to improve sleep quality and to reduce blood pressure and might explain, in part, the observed association of short sleep with hypertension. Although data on activity level were not available for all SHHS parent cohorts, a prior report from the Nurses Health Study suggests that short sleep duration is not associated with lower levels of voluntary activity.²⁸

The mechanisms mediating the association of long sleep duration with hypertension are even less certain, although the consistency of epidemiologic data showing increased morbidity and mortality in individuals who are long sleepers suggests the need to consider a causal basis for such associations. Women in the Nurses Health Study who reported sleeping 9 or more hours per night reported 15% less physical activity per week than those sleeping 7 to 8 hours per night,²⁸ and inactivity may place these long sleepers at increased risk of hypertension. Depression is often associated with altered sleep duration and also may be associated with an increased cortisol level, a neurohumoral response

that may increase blood pressure. Although depression was not formally assessed in our subjects, both short and long sleep durations were significantly associated with depressive symptoms obtained from the SF-36. Adjustment for depressive symptoms did not, however, meaningfully alter the observed association of sleep duration with hypertension. Although adjustment for usual alcohol consumption did not meaningfully alter the association of sleep duration with hypertension, long sleep duration was significantly associated with higher alcohol consumption. Because heavy alcohol users may underreport their actual consumption, it is possible that alcohol use contributes to the higher prevalence of hypertension in those sleeping 8 or more hours per night. It is possible that long sleep duration reflects poor sleep quality. Subjects reporting a usual sleep duration of 9 or more hours per night did have slightly higher AHIs and lower sleep efficiencies than subjects sleeping 7 to less than 8 hours per night, but the association of sleep duration with hypertension was not meaningfully affected by adjustment for these variables.

Several limitations of this study merit discussion. Usual sleep duration was obtained by self-report. In the Nurses Health Study, self-reported usual sleep time determined using a similar question has been validated against 1 week of sleep diaries ($r=0.79$),⁹ and the stability of self-reported usual sleep time over a mean interval of 2.4 years in SHHS participants has been reported ($r=0.57$).¹⁵ Although the correlation of self-reported sleep duration with total sleep time measured on a single night of unattended polysomnography was weak, a night of sleep while wearing instrumentation for polysomnography is sufficiently different from a typical night of sleep that it is unlikely to be a valid measure of usual sleep duration. Whereas some misclassification on sleep duration is likely, this is likely to be nondirectional and thus should bias the study toward a null result. The results of this study add to a growing literature relating self-reported sleep duration with adverse health outcomes,^{4-9,15,28} which indicates the importance of this self-report measure. Because it is likely that the sleep need of individuals varies due to genetic, behavioral, and environmental factors, usual sleep duration is likely to be an imperfect measure of sleep sufficiency. To the extent that short sleep duration does not reflect insufficient sleep in some individuals, this will again bias the study toward a null result. Thus, the true effect of sleep deprivation may be greater than that observed in this study. The development of simple valid methods for assessment of individual sleep need or sleep deprivation per se would facilitate future epidemiologic studies of the effects of sleep duration. The present study utilized evening blood-pressure measurements. While circadian variation in the effect of sleep deprivation on blood pressure is possible and merits further study, circadian effects would likely have little effect on the results of this analysis, as 75% of hypertensive subjects were identified on the basis of antihypertensive medication use rather than on the evening blood-pressure measurement. Because this was a cross-sectional observational study, the possibility of confounding by unmeasured variables, such as sedentary lifestyle and diet, cannot be excluded, and the temporal relationship between sleep duration and hypertension is unknown. Finally, this was a study of middle-aged and older adults and may not be relevant to younger subjects; however, the relationship between sleep duration and hypertension was similar in those older and younger than 63 years, the median age of the study sample. Notwithstanding these limitations, the present study provides epidemiologic evidence that the increase in blood pressure reported

from experimental studies of severe sleep restriction is also present at levels of chronic sleep restriction that are common in the adult population. The association of sleep duration with hypertension may explain, in part, the association between sleep duration and both myocardial infarction¹⁶ and mortality⁴⁻⁷ and lends empiric support to the common recommendation to obtain 7 to 8 hours of sleep per night. Moreover, it suggests that obtaining adequate total sleep duration should be tested as a nonpharmacologic treatment modality in the management of patients with hypertension.

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