Short Sleep Duration as an Independent Predictor of Cardiovascular Events in Japanese Patients With Hypertension

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**Background:** It is not known whether short duration of sleep is a predictor of future cardiovascular events in patients with hypertension.

**Methods:** To test the hypothesis that short duration of sleep is independently associated with incident cardiovascular diseases (CVD), we performed ambulatory blood pressure (BP) monitoring in 1255 subjects with hypertension (mean [SD] age, 70.4 [9.9] years) and followed them for a mean period of 50 (23) months. Short sleep duration was defined as less than 7.5 hours (20th percentile). Multivariable Cox hazard models predicting CVD events were used to estimate the adjusted hazard ratio and 95% confidence interval (CI) for short sleep duration. A riser pattern was defined when mean nighttime systolic BP exceeded daytime systolic BP. The end point was a cardiovascular event: stroke, fatal or nonfatal myocardial infarction (MI), and sudden cardiac death.

**Results:** In multivariable analyses, short duration of sleep (<7.5 hours) was associated with incident CVD (hazard ratio [HR], 1.68; 95% CI, 1.06-2.66; \( P = .03 \)). A synergistic interaction was observed between short sleep duration and the riser pattern (\( P = .09 \)). When subjects were classified according to their sleep time and a riser vs nonriser pattern, the group with shorter sleep duration plus the riser pattern had a substantially and significantly higher incidence of CVD than the group with predominant normal sleep duration plus the nonriser pattern (HR, 4.43; 95% CI, 2.09-9.39; \( P < .001 \)), independent of covariates.

**Conclusions:** Short duration of sleep is associated with incident CVD risk and the combination of the riser pattern and short duration of sleep that is most strongly predictive of future CVD, independent of ambulatory BP levels. Physicians should inquire about sleep duration in the risk assessment of patients with hypertension.

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**Reflecting Changing Life-styles, people are sleeping less in modern societies.** A good sleep of adequate duration is essential because sleep fragmentation and sleep deprivation, commonly seen in contemporary society, are associated with multiple health disorders, including cardiovascular diseases (CVD). Several sleep-related phenomena, such as sleep-disordered breathing, nocturnal hypertension, and high variability of nocturnal blood pressure (BP), have been suggested to be independent risk factors for cardiovascular events. Short duration of sleep has been shown to be related to obesity, diabetes mellitus (DM), hypertension, and sleep apnea in elderly individuals. Sleep deprivation is a risk factor for all-cause mortality and coronary heart disease in epidemiological studies, but these studies were performed in relatively young subjects and women. To our knowledge, this is the first such study to assess ambulatory blood pressure (ABP), thus enabling us to examine the role of the nocturnal BP pattern in this relationship.

Nondipping of BP, especially a nocturnal rise of BP, often referred to as the “riser pattern,” has been reported to be associated with cardiovascular events. However, it was reported that the higher nighttime BP than daytime BP might not be a cause but rather a marker of total mortality. Verdecchia et al have reported that the nondipping nocturnal BP pattern was associated with increased cardiovascular risk, but this association became nonsignificant when perceived sleep deprivation was statisti-
been no reports examining the impact on incident CVD of diary-based short sleep duration and its possible interaction with nocturnal BP. Furthermore, these relationships have not been examined in elderly individuals. Thus, we tested 2 hypotheses: first, that short duration of sleep is associated with future cardiovascular events in an elderly population with hypertension, and second, that this association may be moderated by the circadian BP pattern.

METHODS

SETTING AND PATIENT RECRUITMENT

This prospective study was performed in a sample of 1268 asymptomatic patients referred for the evaluation of hypertension who were seen in clinics at 9 participating medical institutions in Japan: 3 clinics, 2 hospitals, and 1 outpatient clinic of a university hospital (the Jichi Medical School ABP Monitoring, hereinafter, the JMS ABPM Study Wave 1), and 1 clinic and 2 hospitals (hereinafter, the Karatsu-Nishiarita Study). The study designs and assessment of the outcome of these 2 cohorts were essentially the same, differing only with respect to the sites and the time performed. This study was approved by the institutional review board of each participating hospital or clinic. All participants were ambulatory and gave informed consent for the study. During the period of recruitment, 1990 to 1998 for the JMS ABPM Study Wave 1 sample and 1996 to 2002 for the Karatsu-Nishiarita Study, all consecutive patients who were being treated or evaluated for hypertension in the clinic and agreed to undergo ABPM were enrolled. Because 13 subjects did not have information regarding their exact duration of sleep and were excluded from the study, a total of 1255 subjects were evaluated. Their mean (SD) age was 70.4 (9.9) years (range, 33-97 years); there were 476 men and 779 women, and 94% of subjects had hypertension.

Hypertension was diagnosed, according to current guidelines, when the clinic systolic BP (SBP) was at least 140 and/or diastolic BP (DBP) was at least 90 mm Hg on at least 2 occasions or when the patient had a previous diagnosis of hypertension and was currently using at least 1 antihypertensive medication. Clinic BP was measured at least twice on 2 separate occasions after at least 5 minutes of rest in the sitting position. Subjects took no antihypertensive medication for a minimum of 7 days before the ABPM, and most took no medication during the 14 days preceding the ABPM study. Type 2 DM was diagnosed according to the guidelines of the American Diabetes Association or if the patient had a previous diagnosis and was currently taking anti-DM medication. We excluded patients with a history of type 1 or secondary DM, renal dysfunction (serum creatinine level >1.9 mg/dL), hepatic damage, ischemic heart disease, or other cardiac diseases, congestive heart failure, arrhythmias (including atrial fibrillation), stroke (including transient ischemic attacks), or other major concomitant noncardiovascular diseases. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Smoking was defined as current smoking. (To convert serum creatinine to micromoles per liter, multiply by 88.4.)

AMBULATORY BP MONITORING

Noninvasive ABPM was performed on a weekday with an automatic system (ABPM-630 [Omron Colin Inc, Tokyo, Japan] or TM2421 or TM2425 [A&D, Tokyo]), which recorded BP by the oscillometric method and pulse rate every 30 minutes for 24 hours. These devices have been previously validated. Awake and sleep times were defined based on patients’ diaries recorded during ABPM. The percentage of nocturnal BP fall was calculated as (awake SBP – sleep SBP)/awake SBP. We classified subjects’ nocturnal BP fall as having a riser pattern if the nocturnal BP fall was less than 0% and as having a nondipping pattern if it was less than 10% (including riser patterns). Sleep duration was defined as the difference between sleep and wake times. Subjects who reported apparent sleep disturbances as a result of the ABPM procedure were excluded from all statistical analyses. The subjects were divided into 2 groups according to self-reported sleep duration of more or less than 7.5 hours. This was based on the 20th percentile of sleep duration in the entire sample (Figure 1). The median (25th and 75th percentiles) of sleep duration was 8.5 hours (7.5 and 9.5 hours). The cutoff value was based on previous articles showing that a sleep duration of 7 to 8 hours had the lowest risk. Because older people tend to sleep (or stay in

Figure 1. Histogram of sleep duration in the sample population. The sleep duration was categorized into 30-minute intervals (eg, “6 hours 1 minute to 6 hours 30 minutes” as 6.5 hours, and “6 hours 31 minutes to 7 hours” as 7 hours). The median sleep duration was 8.5 hours, and the 5 quintiles were 4.0 to 7.4, 7.5 to 7.8, 8.0 to 8.8, 9.0 to 9.4, and 9.4 to 13.0 hours, respectively.
Table 1. Baseline Characteristics of Subjectsa

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Quintileb</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>First (n=248)</td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>67.4 (10.2)</td>
</tr>
<tr>
<td>Male sex, %</td>
<td>37.5</td>
</tr>
<tr>
<td>BMI</td>
<td>24.4 (3.5)</td>
</tr>
<tr>
<td>Smoking, %</td>
<td>22.6</td>
</tr>
<tr>
<td>DM, %</td>
<td>26.6</td>
</tr>
<tr>
<td>Cholesterol level, mg/dL</td>
<td>204 (36)</td>
</tr>
<tr>
<td>Triglyceride level, mg/dL</td>
<td>143 (82)</td>
</tr>
<tr>
<td>Serum creatinine level, mg/dL</td>
<td>0.85 (0.22)</td>
</tr>
<tr>
<td>Clinic SBP, mm Hg</td>
<td>160 (20)</td>
</tr>
<tr>
<td>Clinic DBP, mm Hg</td>
<td>88 (13)</td>
</tr>
<tr>
<td>24-h SBP, mm Hg</td>
<td>140 (17)</td>
</tr>
<tr>
<td>24-h DBP, mm Hg</td>
<td>80 (10)</td>
</tr>
<tr>
<td>24-h PR, beats/min</td>
<td>72 (7)</td>
</tr>
<tr>
<td>Awake SBP, mm Hg</td>
<td>146 (18)</td>
</tr>
<tr>
<td>Awake DBP, mm Hg</td>
<td>83 (10)</td>
</tr>
<tr>
<td>Awake PR, beats/min</td>
<td>76 (8)</td>
</tr>
<tr>
<td>Sleep SBP, mm Hg</td>
<td>128 (18)</td>
</tr>
<tr>
<td>Sleep DBP, mm Hg</td>
<td>73 (10)</td>
</tr>
<tr>
<td>Sleep PR, beats/min</td>
<td>62 (9)</td>
</tr>
<tr>
<td>Night-day SBP ratio</td>
<td>0.88 (0.09)</td>
</tr>
<tr>
<td>Nondipping pattern, %</td>
<td>37.1</td>
</tr>
<tr>
<td>Risers, %</td>
<td>8.1</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); DBP, diastolic blood pressure; DM, diabetes mellitus; PR, pulse rate; SBP, systolic blood pressure.

SI conversion factors: To convert total cholesterol and triglycerides to millimoles per liter, multiply by 0.0259 and 0.0113, respectively; to convert creatinine to micromoles per liter, multiply by 88.4.

a Data are shown as percentage or mean (SD) unless otherwise indicated.

b Quintile ranges: first, 4.0 to 7.4 hours; second, 7.5 to 7.8 hours; third, 8.0 to 8.8 hours; fourth, 9.0 to 9.4 hours; fifth, 9.4 to 13.0 hours.

c P values are based on a 1-way analysis of variance for the continuous measures and the χ2 test of independence for dichotomous measures.

FOLLOW-UP AND EVENTS

The subjects’ medical records were reviewed once a year after ABPM for the purpose of identifying any new onset of CVD: the 798 participants enrolled in the 1990-1998 period for the JMS ABPM Study Wave 1 were followed starting in the period 1996-1998 for up to 5.7 years or until they moved, changed their telephone number, or died; the 457 participants enrolled during the 1996-2002 period for the Karatsu-Nishiarita Study were similarly followed starting in the period March 2004 through October 2006, giving a follow-up period of up to 9.7 years. Participants who died from noncardiovascular causes were censored as of the time of their death. The mean (SD) follow-up period was 41 (14) months (range, 1-68 months) in the JMS ABPM Study Wave 1 and 66 (27) months (range, 9-116 months) in the Karatsu-Nishiarita Study. When subjects did not visit the clinics, we interviewed them by telephone. We assessed 3 outcomes: stroke, fatal or nonfatal myocardial infarction (MI), and sudden cardiac death. Strokes and cardiac events were diagnosed by the physician who was caring for the patient at the time of the event, and independent neurologists or cardiologists reviewed the cases and confirmed the diagnosis. Stroke was diagnosed on the basis of sudden onset of a neurological deficit that persisted for more than 24 hours in the absence of any other disease process that could explain the symptoms. Stroke events were further classified as ischemic stroke (cerebral infarction and cerebral embolism), hemorrhagic stroke (cerebral hemorrhage and subarachnoid hemorrhage), and undefined types of stroke. We excluded transient ischemic attacks, in which the neurological deficit cleared completely in less than 24 hours.6 Myocardial infarction was diagnosed based on the American Heart Association criterion of “definite” MI. We did not include angina or congestive heart failure as end points.

STATISTICAL ANALYSES

All statistical analyses were performed with SPSS software for Windows (version 13.0; SPSS Inc, Chicago, Illinois). The χ2 test was used to compare proportions. A 1-way analysis of variance was performed to test differences among group means (Table 1). Kaplan-Meier curves were used to compare the unadjusted survival functions of subgroups, and the modified Bonferroni correction for multiple tests of significance was used to evaluate the significance of differences between groups (Figure 2). Adjusted hazard ratios (HRs) with 95% confidence intervals (CIs) were based on multivariable Cox regression analyses predicting incident CVD with the primary independent variable being sleep duration (dichotomized as <7.5 hours or ≥7.5 hours). Because the durations of follow-up of the 2 participant groups were different, the Cox regression models were stratified by site. Model 1 statistically controlled for age (years) and sex (male=1, female=0), with model 2 further
controlling for BMI, current smoking (yes=1, no=0), DM status (yes=1, no=0), total cholesterol level (in milligrams per deciliter), serum creatinine (in milligrams per deciliter), log-transformed triglyceride level, and 24-hour SBP; model 3 added the riser pattern (yes=1, no=0) as additional control variables. Sleep duration was not treated as a continuous variable because a U-shaped relationship between sleep duration and mortality has been reported. To examine the potential moderating effect of the circadian BP pattern on the relationship of short sleep duration to incident CVD, an interaction term (between sleep duration and the riser pattern vs longer sleep duration and the nonriser pattern) was tested as a post hoc analysis. The null hypothesis was rejected when 2-tailed \( P < .05 \). Because the power to detect interaction effects is low, we used 2-tailed \( P < .10 \) as the criterion to judge statistical significance.

**RESULTS**

When the subjects were divided into 5 groups according to sleep duration, the groups with shorter sleep durations were younger and had higher BMI, pulse rates, and DM rates as graded relationships; however, other variables were similar among the 5 groups (Table 1).

During the mean (SD) follow-up period of 50 (23) months, 99 cardiovascular events occurred. The incidence of CVD was 2.4 per 100 person-years in subjects with less than 7.5 hours of sleep and 1.8 per 100 person-years in subjects with longer sleep duration. The Kaplan-Meier survival curves for the 4 categories of shorter vs longer sleep duration and a riser vs nonriser pattern are shown in Figure 2. The group with a shorter sleep duration who also had a riser pattern had the highest incidence of CVD among the 4 groups. Subjects with a shorter sleep duration had a higher incidence of CVD than those with longer sleep duration within the riser pattern group, but the incidence of CVD in those with a longer sleep duration vs those with a shorter sleep duration was similar in the nonriser pattern group. In multivariable Cox regression analyses, a shorter sleep duration was significantly associated with incident CVD independent of demographic variables (model 1 in Table 2), plus traditional risk factors (model 2), plus abnormal (riser pattern) circadian BP pattern (model 3), which was not significant (see Table 2 for \( P \) values).

Because higher ABP levels and the riser pattern are likely to reflect a failure of the restorative function of sleep, we decided to test whether there was a synergistic effect of ABP and/or the riser pattern on the relationship of short sleep duration to CVD risk. The interactions between shorter sleep duration (<7.5 hours) and 24-hour SBP and sleep SBP were not significant (\( P = .92, P = .97 \), respectively), but the interaction of a shorter sleep duration (<7.5 hours) with the riser pattern suggests that this combination of risk factors is associated with a marked increase in CVD risk (Table 3). This finding needs to be empirically replicated in an independent study. When the same analysis was performed using the nondipper (vs dipper) pattern instead of the riser (vs nonriser) pattern, the interaction of nondipper status with shortened sleep duration was not significant (\( P = .51 \)). We performed parallel analyses using 24-hour DBP, and the results were essentially the same.

Using the estimates for this model, Table 3 summarizes the relationship of the 4 groups, defined by the duration of sleep of more or less than 7.5 hours and the presence or absence of the riser pattern, to CVD. The group that had both shorter sleep duration plus the riser pattern had a 4.4-fold increase of CVD compared with those with longer sleep duration who did not have the riser pattern. However, the other groups did not exhibit an increased risk of CVD. Thus, although we cannot definitively conclude that there is a synergistic interaction of sleep duration with riser status (as opposed to these 2 factors having additive effects on the log hazard), it is clear that the group at greatest risk comprises those who have riser patterns and shortened duration of sleep.

**COMMENT**

In this study, a shorter duration of sleep (<7.5 hours) evaluated by a diary during ABPM was an independent predictor for the future incidence of CVD. The riser pattern, a known predictor of CVD, showed a significant interaction (\( P = .09 \)) with shorter sleep duration. To our knowledge, this is the first report showing the prognostic importance of shorter sleep duration in combination with the nocturnal BP dipping pattern as a risk for incident CVD.

**SLEEP DURATION AND CVD**

In our study, shorter sleep duration was associated with increased cardiovascular events independently of other covariates. There have been a few articles showing a significant relationship between short duration of sleep and cardiovascular events. Ayas et al reported that short duration
of sleep was associated with increased risk of coronary events in women. But their study was very different from ours: the outcome measure was only coronary events, the subjects were much younger (52 vs 70 years), and long (as well as short) sleep duration was also associated with an increased risk. Some other prospective studies examined the relationship of sleep duration and all-cause mortality in large epidemiological databases, 

Table 2. Multivariable Cox Regression Analyses of Shortened Sleep Duration as a Predictor of Incident Cardiovascular Events

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Model 1 (HR [95% CI])</th>
<th>P Value</th>
<th>Model 2 (HR [95% CI])</th>
<th>P Value</th>
<th>Model 3 (HR [95% CI])</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep duration &lt;7.5 h vs ≥7.5 h</td>
<td>1.68 (1.06-2.66)</td>
<td>.03</td>
<td>1.59 (1.00-2.32)</td>
<td>.049</td>
<td>1.59 (0.99-2.49)</td>
<td>.06</td>
</tr>
<tr>
<td>Age, mean per 5 y^a</td>
<td>1.48 (1.32-1.65)</td>
<td>&lt;.001</td>
<td>1.85 (1.26-1.62)</td>
<td>&lt;.001</td>
<td>1.48 (1.63-1.59)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Male sex, %</td>
<td>1.71 (1.56-2.45)</td>
<td>.01</td>
<td>1.77 (0.54-1.96)</td>
<td>.40</td>
<td>1.72 (0.78-2.01)</td>
<td>.36</td>
</tr>
<tr>
<td>BMI</td>
<td>1.00 (0.94-1.17)</td>
<td>.94</td>
<td>1.01 (0.95-1.08)</td>
<td>.77</td>
<td>1.01 (0.95-1.08)</td>
<td>.77</td>
</tr>
<tr>
<td>Current smoking (yes or no)</td>
<td>1.80 (1.22-2.77)</td>
<td>.02</td>
<td>1.78 (1.11-2.85)</td>
<td>.02</td>
<td>1.78 (1.11-2.85)</td>
<td>.02</td>
</tr>
<tr>
<td>Diabetes mellitus (yes or no)</td>
<td>1.48 (0.93-2.37)</td>
<td>.10</td>
<td>1.38 (0.86-2.22)</td>
<td>.19</td>
<td>1.38 (0.86-2.22)</td>
<td>.19</td>
</tr>
<tr>
<td>Total cholesterol per 20 mg/dL</td>
<td>1.01 (0.89-1.15)</td>
<td>.88</td>
<td>1.02 (0.89-1.16)</td>
<td>.79</td>
<td>1.02 (0.89-1.16)</td>
<td>.79</td>
</tr>
<tr>
<td>Creatinine level per 0.1 mg/dL</td>
<td>1.09 (1.00-1.19)</td>
<td>.06</td>
<td>1.01 (1.00-1.20)</td>
<td>.04</td>
<td>1.01 (1.00-1.20)</td>
<td>.04</td>
</tr>
<tr>
<td>Log triglyceride level</td>
<td>0.87 (0.31-2.50)</td>
<td>.80</td>
<td>0.85 (0.30-2.47)</td>
<td>.77</td>
<td>0.85 (0.30-2.47)</td>
<td>.77</td>
</tr>
<tr>
<td>24-h SBP, mean per 10 mm Hg^b</td>
<td>1.36 (1.22-1.51)</td>
<td>&lt;.001</td>
<td>1.33 (1.19-1.48)</td>
<td>&lt;.001</td>
<td>1.33 (1.19-1.48)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Riser pattern (yes or no)</td>
<td></td>
<td></td>
<td>1.86 (1.30-3.16)</td>
<td>.02</td>
<td>1.86 (1.30-3.16)</td>
<td>.02</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); CI, confidence interval; HR, hazard ratio; SBP, systolic blood pressure.

^a Mean value of age was 70 years.

^b The mean value of age was 70 years.

^c Mean value of 24-hour SBP was 139 mm Hg.

We found a significant interaction effect of shorter sleep duration and ABP level on the risk of CVD ($P = .92$). Verdecchia et al. reported that the prognostic impact of nocturnal BP disappeared when subjects reported that they slept at least 2 hours less than usual on the night of the ABPM. Our study does not have data on perceived sleep deprivation, and their focus was different from ours: they did not analyze the riser pattern and actual sleep duration. We found a significant interaction ($P = .09$) of shorter sleep duration ($<7.5$ hours) and the riser pattern on the risk of CVD, which in combination resulted in a 4.4-fold increased risk of CVD when compared with the subjects who slept longer than 7.5 hours and had the nonriser pattern. Lack of sleep in patients with hypertension and higher night to day ratio of SBP have both been shown to increase sympathetic nervous activity during the night. Therefore, short sleep duration and the riser pattern might have an interactive effect to increase cardiovascular risk. It is a relatively small.

NOCTURNAL BP PATTERN AND SLEEP DURATION

In addition to BP levels, nocturnal BP dipping patterns, especially nondipping, are established risk factors for cardiovascular events. In a recent population study, subjects with a higher night to day ratio of SBP of 1.0 or more were older and at higher risk of death than those whose night to day ratio was within reference range ($>0.80$ to $<0.90$). One of the presumed causes of an abnormal nocturnal BP dipping pattern is disordered sleep. Greater physical activity during sleep, poor sleep quality, and sleep apnea episodes have been implicated as causes of abnormal BP dipping patterns. Sleep deprivation is related to poor sleep quality, and it worsens obstructive sleep apnea. Taking all these findings together, short sleep duration might be related to ABP levels and the nondipping pattern. However, in our study, ABP levels and the percentage of those with a nondipping pattern, which included those with a riser pattern, were similar between the shorter sleep duration and the longer sleep duration groups. There was also no significant interaction effect of shorter sleep duration and ABP level on the risk of CVD ($P = .92$). Verdecchia et al. reported that the prognostic impact of nocturnal BP disappeared when subjects reported that they slept at least 2 hours less than usual on the night of the ABPM. Our study does not have data on perceived sleep deprivation, and their focus was different from ours: they did not analyze the riser pattern and actual sleep duration. 

Table 3. Hazard Ratios (HRs) Predicting Incident Cardiovascular Events for Subgroups Defined by Sleep Duration and Nocturnal Blood Pressure Pattern

<table>
<thead>
<tr>
<th>Duration of Sleep, h</th>
<th>Nonriser Pattern Group (n=932)</th>
<th>Riser Pattern Group (n=75)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group with ≥7.5 h</td>
<td>1.37 (0.70-2.68)</td>
<td>4.43 (2.09-9.39)^b</td>
</tr>
<tr>
<td>Group with &lt;7.5 h</td>
<td>1.27 (0.73-2.18)</td>
<td>4.43 (2.09-9.39)^b</td>
</tr>
</tbody>
</table>

^a The interaction term of the riser blood pressure pattern with short sleep duration was significant ($P = .09$). Data are given as HRs (95% confidence intervals), adjusted for the same variables of model 2. 

^b $P < .001$. 

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group of individuals who have this combination, and thus it is a group that could be easily identified and monitored more closely.

In our data, serum creatinine level was a significant predictor of incident CVD (P = .01). This is consistent with serum creatinine as a marker for hypertensive target organ damage and a strong risk factor for future CVD even within the reference range.

STUDY LIMITATIONS

This study has some limitations. We have no data relating to polysomnography, snoring, or the use of hypnotics. However, there are also no data in the literature showing that the use of hypnotics affects cardiovascular outcomes. Because our sleep duration data are from a diary filled out at the time of the ABPM, the sleep duration could have been a little different from the subjects’ usual duration. Because our data did not include the subjects who reported sleep disturbance, and our subjects reported a very wide range of sleep durations (range, 4-13 hours), it was unlikely that the possible minor difference of sleep duration during the ABPM interfered with the results. Because of the limited number of subjects with the riser pattern, the study was underpowered to detect an interaction at the P < .05 level. Nevertheless the group with both short sleep duration and the riser BP pattern were at much higher risk of incident CVD than the other groups shown in Table 3. Finally, most of our subjects were elderly, so our results may not be applicable to younger populations.

In conclusion, shorter duration of sleep is a predictor of incident cardiovascular disease in elderly individuals with hypertension, particularly when it co-occurs with a riser pattern of nocturnal BP. Its effect tended to be independent of ambulatory BP levels and other standard cardiovascular risk factors.

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REFERENCES


Error in Table. The Original Investigation titled “Short Sleep Duration as an Independent Predictor of Cardiovascular Events in Japanese Patients With Hypertension,” published in the November 10, 2008, issue of the Archives (2008;168[20]:2225-2231) contained an error in Table 3. The numbers of patients in the Nonriser Pattern Group and Riser Pattern Group should have read 1160 and 95, respectively.