

Sleep Apnea and Hypertension

A Population-based Study

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■ **Objective:** To measure the independent association of sleep-disordered breathing (sleep apnea and habitual snoring) and hypertension in a healthy adult population.

■ **Design:** A cross-sectional study of blood pressure during wakefulness and sleep among participants with and without sleep-disordered breathing.

■ **Setting:** Community-based study.

■ **Participants:** 147 men and women, aged 30 to 60 years, selected from Wisconsin State employees enrolled in the Wisconsin Sleep Cohort Study, an ongoing, prospective, epidemiologic study of sleep-disordered breathing.

■ **Measurements:** Sleep and medical history interview, nocturnal polysomnography, and 24-hour ambulatory blood pressure monitoring in all participants.

■ **Results:** Mean blood pressures were significantly higher among participants with sleep apnea (≥ 5 apneas or hypopneas per hour of sleep) compared with those without ($131/80 \pm 1.7/1.1$ mm Hg compared with $122/75 \pm 1.9/1.2$ mm Hg during wakefulness and $113/66 \pm 1.8/1.1$ mm Hg compared with $104/62 \pm 2/1.3$ mm Hg during sleep, respectively; $P < 0.05$). The variability of the blood pressure during sleep was significantly greater in participants with sleep apnea or a history of snoring compared with those without ($P < 0.05$). After controlling for obesity, age, and sex, sleep apnea was significantly associated with hypertension in a dose-response fashion, with odds ratios ranging from 2.0 for 5 apneic or hypopneic episodes per hour of sleep to 5.0 for 25 apneic or hypopneic episodes.

■ **Conclusions:** Our data indicate an association between hypertension and sleep apnea independent of obesity, age, and sex in a nonselected, community-based adult population.

Transient, nocturnal elevations of blood pressure have been observed during apneic episodes in the sleep apnea syndrome and may be caused by the acute consequences of sleep-disordered breathing such as arousals, high negative intrathoracic pressures, nocturnal desaturation of oxyhemoglobin, hypercapnia, or increased sympathetic nerve activity (1). However, the mechanism by which these intermittent nocturnal events contribute to sustained, daytime hypertension is not known. It has been postulated that repetitive hemodynamic oscillations caused by frequent apneic episodes occurring in rapid succession may prevent systemic blood pressures from returning to baseline levels and that this may in turn result in neurohumoral or vascular changes leading to elevated waking pressures and sustained hypertension (2).

Several case-control and cross-sectional studies suggest that hypertension is highly prevalent (50% to 90%) in patients with the sleep apnea syndrome (1–5). Sleep apnea has also been reported to occur frequently (22% to 62%) in patients with essential hypertension (6–9). However, in these studies blood pressure readings were collected at one time of day in the clinic (6, 9) or from records of reported hypertension (7, 8) in highly selected patient populations such as patients with severe obstructive sleep apnea (1–5). Some studies found a strong association between sleep apnea and hypertension whereas others failed to do so. Inconsistencies in the results are probably caused by nonuniform definitions of hypertension and apnea, differences in methods and populations studied, lack of appropriate control participants, and failure to control for confounding factors such as age, sex, and obesity.

Self-reported habitual, heavy snoring has also been associated with hypertension (10–13). Because most people with sleep apnea snore, however, polysomnographic data are needed to separate participants with sleep apnea and to assess the association with snoring alone. In the earlier surveys, no objective data on breathing during sleep were available, so it was not possible to determine if any or all of the measured association was caused by the inclusion of apneic persons among snorers. In recent epidemiologic studies that included polysomnographic data, it was found that once sleep apnea was incorporated into the analysis, snoring did not contribute independently to the prediction of hypertension (14).

The most compelling evidence that sleep apnea can cause sustained high blood pressure comes from studies that have shown a reduction in blood pressure after sleep apnea was treated (6, 15–22). However, interpre-

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tation of these intervention studies is difficult because of the confounding effect of weight loss that occurred in several studies (17–19, 22) or lack of data on weight change (6, 12, 16, 20, 23). Weight loss alone has been shown to reduce blood pressure (23). Collectively, these studies suggest the hypothesis that sleep-disordered breathing plays a causal role in hypertension. In view of recent estimates of sleep apnea prevalence of 9% for women and 24% for men (24), there is a clear need to investigate how sleep-disordered breathing at the milder end of the severity spectrum, including habitual snoring without sleep apnea, is related to blood pressure.

Our purpose was to determine how ambulatory blood pressure measurements obtained during an entire 24-hour period, while awake and asleep, differed among people with and without sleep-disordered breathing in the general population. To avoid previous problems of relying on casual blood pressure measurements, of difficulty in separating effects of snoring from those of sleep apnea, and of selection bias between groups, we studied 147 employed adults enrolled in a long-term study of risk factors, natural history, and health consequences of sleep-disordered breathing using 24-hour ambulatory blood pressure monitoring and overnight polysomnography.

Methods

One hundred and forty-seven persons were recruited from the Wisconsin Sleep Cohort Study, an ongoing prospective study of sleep-disordered breathing. The details of the design and sampling scheme for the Sleep Cohort Study have been described previously (24). We used a two-stage sampling scheme designed to yield a cohort of men and women with a wide range of sleep-disordered breathing. For the first stage, all state employees, aged 30 to 60 years, from four large agencies were surveyed on sleep characteristics, health history, and sociodemographics by mailed questionnaire. For the second stage, all survey participants reporting habitual (almost every night) snoring, snorting, or breathing pauses, or extremely loud snoring (designated “snorers”) and a random sample of the remaining participants (designated “nonsnorers”) were recruited for laboratory sleep studies, with an overall ratio of one “nonsnorer” for every two “snorers.” During a 1-year period, 163 employees consecutively studied by overnight polysomnography in the Sleep Research Laboratory at the University of Wisconsin Clinical Research Center were asked to participate in the blood pressure study: Of these, 147 participants agreed and were successfully studied (90% response rate). The protocol was approved by the Human Subjects Committee at the University of Wisconsin Hospital and Clinics, and all participants gave written, informed consent.

Data Collection

Overnight polysomnography conducted in our sleep laboratory consisted of electroencephalography, electro-oculography, and electromyography to identify sleep stages (25); measurements of nasal and oral airflow by end-tidal carbon dioxide detection and thermistery; oximetry for arterial oxyhemoglobin saturation; and inductance plethysmography to detect respiratory effort. Body weight and height were measured on all participants to calculate body mass index (kg/m^2). Data on sleep problems, sociodemographics, and health history were collected via a structured questionnaire and personal interview. The complete sleep study protocol has been previously reported (24).

Criteria for Sleep Apnea and Habitual Snoring

An abnormal breathing event was defined as either a complete cessation of airflow lasting 10 seconds or more (apnea) or a decrease in airflow accompanied by a 4% or greater decrease

in arterial oxygen saturation (hypopnea). The average number of abnormal events per hour of sleep (apnea-hypopnea index) for each person was used as a measure of sleep apnea. For categorical data analysis, cut points were used to represent mild or worse sleep apnea (apnea-hypopnea index ≥ 5) and little or no sleep apnea (apnea-hypopnea index < 5). Participants with little or no sleep apnea were further categorized as “snorers” or “nonsnorers” based on self-reported habitual snoring (every night or almost every night).

Criteria for Sleep-disordered Breathing

We categorized participants as “participants with sleep-disordered breathing” (those with apnea-hypopnea index ≥ 5 and nonapneic snorers with apnea-hypopnea index < 5) and “participants with no sleep-disordered breathing” (nonsnorers with apnea-hypopnea index < 5).

Ambulatory Blood Pressure Measurements

These measurements were obtained with the Accutrack II (Suntech Medical Instruments/Eutectics Electronics, Raleigh, North Carolina), a 24-hour blood pressure monitoring device that uses a modified auscultatory method of blood pressure measurement. The system consists of a portable 2.25-kg console that is connected to three electrocardiographic leads, a cuff, and a microphone positioned over the left brachial artery. The console initiates inflation of the cuff. During the deflation of the cuff set at a rate of 3 mm Hg per second, the person's R-wave complex triggers the microphone to listen for Korotkoff sounds during a window period after a brief pulse-propagation delay. This system, called “R-wave gating,” reduces the effect of muscle artifact or amount of artifactual sound encountered in noisy environments outside of the office setting. Special computer software identifies the cuff pressures at the onset and disappearance of Korotkoff sounds for each cuff deflation cycle as systolic and diastolic pressures, respectively.

At the beginning of all ambulatory blood pressure monitor placements, three seated and three standing pressures were obtained on each participant and calibrated to within 5 mm Hg of a standard mercury sphygmomanometer using a T-tube assembly. Blood pressures were measured at random intervals of 15 to 20 minutes during waking hours and every 30 minutes during periods of sleep. The deflation rate was set at 3 mm Hg per second. All measurements were done with the display monitor off to prevent anticipation of the readings by the participants. Ambulatory pressures while awake included those taken during activities of daily living such as sitting, standing, walking, and eating. Detailed diaries of activity, posture, bedtime, and time on awakening from sleep were kept by all the participants. Participants were instructed to refrain from vigorous exercises and arm movements during inflations. Specific information relevant to blood pressure such as data on history of hypertension and use of antihypertensive medications were obtained at the time of placement of the 24-hour blood pressure monitor.

The ambulatory pressure data record for each person included individual readings of systolic blood pressure, diastolic blood pressure, mean arterial pressure, and heart rate at random intervals. The blood pressure data were edited using predetermined criteria and without knowledge of the sleep data. Criteria for deleting individual blood pressure readings included a pulse pressure that was greater than 120 mm Hg or less than 15 mm Hg (biologically implausible), an inconsistent increase or decrease in systolic or diastolic blood pressure greater than 30 mm Hg from previous or subsequent reading occurring during test codes indicating major arm movement, or weak Korotkoff sounds.

The reproducibility of the ambulatory blood pressure data was examined in 11 participants. Repeated 24-hour measurements on 2 separate days showed no significant difference in the mean arterial pressures between days: The mean (SE) difference in mean arterial pressures between days was 2.6 (1.7) mm Hg during wakefulness ($P = 0.2$) and 2.2 (1.7) mm Hg during sleep ($P = 0.2$).

The effect of intermittent cuff inflation on sleep state was investigated in three volunteers during sleep by overnight monitoring of continuous beat by beat photoplethysmographic

Table 1. Characteristics of Participants

Characteristic	Apnea-Hypopnea Index		
	<5 in Nonsnorers	<5 in Snorers	≥5 in Apneics
Participants, <i>n</i>	41	53	53
Men, <i>n</i> (%)	17 (41)	21 (40)	37 (70)
Women, <i>n</i> (%)	24 (59)	32 (60)	16 (30)
Age, mean ± SE, <i>y</i>	41.8 ± 1.1	43.2 ± 1.1	44.4 ± 1.0
Body mass index			
Mean ± SE, kg/m ²	25.7 ± 0.8	27.6 ± 0.7	31.7 ± 0.8
≥27, <i>n</i> (%)	12 (29)	25 (47)	40 (75)
Apnea-hypopnea index			
Mean ± SE	0.8 ± 0.2	1.4 ± 0.2	16.6 ± 2.0
Median (range)	0.4 (0 to 4)	0.6 (0 to 4)	10.5 (5 to 67)
Total sleep time, mean ± SE <i>min</i>	371.4 ± 77.8	397.6 ± 74.7	355.8 ± 61.2

blood pressure (Finipress, Ohmeda, Inc., Englewood, New Jersey) during simultaneous Accutracker pressure measurements and electroencephalographic recordings. Sixty percent (37 of 62) of the Accutracker cuff inflations interrupted sleep and caused mild, short-term arousals or changes in sleep state. Transient blood pressure elevations lasted approximately 10 seconds (9.1 ± 2.8 seconds) as shown by the simultaneous beat-to-beat Finipress measurements during arousals caused by the Accutracker cuff inflation. However, the Accutracker measurement of systolic and diastolic blood pressure during deflation of the cuff started 15.6 ± 3.8 seconds after the elevated pressures monitored by the Finipress tracings had returned to pre-arousal pressure values.

Definition of Variables

We constructed several variables from the 24-hour blood pressure records (indicated by diary entries) to describe blood pressure at different time and activity periods. Average pressures for systolic, diastolic, and mean arterial pressures were calculated for the overall wakefulness period (beginning of study to "went to bed," excluding any daytime nap periods) and overall sleep period (between "went to bed" and "got up," excluding any awake readings as noted in the diary).

Systolic and diastolic loads were defined as the proportion of readings of 140 and 90 mm Hg or greater, respectively, during wakefulness and the proportion of readings of 127 and 79 mm Hg or greater, respectively, during sleep (26). Hypertension was defined by a mean waking systolic blood pressure of 140 mm Hg or greater or a mean diastolic blood pressure of 90 mm Hg or greater or both (27) or a history of diagnosis of hypertension and current use of antihypertensive medications.

Data Analysis

Data were analyzed using SAS statistical software (SAS Institute, Cary, North Carolina) for descriptive statistics, analysis of variance, and multiple logistic regression. Using analysis of variance and unpaired *t*-tests, the three study groups (apnea-hypopnea index ≥ 5 , snorers with apnea-hypopnea index < 5 , and nonsnorers with apnea-hypopnea index < 5) were compared on variables for mean blood pressure (systolic, diastolic, and mean arterial pressures), percent systolic and diastolic load, and heart rate during wake and sleep periods. Within-individual blood pressure variability during sleep was measured by the median of the individual coefficients of variation of the mean arterial pressure and by the square root of the pooled within-individual variances for systolic, diastolic, and mean arterial pressures (within-individual standard deviations). The coefficients of variation were compared using the Kruskal-Wallis test and the variances by *F*-tests (28). For all statistical tests, results with two-tailed *P* values < 0.05 were considered statistically significant.

The association of hypertension (as defined above) with sleep-disordered breathing (frequency of apneas and hypopneas during sleep indicated by apnea-hypopnea index as a continuous variable) was examined by multiple logistic regres-

sion analysis, controlling for obesity (body mass index ≥ 27 kg/m²), sex, and age (< 45 years, ≥ 45 years).

Results

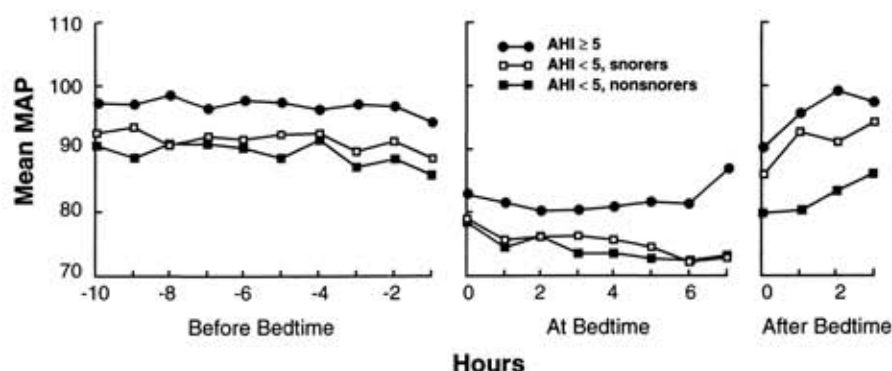
Fifty-three of the 147 participants had sleep apnea (apnea-hypopnea index ≥ 5). Of the 94 participants without apnea (apnea-hypopnea index < 5), 53 reported heavy or habitual snoring (snorers) and 41 did not (nonsnorers) (Table 1). In general, people with sleep apnea were older, more obese, and more likely to be men than those without apnea. Participants with sleep apnea had a median apnea-hypopnea index of 10.6 (range, 5 to 67).

All participants had total sleep times in excess of 240 minutes and achieved rapid eye movement (REM) stage of sleep. The average total sleep time was 375 (SD = 72.8) minutes. The distribution of time by sleep state (mean percent of total sleep time in each stage and SD) was 8.8% (5.2%) in stage 1, 63.8% (9.5%) in stage 2, 10.2% (8.1%) in stages 3 and 4, and 17% (6.3%) in REM. The mean (SD) number of REM periods was 3.5 (1.3). The percent of total sleep duration in each sleep state (with the exception of stages 3 and 4) of the study group was similar to normal values for adults (29).

Temporal plots of the mean blood pressures of all the participants averaged at each hour during a 24-hour period showed that participants with sleep apnea (apnea-hypopnea index ≥ 5) had consistently higher blood pressure values during both wakefulness and sleep than did snorers and nonsnorers with little or no apnea (apnea-hypopnea index < 5) (Figure 1).

Participants with sleep apnea had higher systolic blood pressure, diastolic blood pressure, and mean arterial pressures than did snorers and nonsnorers with little or no apnea during both wakefulness and sleep ($P < 0.05$; Table 2). The proportion of readings exceeding normative cut points for systolic blood pressure load during wakefulness was significantly higher for persons with sleep apnea compared with snorers and nonsnorers (30% compared with 16% and 14%, respectively). The systolic blood pressure load and heart rate during sleep and diastolic blood pressure load during wakefulness differed statistically only between participants with sleep apnea and nonsnorers with an apnea-hypopnea index of less than 5. Although the diastolic load during sleep was the highest in those with sleep apnea, the three groups did not differ statistically ($P = 0.10$).

Figure 1. Temporal plots of the average mean arterial pressures according to sleep-disordered breathing status. Mean arterial pressures (MAPs) of all participants ($n = 147$) averaged at each hour during the hours before bedtime, at bedtime, and after bedtime. AHI = apnea-hypopnea index (number of episodes of apnea and hypopnea per hour of sleep).



We found a significant difference in the variability of the blood pressure during sleep among the three groups (Table 3). The coefficient of variation for the average mean arterial pressures during sleep was higher for participants with sleep-disordered breathing (those with apnea-hypopnea index ≥ 5 and snorers with apnea-hypopnea index < 5) compared with participants without sleep-disordered breathing (nonsnорers with apnea-hypopnea index < 5): ten percent and 11% compared with 8%, respectively ($P = 0.05$). Pooled standard deviations of systolic, diastolic, and mean arterial pressures during sleep were 10.6, 9.2, and 8.9 mm Hg for persons with apnea; 10.2, 8.8, and 8.5 mm Hg for snorers without apnea; and 8.9, 7.8, and 7.5 mm Hg for nonsnорers without apnea, respectively. The values for participants with sleep-disordered breathing were significantly different from those of participants without sleep-disordered breathing ($P < 0.01$).

We found no significant difference in activity levels of the three groups at the time the blood pressure was measured during wakefulness: The proportions of blood

pressure readings during activity (walking or exercising) among the three groups were 41%, 53%, and 52% for nonapneic nonsnорers, nonapneic snorers, and apneic persons, respectively ($P > 0.2$).

Twenty-nine participants (19 with sleep apnea, 7 snorers, and 3 nonsnорers) were classified as hypertensive according to our definition. Thirty-six percent of those with apnea (19 of 53) were hypertensive compared with 13% (7 of 53) of snorers without apnea and 7% (3 of 41) of nonsnорers without apnea. The participants with apnea had a higher prevalence of hypertension compared with snorers and nonsnорers without apnea, regardless of body mass, sex, or age (Figure 2). Obese persons with apnea had a higher prevalence of hypertension than did obese snorers and nonsnорers without apnea (38%, 16%, and 8%, respectively). Further, among nonobese persons, those with sleep apnea were at least 2.8 times more likely to have hypertension than were snorers and nonsnорers without apnea (31%, 11%, and 7%, respectively). Similarly, the prevalence of hypertension was higher in persons with apnea compared with

Table 2. Ambulatory 24-Hour Blood Pressure Data in 147 Participants according to Sleep-disordered Breathing Stratum*

Blood Pressure Data	Apnea-Hypopnea Index		
	<5 in Nonsnорers	<5 in Snorers	≥ 5 in Apneics
Systolic blood pressure, mm Hg			
Wake	122 \pm 1.9	124 \pm 1.7	131 \pm 1.7†‡
Sleep	104 \pm 2.0	106 \pm 1.8	113 \pm 1.8†‡
Diastolic blood pressure, mm Hg			
Wake	75 \pm 1.2	76 \pm 1.1	80 \pm 1.1†‡
Sleep	62 \pm 1.3	63 \pm 1.2	66 \pm 1.1†‡
Mean arterial pressure, mm Hg			
Wake	91 \pm 1.4	92 \pm 1.2	97 \pm 1.2†‡
Sleep	76 \pm 1.5	77 \pm 1.3	82 \pm 1.3†‡
Systolic blood pressure load			
Wake, % ≥ 140	14 \pm 3.7	16 \pm 3.2	30 \pm 3.2†‡
Sleep, % ≥ 127	6 \pm 3.5	11 \pm 3.1	19 \pm 3.1†
Diastolic blood pressure load			
Wake, % ≥ 90	13 \pm 3.0	15 \pm 2.7	22 \pm 2.6†
Sleep, % ≥ 79	8 \pm 3.2	11 \pm 2.8	15 \pm 2.8
Heart rate, beats/min			
Wake	77 \pm 1.5	78 \pm 1.3	79 \pm 1.3
Sleep	63 \pm 1.4	66 \pm 1.2	68 \pm 1.2†

* Wake blood pressure = average of blood pressure measurements taken from beginning of study to bedtime and after awakening from sleep; sleep blood pressure = average of all blood pressure measurements taken during bedtime, excluding any awake readings; systolic and diastolic blood pressure loads = percent systolic and diastolic blood pressure measurements $\geq 140/90$ mm Hg during wakefulness and percent systolic and diastolic blood pressure measurements $\geq 127/79$ mm Hg during sleep. All values are expressed as mean \pm SE.

† Apneics and nonapneics different at $P \leq 0.05$.

‡ Apneics and snorers different at $P \leq 0.05$.

snorers and nonsnorers without apnea among both men and women and both older and younger age groups.

Multiple logistic regression analysis controlling for age, sex, and body mass index showed that apnea-hypopnea index was independently associated with hypertension. We found that the log odds of hypertension varied linearly with apnea-hypopnea index across the range of mild-to-moderate sleep apnea (apnea-hypopnea index from 5 to 25) in the model. Odds ratios ranged from 2.0 to 5.0 for hypertension associated with an apnea-hypopnea index of 5 to 25 compared with that of an apnea-hypopnea index of less than 5 in a dose-response fashion.

Discussion

Our main findings were that during both wakefulness and sleep, participants with sleep apnea had higher serial mean blood pressures than snorers and nonsnorers without sleep apnea; participants with sleep-disordered breathing (those with sleep apnea and habitual snorers without sleep apnea) had greater variability of blood pressures during sleep; and participants with sleep apnea, regardless of obesity, age, and sex, had a higher prevalence of hypertension. The association between sleep apnea and blood pressure was present even though the participants with sleep apnea in our study were asymptomatic and had less severe apnea than is generally seen in sleep disorders clinic populations.

Our study avoided selection bias by studying people derived from the same defined sampling population and recruited identically. The study sample was drawn from a surveyed population with known sleep characteristics and sociodemographic characteristics, allowing us to generalize our findings to working adults.

An important strength of our study was the use of an ambulatory continuous 24-hour blood pressure monitor (in contrast to the casual clinic blood pressure measurements used in previous studies), which provided more representative and accurate measurements of arterial pressures during sleep and wakefulness as well as additional information about temporal trends and variations of blood pressure during sleep and wakefulness. Several studies have shown that 24-hour ambulatory blood pressures are more reproducible and representative of the true arterial pressure and also more predictive of hypertensive cardiovascular outcomes than casual clinic blood pressure measurements (30–41).

Our data extend previous findings by showing a significant association between hypertension and sleep apnea even after adjusting for body mass, age, and sex. Obesity, older age, and male sex are recognized risk factors for both sleep-disordered breathing (24) and hypertension (42–44). We found that obese persons with an apnea-hypopnea index of 5 or greater were five times more likely to have hypertension than similarly obese persons without sleep-disordered breathing. An even more striking finding was that persons with an apnea-hypopnea index of 5 or greater had a high prevalence of hypertension whether they were obese (38%) or not (31%). These results indicate that episodes of apnea and hypopnea during sleep do contribute independently to sustained elevated blood pressure.

Our data did not support an association between hy-

pertension and habitual snoring history, independent of co-existing sleep apnea, after controlling for age, sex, and body mass index. This finding is contrary to earlier epidemiologic surveys that have linked snoring and hypertension but concurs with the only other study that had polysomnographic data to assess adequately the association, although the latter study used only single office and clinic daytime pressures (14). Together, these findings suggest that in earlier surveys of hypertension and snoring, the apneic snorers, included among the snoring groups, accounted for the reported relation. It is important to note, however, that the snoring status in our study was based on self-report. Misclassification of snoring would tend to reduce the ability to detect a risk. Despite the lack of association between snoring and hypertension, we found that snorers showed increased blood pressure variability similar to persons with apnea. Thus, the snorers in our study may be showing early, acute effects of sleep-disordered breathing and associated transient arousals on blood pressure during sleep and may, over time, develop sustained daytime and night-time elevations in blood pressure similar to persons with apnea.

A concern regarding use of intermittent blood pressure readings during sleep is the possible effect of the automatic cuff inflation on arousals or sleep state and, in turn, on blood pressure. We found mild, short-term arousals or changes in sleep state in 60% of cuff inflations during overnight monitoring of volunteers with continuous Finipress measurements during simultaneous Accutacker pressure measurements and electroencephalographic recordings. However, we also showed that pressure increases during cuff inflation did not last long enough to influence the subsequent determination of pressure by Finipress measurements during sleep. Our data agree with previously published data based solely on measurements during wakefulness (45). Thus, we believe that the actual blood pressure measurements by Accu-

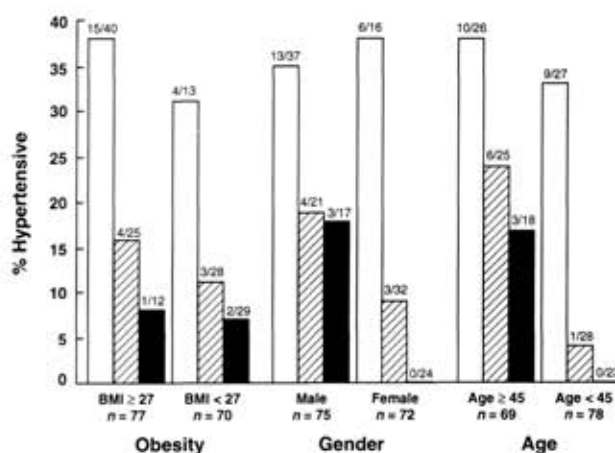


Figure 2. Prevalence of hypertension among participants with and without sleep-disordered breathing stratified by obesity, sex, and age. Frequency distribution of hypertension in participants with sleep-disordered breathing (apnea-hypopnea index ≥ 5 as indicated by open bars, snorers with an apnea-hypopnea index ≥ 5 as indicated by hatched bars) and without sleep-disordered breathing (nonsnorers with an apnea-hypopnea index < 5 as indicated by shaded bars). BMI = body mass index measured in kg/m².

Table 3. Variability of Blood Pressure during Sleep according to Sleep-disordered Breathing Stratum

Blood Pressure Variability	Apnea-Hypopnea Index		
	<5 in Nonsnorers	<5 in Snorers	≥5 in Apneics
Coefficient of variation, %			
%SD/mean arterial blood pressure	8	11*	10*
Within-participant variation			
Systolic blood pressure, mm Hg	8.9	10.2†	10.6†
Diastolic blood pressure, mm Hg	7.8	8.8†	9.2†
Mean arterial blood pressure, mm Hg	7.5	8.5†	8.9†

* Apneics and snorers greater than nonapneics at $P = 0.05$.

† Apneics and snorers greater than nonapneics at $P < 0.01$.

tracker devices were not affected by the transient arousals caused by cuff inflation either during sleep or wakefulness. This impression is confirmed by the similarity we obtained in mean systolic and diastolic pressures in the volunteers during interrupted and uninterrupted sleep ($101/59 \pm 14/11$ mm Hg compared with $101/60 \pm 15/9$ mm Hg, respectively). Schwan and colleagues (46) have described similar findings in 24 patients.

Arterial blood pressure is affected by activity (47, 48). A difference in activity levels among the three groups could bias the measurement of the association between apnea and blood pressure during wakefulness. However, we found no significant difference in the proportion of blood pressure measurements taken during walking or exercise in the three groups.

It has been hypothesized that the normal decline in blood pressure during sleep is diminished in persons with sleep apnea. Indeed, transient elevations of blood pressure have been observed during sleep immediately after acute apneic episodes in patients with severe obstructive sleep apnea syndromes (1, 16, 17). We have also observed significant transient increases in blood pressure during the much less severe episodes of apnea and hypopnea in a subsample of the sleep cohort participants from our study who were studied by continuous blood pressure monitoring during overnight polysomnography (unpublished data). These pressor responses to sleep-disordered breathing events are believed to result from the sympatho-excitation provided by a combination of the mild to moderate asphyxia and the transient arousal accompanying the apneic or hypopneic event (49–51).

We were not able to address with certainty whether there was a difference in blood pressure decline among the three study groups caused by an independent sleep effect. Our data indicated that each group's average blood pressure during bedtime was 15 mm Hg lower than it was before bedtime. However, interpretation of these data with respect to the role of sleep in blood pressure reduction is difficult because the data are confounded by postural changes from upright to recumbent position. Previous studies have shown that posture affects blood pressure reactivity (52–54). In addition, without simultaneous electroencephalographic data, we were not able to measure the sleep state objectively. When the blood pressure readings were taken during the time in bed, some participants may still have been awake, particularly during the early part of the night. Thirdly, the Accutacker blood pressure measurements,

lasting 20 to 30 seconds, were done at 30-minute intervals during sleep. This sampling period is unlikely to capture many of the acute elevations in blood pressure associated with sleep-disordered breathing events, which occurred at an average of 10 per hour. On the other hand, we did find higher variability of Accutacker blood pressure measurements during sleep among participants with sleep-disordered breathing, which probably reflected the fluctuation of blood pressure associated with sleep-disordered breathing events.

We showed that asymptomatic sleep-disordered breathing, ranging from mild to moderate severity, is an independent risk factor for hypertension and elevated blood pressure over a 24-hour period in a population-based sample representative of working adults. It has been strongly recommended by the National Commission on Sleep Disorders Research (55) and others (56, 57) that, given the recently recognized high prevalence of undiagnosed sleep-disordered breathing in the adult population, all primary care clinicians inquire more frequently about sleep-related breathing problems for early detection and referral of patients with sleep apnea. A concern with this recommendation has been whether asymptomatic sleep apnea is of clinical significance. Our findings of the high prevalence of hypertension in people with asymptomatic sleep apnea support the importance of incorporating a sleep history in the general systems review of all patients seen in the primary care setting.

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