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Prospective Study of Snoring and Risk of Hypertension in Women

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Whether snoring increases the risk of hypertension remains unclear. The authors examined the association between snoring and risk of hypertension in a cohort of 73,231 US female nurses aged 40–65 years and without diagnosed cardiovascular disease or cancer in 1986. Blood pressure levels and physician-diagnosed hypertension were self-reported through validated questionnaires. During 8 years of follow-up, 7,622 incident cases of physician-diagnosed hypertension were reported. Older age, smoking, body mass index, waist circumference, waist-hip ratio, weight gain, less physical activity, and sleeping on the back were directly associated with regular snoring. After adjustment for age, body mass index, waist circumference, and other covariates, snoring was associated with a significantly higher prevalence of hypertension at baseline (odds ratio = 1.22, 95% confidence interval (CI): 1.16, 1.27 for occasional snoring and odds ratio = 1.43, 95% CI: 1.33, 1.5 for regular snoring). In prospective analyses using incident cases of hypertension as the outcome, the multivariate relative risks of hypertension were 1.29 (95% CI: 1.22, 1.37) for occasional snoring and 1.55 (95% CI: 1.42, 1.70) for regular snoring. In addition, snoring was associated with significantly higher systolic and diastolic blood pressure levels. These data suggest that snoring may increase risk of hypertension in women, independent of age, body mass index, waist circumference, and other lifestyle factors. *Am J Epidemiol* 1999;150:806–16.

blood pressure; hypertension; snoring; women

Systemic hypertension is highly prevalent among patients with obstructive sleep apnea syndrome (1, 2), and sleep apnea tends to occur more frequently among patients with hypertension (3, 4). In several studies, reductions in blood pressure after successful treatment of obstructive sleep apnea were observed (5, 6), supporting the hypothesis that sleep apnea is causally related to hypertension. A causal relation between sleep apnea and hypertension is biologically plausible because sleep apnea causes hypoxemia, hypercapnia, and arousal from sleep, which, in turn, chronically activates the sympathetic nervous system (7).

Snoring may increase risk of hypertension through sleep apnea because it is a cardinal symptom of obstructive sleep apnea (8). However, most snorers do not have sleep apnea. Whether snoring itself increases the risk of hypertension and cardiovascular disease remains controversial (9–11). A positive association between regular snoring and hypertension has been found in several studies (4, 12–15). However, some did not control for body weight or body mass index (BMI) (4, 12), and none were prospective. Several other cross-sectional studies failed to confirm a significant positive association between snoring and elevated blood pressure after adjustment for BMI (16–19). Thus, confounding by overweight and obesity has been suggested as a possible alternative explanation for the positive association found in previous studies (10).

In this report, we examined the relation of snoring to risk of hypertension, both cross-sectionally and prospectively, among participants in the Nurses' Health Study. Detailed data on risk factors for hypertension, including BMI, weight change, waist circumference, and alcohol use were accounted for in the analysis. A secondary aim of this analysis was to examine predictors of regular snoring in women.

MATERIALS AND METHODS

Study population

The Nurses' Health Study cohort was established in 1976 when 121,700 married female registered nurses aged 30–55 years completed a mailed questionnaire on

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Abbreviations: BMI, body mass index; CI, confidence interval; MET, metabolic equivalent; RR, relative risk.

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their medical history and lifestyle. Every 2 years, follow-up questionnaires have been sent to update information on potential risk factors and to identify newly diagnosed cases of hypertension and coronary and other diseases. On the 1986 questionnaire, we asked the following: 1) Indicate total hours of actual sleep in a 24-hour period (response categories from ≤5 hours to ≥11); 2) What is your usual sleeping position? (on back, on side, on front); 3) Do you snore? (regularly, occasionally, or never)? A total of 76,195 women responded to these questions. After the exclusion of women with diagnosed cardiovascular disease or cancer at baseline, the final population for analysis included 73,231 women. In the prospective analyses using incidence of hypertension as the outcome (described below), we also excluded women who had hypertension at baseline (n = 17,511).

Assessment of hypertension

Incident cases of hypertension were identified by self-reports of physician-diagnosed hypertension. On the 1986 and all subsequent biennial questionnaires, we asked whether hypertension (except that occurring during pregnancy) had been diagnosed by a physician and, if so, the date of diagnosis. In 1986, 1988, 1990, and 1994, participants were also asked to report their usual systolic and diastolic blood pressures, choosing among the following categories: in 1986, less than 75, 75–84, 85–89, 90–94, 95–104, and ≥105 mmHg for diastolic pressure and less than 120, 120–139, 140–149, 150–159, 160–169, and ≥170 mmHg for systolic pressure, and in 1988, 1990, and 1994, less than 65, 65–74, 75–84, 85–89, 90–94, 95–104, and ≥105 mmHg for diastolic pressure and less than 115, 115–124, 125–134, 135–144, 145–154, 155–164, 165–174, and ≥175 mmHg for systolic pressure. For each individual, we assigned the mean blood pressure value for the category reported, e.g., we assigned a value of 145 mmHg to the category 140–149 mmHg. For the extreme categories, we added 5 mmHg to the highest value and subtracted 5 mmHg from the lowest value, e.g., we assigned a value of 60 mmHg to the category <65 mmHg and a value of 110 mmHg to the category ≥105 mmHg. For those who reported taking antihypertensive medication but whose blood pressure levels were less than 160/95 mmHg, we assigned the values of 160/95 mmHg.

The validity of self-reported diagnosis of hypertension was assessed in a random sample of 100 nurses who had reported a diagnosis of high blood pressure on the 1982 questionnaire (20). Of the 85 women who responded to a supplementary questionnaire, all but one confirmed their previous reports of hypertension; 62 of 85 women gave written permission for review of their medical records. We obtained records for 51 women; all of them had blood pressure measurements higher than 140/90 mmHg, and 39 (76 percent) had blood pressure measurements greater than 160/95 mmHg. To investigate the likelihood of false-negative responses, blood pressure was measured in another sample of 194 nurses living in the Boston area. Among the 161 women without a previous self-report of hypertension, 7 percent had a blood pressure level higher than 140/90 mmHg, but none had a level greater than 160/95 mmHg. In addition, self-reported physician diagnosis of hypertension is a strong predictor of myocardial infarction and stroke in this cohort (21), providing further evidence for the validity of self-reported hypertension.

Assessment of covariates

The 1986 questionnaire included items on age, current weight, smoking status, physical activity, alcohol use, and other variables. Updated information on these variables was collected on biennial follow-up questionnaires. In a validation study, self-reported weight was highly correlated with actual weight (r = 0.96) as measured by a technician (22). We calculated weight change from 1976 to 1986. In 1986, the participants measured and reported their waist (measured at the umbilicus) and hip (measured at the largest circumference) measurements to the nearest quarter of an inch by using a tape measure. For validation of these measurements, self-reported measures in a sample of 140 nurses were compared with two standardized measurements, taken approximately 6 months apart, by technicians who visited participants in their homes. The correlations between self-reported and technician-measured circumferences were 0.89 for the waist, 0.84 for the hip, and 0.70 for the waist-hip ratio (22). The biennial questionnaires included questions about time per week engaged in 10 specified physical activities and four sedentary activities during the previous year (23). Metabolic equivalents (METs) per week were defined for each activity as a multiple of the metabolic equivalent of sitting quietly for 1 hour. For example, a participant who runs (7 METs/hour) 1 hour a day three times a week would have a MET score of 21.

In 1972, all of the nurses were married. We reassessed their marital status in 1980 (married, divorced, or widowed). In 1992, we asked the participants whether they lived with their spouses or partners.

Statistical analysis

To examine correlates of snoring, we performed multivariate logistic regression analyses to predict

TABLE 1. Prevalence and predictors of regular snoring in the Nurses' Health Study, 1986

	No. of women*	Prevalence of occasional snoring (%)	Occasional snoring vs. nonsnoring†		Prevalence of regular snoring (%)	Regular snoring vs. nonsnoring†	
			Multivariate OR	95% CI‡		Multivariate OR	95% CI
Age (years)							
<45	13,290	55.8	1.0	Reference	6.7	1.0	Reference
45–49.9	14,783	61.0	1.29	1.22, 1.35	8.2	1.41	1.27, 1.56
50–54.9	15,701	66.5	1.79	1.69, 1.88	10.1	2.14	1.93, 2.37
55–59.9	14,347	68.4	2.14	2.02, 2.26	11.8	2.96	2.66, 3.29
≥60	15,110	71.2	2.48	2.35, 2.63	10.8	3.06	2.75, 3.40
Smoking							
Past smoking (years since quitting)	32,322	62.3	1.0	Reference	7.9	1.0	Reference
>10	7,667	65.1	1.08	1.02, 1.15	9.2	1.27	1.14, 1.41
5.1–10	7,613	64.4	1.19	1.12, 1.26	8.7	1.37	1.23, 1.53
≤5	9,659	66.6	1.42	1.34, 1.50	10.9	1.91	1.74, 2.11
Current smoking (cigarettes/day)							
1–14	4,951	67.9	1.53	1.42, 1.65	9.3	1.90	1.67, 2.17
15–24	9,115	69.4	2.00	1.88, 2.13	12.4	3.25	2.94, 3.60
≥25	1,553	66.7	2.12	1.84, 2.46	17.8	4.75	3.88, 5.80
Body mass index in 1986 (kg/m²)							
<21	10,985	58.8	1.0	Reference	4.3	1.0	Reference
21–25	30,230	63.8	1.23	1.17, 1.30	6.4	1.59	1.41, 1.78
25.1–27	10,337	67.4	1.54	1.44, 1.65	9.4	2.58	2.24, 3.00
27.1–29.9	9,878	68.6	1.96	1.82, 2.11	13.1	4.16	3.61, 4.80
≥30	10,271	67.3	3.11	2.85, 3.38	21.1	10.6	9.2, 12.3
Waist circumference (inches)§							
<26	2,522	54.6	1.0	Reference	3.5	1.0	Reference
26–29.9	17,713	61.1	1.19	1.08, 1.30	4.8	1.35	1.06, 1.72
30–33.9	14,965	67.9	1.45	1.31, 1.60	8.2	1.94	1.52, 2.49
34–37.9	6,997	69.2	1.52	1.35, 1.70	13.8	2.35	1.82, 3.05
≥38	3,706	66.0	1.68	1.44, 1.94	23.1	3.63	2.75, 4.79
Waist/hip ratio							
<0.72	7,745	59.2	1.0	Reference	4.5	1.0	Reference
0.72–0.75	11,517	62.4	1.09	1.02, 1.16	5.8	1.25	1.08, 1.44
0.76–0.79	8,850	65.8	1.20	1.12, 1.29	8.0	1.54	1.32, 1.79
0.80–0.87	11,912	68.0	1.35	1.26, 1.44	11.7	2.04	1.77, 2.35
≥0.88	3,419	67.0	1.38	1.24, 1.54	17.0	2.58	2.15, 3.08
Body mass index in 1976 (kg/m²)							
<21	19,279	60.0	1.0	Reference	4.8	1.0	Reference
21–25	33,873	65.3	1.36	1.30, 1.41	8.1	2.07	1.90, 2.25
25.1–27	7,800	69.7	2.07	1.93, 2.22	12.1	4.50	4.02, 5.05
27.1–29.9	6,257	69.5	2.65	2.45, 2.87	16.0	7.42	6.57, 8.38
≥30	5,347	67.2	3.87	3.50, 4.27	22.6	16.4	14.4, 18.8

Table continues

occasional snoring versus nonsnoring and regular snoring versus nonsnoring separately. The model included age (<45, 45–49.9, 50–54.9, 55–59.9, and ≥60 years); smoking status (never, past, and current smoking of 1–14, 15–24, and ≥25 cigarettes per day); BMI (<21, 21–25, 25.1–27, 27.1–29.9, and ≥30); waist circumference (<26, 26–29.9, 30–33.9, 34–37.9, ≥38 inches (<66.0, 66.0–76.0, 76.1–86.1, 86.2–96.3, and ≥96.4 cm)); alcohol use (0, 0.1–4.9, 5–14.9, and ≥15 g/day); physical activity (quartiles of weekly METs score); usual sleep position (on side, on back, on front, and mixed position), and usual sleeping hours (≤5, 6, 7–8, and ≥9). In a separate model, we included waist-hip ratio (<0.72, 0.72–0.75, 0.76–0.79, 0.80–0.87, and ≥0.88) rather than waist circumference. In an additional analysis, we included BMI assessed in 1976 and weight change between 1976 and 1986 (lost ≤15 kg, lost 2–14.9 kg, lost or gained ≥2 kg, gained 2–14.9 kg,

TABLE 1. Continued

	No. of women	Prevalence of occasional snoring (%)	Occasional snoring vs. nonsnoring		Prevalence of regular snoring (%)	Regular snoring vs. nonsnoring	
			Multivariate OR	95% CI		Multivariate OR	95% CI
Weight change between 1976 and 1986							
Lost ≤15 kg	16,530	63.2	0.57	0.42, 0.75	6.8	0.45	0.30, 0.68
Lost 2–14.9 kg	7,628	66.8	0.95	0.89, 1.02	8.8	0.80	0.71, 0.90
Lost or gained ≥2 kg	2,332	66.0	1.0	Reference	11.5	1.0	Reference
Gained 2–14.9 kg	42,548	65.0	1.24	1.19, 1.29	9.4	1.58	1.45, 1.71
Gained 15–24.9 kg	3,465	64.0	1.62	1.46, 1.79	18.6	3.47	3.01, 4.01
Gained >25 kg	728	65.4	2.43	1.93, 3.08	22.8	6.09	4.54, 8.16
Current alcohol use (g/day)							
0	22,721	63.6	1.0	Reference	10.4	1.0	Reference
0.1–4.9	21,039	64.8	1.07	1.03, 1.12	8.7	0.97	0.89, 1.05
5–14.9	13,128	65.7	1.11	1.05, 1.17	7.9	1.00	0.91, 1.10
≥15	8,650	67.5	1.21	1.13, 1.29	9.9	1.26	1.13, 1.41
Physical activity (quartiles of METS¶ score)							
<2.7	18,139	64.7	1.0	Reference	12.3	1.0	Reference
2.7–7.7	19,063	65.5	1.01	0.96, 1.07	10.1	0.90	0.83, 0.98
7.8–19	17,806	64.8	0.96	0.91, 1.07	8.2	0.73	0.67, 0.80
>19	17,980	63.8	0.92	0.87, 0.96	7.0	0.66	0.61, 0.73
Usual sleep positions							
On side	59,200	65.2	1.0	Reference	9.4	1.0	Reference
On back	4,855	63.3	1.08	1.01, 1.16	11.5	1.59	1.41, 1.79
On front	8,119	62.5	0.83	0.79, 0.88	8.0	0.71	0.64, 0.78
Mixed position	641	63.7	0.94	0.78, 1.14	12.3	1.31	0.96, 1.80
No. of hours of sleep per day							
≤5	3,236	65.1	0.95	0.87, 1.04	10.4	0.94	0.81, 1.10
6	18,613	65.9	1.07	1.03, 1.12	10.0	1.08	1.00, 1.16
7–8	47,718	64.5	1.0	Reference	8.9	1.0	Reference
≥9	3,347	62.2	0.91	0.84, 0.99	11.8	1.06	0.92, 1.22

* Numbers across categories for some variables do not add up to the total because of missing data.

† The odds ratios (OR) were obtained from a model that included age, smoking, alcohol use, body mass index in 1986, waist circumference, physical activity, and usual sleep positions and hours. The odds ratios for waist-hip ratio were obtained from another model that included all of the above variables except waist circumference. The odds ratios for body mass index in 1976 and weight gain between 1976 and 1986 were obtained from a separate model with age, smoking, alcohol use, body mass index in 1976, weight gain between 1976 and 1986, physical activity, and usual sleep positions and hours.

‡ CI, confidence interval.

§ 1 inch = 2.54 cm.

¶ METS, metabolic equivalents per week (see Materials and Methods).

gained 15–24.9 kg, and gained ≥25 kg), and other covariates. We did not include BMI or waist circumference assessed in 1986 in this model.

To examine the association between snoring and blood pressure levels, we used the self-reported blood pressure levels in 1986, 1988, 1990, and 1994 as dependent variables (in separate models). We used multiple linear regression models to estimate differences in blood pressure levels according to categories of snoring (nonsnoring as reference group), adjusting for age, age squared, smoking status (never, past, and current smoking of 1–14, 15–24, and ≥25 cigarettes per day), BMI (deciles), waist circumference (seven categories), alcohol consumption (0, 1–4, 5–14, and ≥15 g/day), and physical activity (quartiles of MET hours per week). A quadratic term for age was included to account for the nonlinearity of the age-blood pressure association and to control tightly for the potential confounding effect of age. Analyses using 5-year categories yielded similar results.

In the analyses of diagnosed hypertension as the outcome, we conducted both cross-sectional and prospective analyses. We used a logistic model to predict prevalence of hypertension at baseline. Odds

ratios of hypertension were calculated using nonsnorers as the reference group. In the prospective analyses of incidence of hypertension as the dependent variable, follow-up time for each category of snoring (nonsnoring and occasional and regular snoring) accrued until the date of diagnosis of hypertension; use of antihypertensive medications; or censoring due to diagnosis of myocardial infarction, angina pectoris, coronary artery surgery, stroke, death, or June 1, 1994, whichever came first. Incidence rates were calculated by dividing the number of events by person-time of follow-up in each category. The relative risk was computed as the rate in a specific category of the exposure divided by that in the reference category (nonsnorers), with adjustment for age. In multivariate analyses using pooled logistic regression (24), we simultaneously adjusted for potential confounding variables, including age, age squared, BMI in 1986, waist circumference, smoking status, physical activity, and alcohol use. We did not adjust for sleep position and sleeping hours because neither variable was associated with hypertension. In additional analyses, we adjusted for marital status and living arrangements.

RESULTS

At baseline, 17,511 women (23.9 percent) had reported physician-diagnosed hypertension. During 8 years of follow-up (358,928 person-years), we ascertained 7,622 incident cases of hypertension. Approximately 26 percent of women reported never snoring, 65 percent reported occasional snoring, and 9 percent regular snoring. Five percent of women reported sleeping 5 hours or less a day, 25 percent reported 6 hours, 41 percent reported 7 hours, 24 percent reported 8 hours, and 5 percent reported 9 hours or more. About 81 percent women reported usually sleeping on their side, 7 percent on their back, and 11 percent on their front.

In 1972, all participants were married, and in 1980, the majority of women (84 percent) remained married. The prevalence of regular snoring was slightly higher among those who were divorced or widowed (11 percent) than among those who remained married (9 percent). In 1992, 74 percent of the women reported living with their spouses or partners. The prevalence of regular snoring was similar between those who were living with spouses or partners (9 percent) and those were not (10 percent).

Older women were more likely to snore regularly (table 1), but the prevalence of regular snoring did not increase further after age 60 years. Past smokers, even those who had stopped smoking more than 10 years before, had a significantly higher prevalence of regular snoring than did never smokers. Among current smokers, the risk of snoring increased with increasing number of cigarettes smoked per day. BMI assessed in 1986 was strongly associated with regular snoring. Obese women (BMI ≥ 30 kg/m^2) were approximately 11 times more likely to snore regularly than were lean women (BMI ≤ 21 kg/m^2). After BMI was controlled for, both waist circumference and waist-hip ratio were significantly associated with regular snoring. After adjustment for BMI in 1976, women who lost weight during the subsequent 10-year period had a lower prevalence of regular snoring compared with women who had stable weight (loss or gain ± 2 kg). In contrast, women who had gained weight had a significantly increased risk of regular snoring. When BMIs for both 1976 and 1986 were entered into the model simultaneously, the associations were substantially attenuated for the 1976 BMI (odds ratio for BMI ≥ 30 kg/m^2 = 2.85, 95 percent confidence interval (CI): 2.39, 3.40), but the association for the 1986 BMI remained strong (odds ratio for BMI ≥ 30 kg/m^2 = 8.58, 95 percent CI: 7.21, 10.2).

In the multivariate analyses, physical activity was inversely associated with snoring; more active women were significantly less likely to snore (table 1). Those who drank 15 g of alcohol or more per day were slightly more likely to snore regularly than were nondrinkers. Compared with women who usually slept on their side, women who usually slept on their back were more likely to snore, while women usually slept on their front were less likely to snore. The number of hours of sleep per day was not appreciably associated with regular snoring. The predictors of occasional snoring were similar to those for regular snoring, although the magnitude of the associations was somewhat weaker.

Table 2 shows average reported systolic and diastolic pressure differences between snorers and nonsnorers, adjusted for age, smoking, BMI in 1986, waist circumference, alcohol use, and physical activity. Compared with nonsnorers, blood pressure levels reported in 1986, 1988, 1990, and 1994 were all significantly increased among occasional snorers and further increased among regular snorers. The average difference in systolic pressure between regular snorers and nonsnorers was 2.0 mmHg, and the difference in diastolic pressure was about 1.2 mmHg. The R^2 for these models including snoring and covariates ranged from 0.18 to 0.21 for systolic pressure and from 0.08 to 0.14 for diastolic pressure.

Figure 1 shows the distribution of blood pressure among those who did not report physician-diagnosed hypertension or take antihypertensive medication. Clearly, the distribution has shifted across the snoring categories. The prevalence of physician-diagnosed

TABLE 2. Average systolic and diastolic blood pressure differences and 95% confidence intervals between regular and occasional snorers and nonsnorers*, Nurses Health Study, 1986

Pressure (mmHg)	Nonsnorers (difference)	Occasional snorers		Regular snorers	
		Difference	95% CI†	Difference	95% CI
1986 (n = 72,649)					
Systolic	0 (reference)	0.99	0.79, 1.19	1.87	1.54, 2.20
Diastolic	0	0.63	0.51, 0.75	1.27	1.05, 1.49
1988 (n = 66,311)					
Systolic	0	1.31	1.09, 1.53	2.03	1.68, 2.38
Diastolic	0	0.71	0.57, 0.85	1.25	1.03, 1.47
1990 (n = 64,944)					
Systolic	0	1.26	1.01, 1.51	1.75	1.34, 2.16
Diastolic	0	0.85	0.69, 1.01	1.25	1.00, 1.50
1994 (n = 67,149)					
Systolic	0	1.40	1.15, 1.66	2.13	1.72, 2.54
Diastolic	0	0.78	0.62, 0.94	1.15	0.90, 1.40

* Snoring reported in 1986. Data were adjusted for age, age^2, smoking (never, past, current (1–14, 15–24, and ≥25 cigarettes/day)), body mass index (deciles), waist circumference (<28, 28–29.9, 30–31.9, 32–33.9, 34–35.9, 36–37.9, and ≥38 inches (1 inch = 2.54 cm)), physical activity (metabolic equivalents score in quartiles), and alcohol use (four categories).

† CI, confidence interval.

hypertension increased monotonically across categories of snoring in each stratum of age (figure 2), BMI (1986) (figure 3), and waist-hip ratio (figure 4). In the cross-sectional analyses (table 3), snoring was significantly associated with prevalence of hypertension at baseline. The odds ratios of hypertension adjusted for age and age squared were 1.46 (95 percent CI: 1.40, 1.53) for occasional snoring and 2.22 (95 percent CI: 2.08, 2.37) for regular snoring. These odds ratios were attenuated after further adjustment for smoking, BMI, waist circumference, and other covariates but remained statistically significant (odds ratios = 1.22, 95 percent CI: 1.16, 1.27 for occasional snoring and odds ratio = 1.43, 95 percent CI: 1.33, 1.53 for regular snoring).

In the prospective analyses (table 3), snoring was significantly associated with incidence of hypertension between 1986 and 1994. The multivariate relative risks (RRs) were 1.29 (95 percent CI: 1.22, 1.37) for occasional snoring and 1.55 (95 percent CI: 1.42, 1.70) for regular snoring. The RRs were not altered when BMI and waist circumference were entered into the model as continuous variables (RR = 1.31, 95 percent CI: 1.23, 1.39 for occasional snoring and RR = 1.55, 95 percent CI: 1.42, 1.70 for regular snoring). The results were similar in the analysis adjusting for the 1976 BMI and weight change (RR = 1.29, 95 percent CI: 1.22, 1.3 for occasional snoring and RR = 1.54, 95 percent CI: 1.41, 1.68 for regular snoring). Finally, further adjustment for marital status and living arrangements did not alter the results (RR = 1.30, 95 percent CI: 1.23, 1.38 for occasional snoring and RR = 1.55, 95 percent CI: 1.41, 1.69 for regular snoring).

We conducted stratified analyses of snoring and incidence of hypertension according to categories of the 1986 BMI. Among women with a BMI of less than 25 kg/m^2, the multivariate RRs of hypertension were 1.39 (95 percent CI: 1.29, 1.50) for occasional snoring and 1.74 (95 percent CI: 1.54, 1.97) for regular snoring; the corresponding RRs were 1.23 (95 percent CI: 1.09, 1.39) and 1.57 (95 percent CI: 1.32, 1.87) for women who had BMIs of 25–29.9 kg/m^2 and 1.21 (95 percent CI: 0.99, 1.47) and 1.45 (95 percent CI: 1.15, 1.83) for women who had BMIs of 30 kg/m^2 or greater.

DISCUSSION

In this large cohort of women, snoring, especially regular snoring, was associated with significantly increased risk of hypertension in both cross-sectional and prospective analyses. Systolic and diastolic pressure levels were highest among regular snorers, lowest among nonsnorers, and intermediate among occasional snorers.

We confirmed several predictors of snoring found in other populations,(16, 25), such as age, smoking, and obesity. We also found several additional predictors, including fat distribution reflected by waist circumference and waist-hip ratio, weight change, physical activity, and usual sleep position. In particular, women who had lost weight had a lower prevalence of regular snoring, whereas women who had gained weight had a

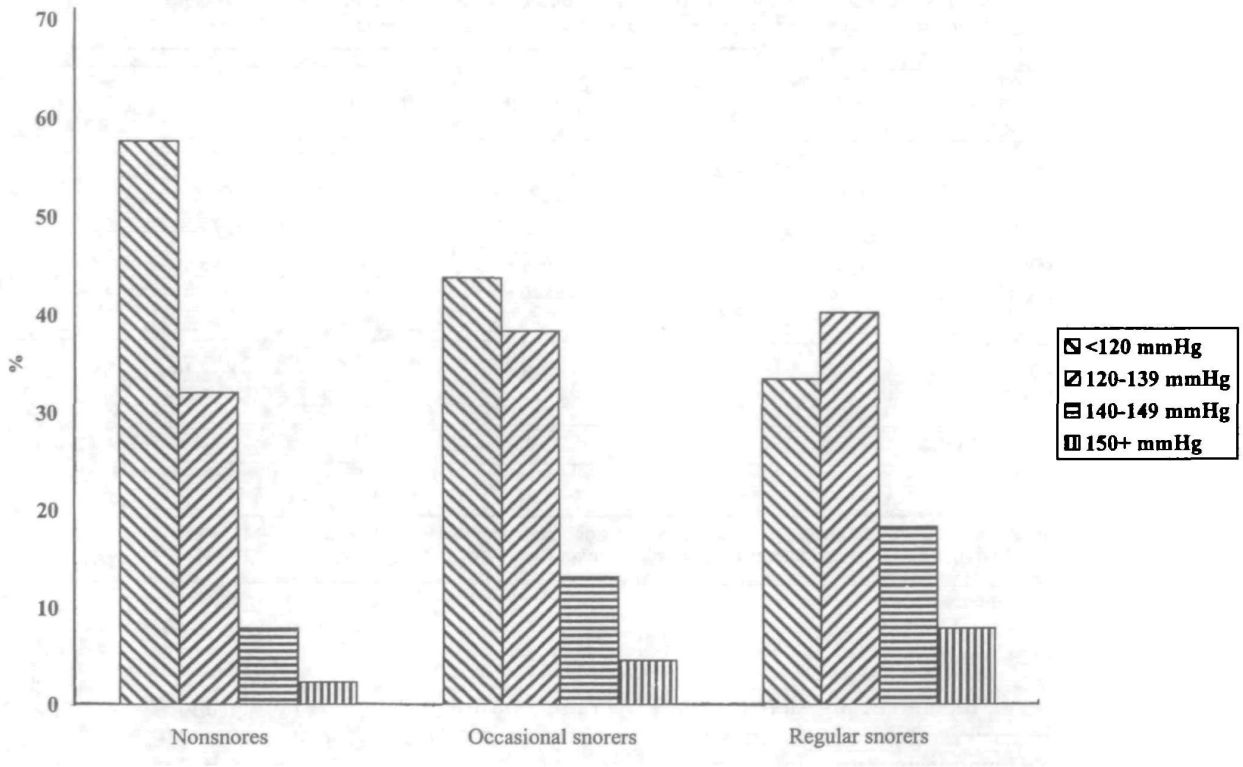

FIGURE 1. Distribution of systolic pressure according to snoring categories in the Nurses' Health Study, 1986. Women who reported physician-diagnosed hypertension or took antihypertensive medications were not included.

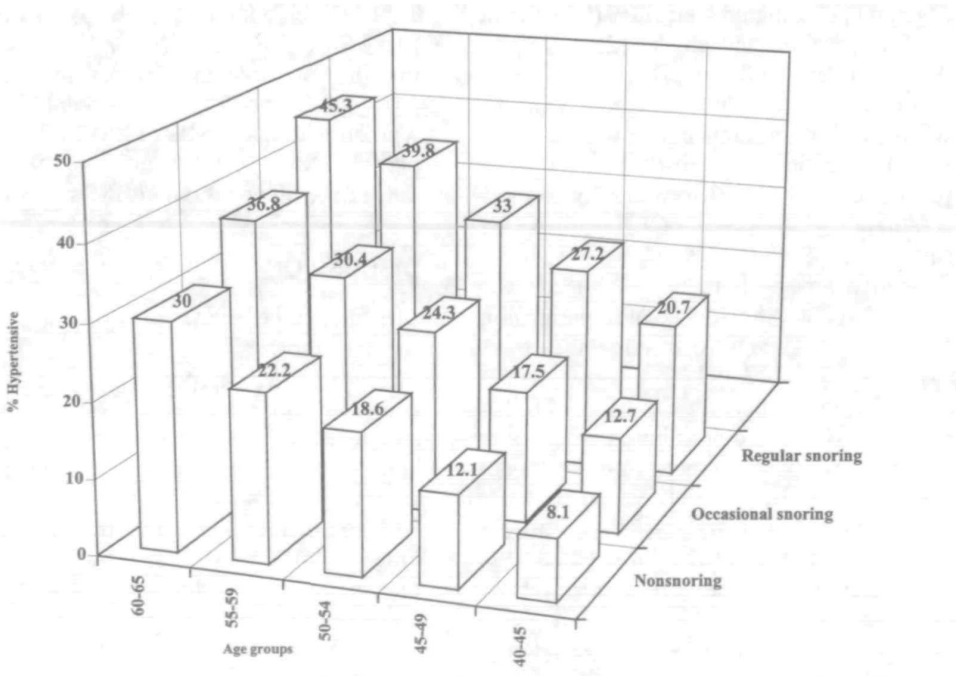

FIGURE 2. Prevalence of reported hypertension (percent) in 1986 according to age groups and frequency of snoring in the Nurses' Health Study.

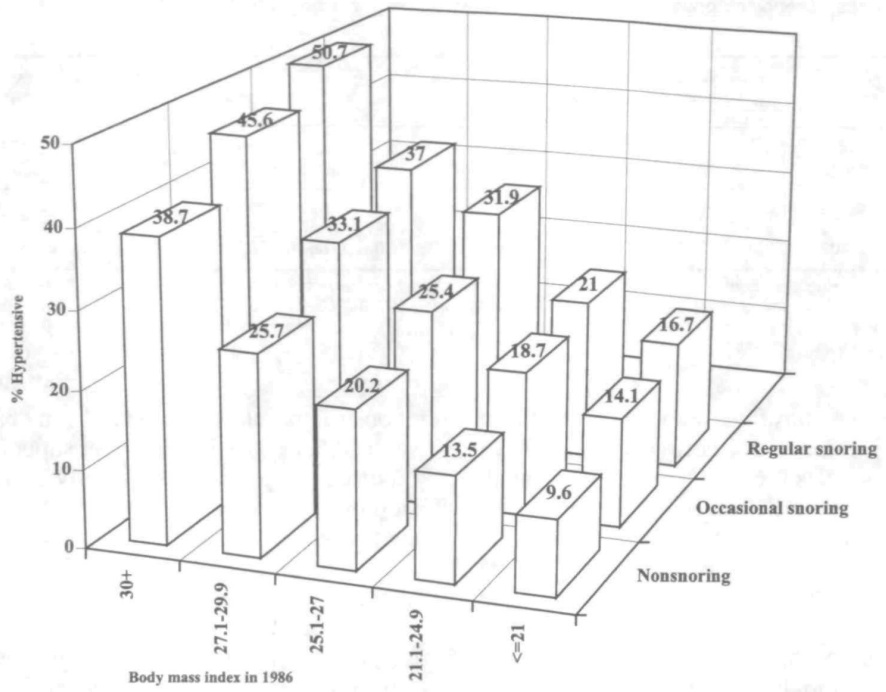

FIGURE 3. Prevalence of reported hypertension (percent) in 1986 according to body mass index and frequency of snoring in the Nurses' Health Study.

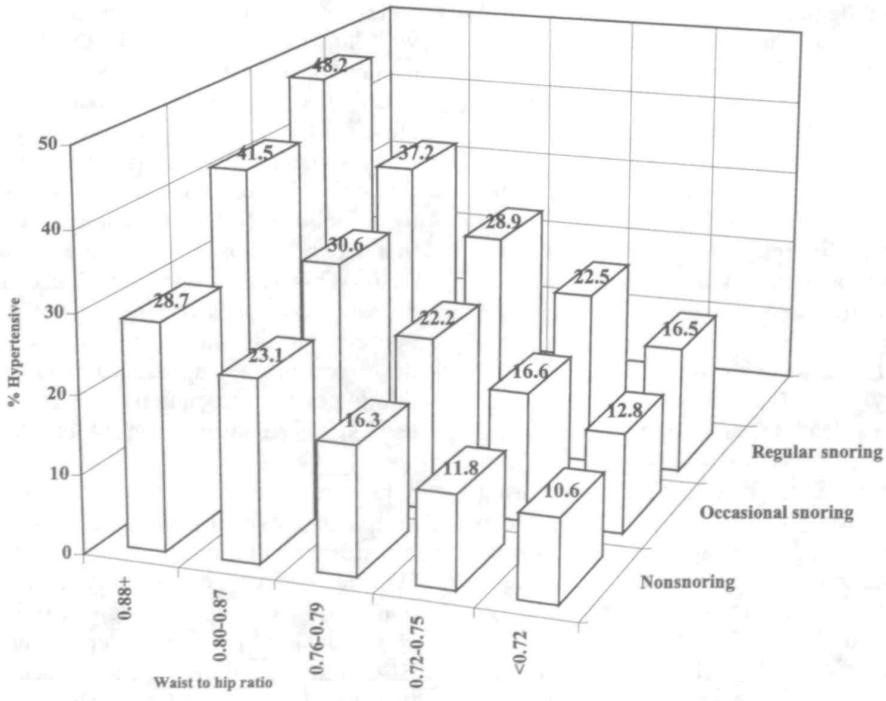

FIGURE 4. Prevalence of reported hypertension (percent) in 1986 according to waist-hip ration and frequency of snoring in the Nurses' Health Study.

TABLE 3. Relative risks of reported physician-diagnosed hypertension according to self-reported snoring, Nurses' Health Study, 1986

Amount of snoring	Cross-sectional analysis (prevalence of hypertension in 1986)						Prospective analysis (incidence of hypertension between 1986 and 1994)					
	No. of cases	Total persons	Adjusted for age, age²		Multivariate*		No. of cases	Total person-years	Adjusted for age, age²		Multivariate*	
			RR†	95% CI†	RR	95% CI			RR	95% CI	RR	95% CI
Nonsnoring	3,116	18,928	1 0		1.0		1,595	105,700	1.0		1.0	
Occasional snoring	11,993	47,419	1.47	1.41, 1.54	1.22	1.16, 1.27	5,147	225,981	1.44	1.36, 1.52	1.29	1.22, 1.37
Regular snoring	2,402	6,884	2.29	2.14, 2.44	1.43	1.33, 1.53	880	27,247	2.03	1.83, 2.21	1.55	1.42, 1.70

* Models include the following: age, age², body mass index in 1986 (deciles), waist circumference (<28, 28–29.9, 30–31.9, 32–33.9, 34–35.9, 36–37.9, and ≥38 inches (1 inch = 2.54 cm)), cigarette smoking (never, past, and current smoking of 1–14, 15–24, and ≥25 cigarettes/day), alcohol consumption (four categories), and physical activity (in quartiles of metabolic equivalents).
† RR, rate ratio; CI, confidence interval.

higher prevalence. A positive association between weight gain and incidence of snoring had also been found in previous studies (26, 27). On the other hand, weight loss may decrease the frequency of apnea (28). Further, physically active women were less likely to snore, even after adjustment for BMI. Sleeping on the back was associated with regular snoring. Women who drank 15 g or more of alcohol per day were slightly more likely to snore than were nondrinkers after accounting for age, smoking, BMI, and other covariates. We did not assess drinking patterns in this study; it is possible that drinking prior to sleep is more strongly related to snoring because the relaxant effect of alcohol on dilator muscles of the upper airway is relatively short term (29).

A number of epidemiologic studies have found a significant positive association between snoring and hypertension (4, 12–15). However, in other studies, the significant association disappeared after controlling for BMI (16–19). Most previous studies were small and were not prospective. We observed a monotonic increase in the prevalence of hypertension across categories of snoring in each stratum of BMI (figure 2) and waist circumference (figure 3). In further analyses, we observed a positive association between snoring and incidence of hypertension among both obese and nonobese women. These results strongly suggest that the association of snoring with hypertension is independent of obesity.

The mechanisms leading to hypertension among snorers are not completely known. Sleep apnea has been postulated to mediate this effect because snoring is a cardinal symptom of this condition (8). Sleep apnea causes hypoxia and hypercapnia, which can lead to chronic stimulation of the sympathetic nervous system, with elevated catecholamine levels contributing to the development of diurnal hypertension (7). In support of this hypothesis, Clark et al. (30) found significantly higher urinary and plasma catecholamine levels in subjects with sleep apnea compared with controls. A reduction in blood pressure after successful treatment of obstructive sleep apnea has been observed in several studies (5, 6), providing further support for the hypothesis that sleep apnea is causally related to hypertension. In addition, sleep apnea has been directly associated with myocardial infarction (31).

Results from epidemiologic studies on the relation between sleep apnea and hypertension have been inconsistent, however (32). Several studies did not find a significant association after adjustment for BMI (18, 19). However, the Wisconsin Sleep Cohort has reported a strong association between the apnea-hypopnea index and significantly elevated risk of hypertension after accounting for age, sex, and BMI (33). The odds ratios of hypertension ranged from 2.0 for five apneic or hypopneic episodes per hour of sleep to 5.0 for 25 apneic or hypopneic episodes. In a subsequent analysis with larger sample size (34), the odds ratio for hypertension associated with an apnea-hypopnea index of 15 (vs. 0) was 1.8. Direct comparison of this study and ours is difficult because we did not assess sleep apnea or hypopnea. However, the magnitude of the association between sleep apnea and hypertension observed in the Wisconsin Sleep Study appeared to be larger than that between snoring and hypertension observed in our study. This is not unexpected because most snorers probably do not have sleep apnea. If the association between snoring and hypertension were entirely due to the effects of sleep apnea, we would expect substantial dilution of the association between snoring and hypertension since snoring is an imperfect measure of sleep apnea.

On the other hand, simple snoring without frank sleep apnea may also increase the risk of hypertension or cardiovascular disease. Among 580 adults from the Wisconsin Sleep Cohort Study, Young et al. (35) found that simple snoring was significantly associated with elevated blood pressure independent of age, sex, and BMI; the magnitude of the association was smaller than that for more severe, sleep-disordered breathing. The authors suggest that simple snoring represents the beginning of the sleep-disordered breathing contin-

uum, which ranges from partial airway collapse and mildly increased upper-airway resistance to complete airway collapse and severe obstructive sleep apnea lasting 60 seconds or more (36). Our study, like most previous epidemiologic studies, cannot distinguish the effects of simple snoring from snoring with sleep apnea on the risk of hypertension. However, we observed a significant increase in risk of hypertension, albeit small, among occasional snorers who were most likely to be simple snorers. We speculate that snoring without sleep apnea may increase risk of hypertension and cardiovascular disease through recurrent sympathetic nervous system activation by repetitive arousal. In epidemiologic studies, snoring has been associated with increased risk of ischemic heart disease (37) and stroke (38); the development of hypertension may be an intermediate step.

Physician-diagnosed hypertension and blood pressure levels were self-reported by the nurses in our cohort. The validity of self-reported hypertension and blood pressure values is supported by the results of a substudy (20) and by confirmation of established associations of age, alcohol intake, relative weight, weight change, and nutritional factors with incidence of hypertension and blood pressure levels (39–41). In addition, history of hypertension is a strong predictor of myocardial infarction and stroke in this cohort (21). While a direct measurement of blood pressure is more objective than self-reported values, the validity of single measurements is limited because of intraindividual variability in blood pressure (42). Hypertension is usually asymptomatic, and its detection depends on behavioral factors that may be associated with snoring. Because of their profession and demonstrated strong motivation, we believe their reports to be quite accurate. In addition, because 97 percent of the women reported having had their blood pressure checked between 1986 and 1988, it is unlikely that detection bias can explain the positive association between snoring and incidence of hypertension.

Information on snoring was also self-reported. The reliability of snoring data was not validated in this population, but similar questions about frequency of snoring have been validated in other populations (43), in which self-reported habitual snoring appeared to be reliable according to all-night sleep recordings with monitoring of respiration, body movements, oxygen saturation, and snoring sound. Since all our participants were married nurses at entry into the cohort and have demonstrated interest in medical research, the possibility of underreporting of snoring is likely to be small compared with that in general population samples. Nevertheless, some misclassification of the snoring variable was inevitable. Perhaps some women were unaware of their snoring. However, the prevalence of snoring in our cohort did not appear to vary with marital status or living arrangement. Because the associations with snoring were assessed prospectively, any misclassification of snoring frequency should be nondifferential with respect to hypertension, leading to underestimation of the effects of snoring.

In summary, our data suggest that snoring is associated with significantly increased risk of hypertension in women, independent of age, BMI, waist circumference, and other lifestyle variables. Because obesity is strongly associated with both snoring and hypertension, weight loss can be beneficial for the treatment of both conditions. Other behavioral approaches, including stopping smoking and increasing physical activity, which have clear beneficial effects on cardiovascular disease, may also be useful for the prevention and treatment of snoring.

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REFERENCES

1. Tilkian AG, Guilleminault C, Schroeder JS, et al. *Hemodynamics in sleep-induced apnea. Studies during wakefulness and sleep.* Ann Intern Med 1976;85:714–19.
2. Millman RP, Redline S, Carlisle CC, et al. Daytime hypertension in obstructive sleep apnea. Prevalence and contributing risk factors. Chest 1991;99:861–6.
3. Fletcher EC, Debehnke RD, Lovoi S, et al. Undiagnosed sleep apnea in patients with essential hypertension. Ann Intern Med 1985;103:190–5.
4. Norton PG, Dunn EV. Snoring as a risk factor for disease: an epidemiological survey. Br Med J (Clin Res Ed) 1985;291: 630–2.
5. Motta J, Guilleminault C, Schroeder JS, et al. Tracheostomy and hemodynamic changes in sleep-induced apnea. Ann Intern Med 1978;89:454–8.
6. Guilleminault C, Simmons FB, Motta J, et al. Obstructive sleep apnea syndrome and tracheostomy. Long-term follow-up experience. Arch Intern Med 1981;141:985–8.
7. Parish JM, Shepard JW. Cardiovascular effects of sleep disorders. Chest 1990;97:1220–6.
8. Shepard JWJ. Hypertension, cardiac arrhythmias, myocardial infarction, and stroke in relation to obstructive sleep apnea. Clin Chest Med 1992;13:437–58.
9. Waller PC, Bhopal RS. *Is snoring a cause of vascular disease? An epidemiological review.* Lancet 1989;1:143–6.
10. Hoffstein V. Is snoring dangerous to your health. Sleep 1996; 19:506–16.

11. Quan SF, Howard BV, Iber C, et al. The Sleep Heart Health Study: design, rational, and methods. Sleep 1997;20:1077–85.
12. Gislason T, Aberg H, Taube A. Snoring and systemic hypertension—an epidemiological study. Acta Med Scand 1987; 222:415–21.
13. Koskenvuo M, Kaprio J, Partinen M, et al. Snoring as a risk factor for hypertension and angina pectoris. Lancet 1985; 1:893–5.
14. Lugaresi E, Cirignotta F, Coccagna G, et al. Some epidemiological data on snoring and cardiocirculatory disturbances. Sleep 1980;3:221–4.
15. Mondini S, Zucconi M, Cirignotta F, et al. Snoring as a risk factor for cardiac and circulatory problems: an epidemiological study. In: Guilleminault C, Lugaresi E, eds. Sleep wake disorders: natural history, epidemiology and long-term evolution. New York, NY: Raven Press, 1983:99–105.
16. Schmidt-Nowara WW, Coultas DB, Wiggins C, et al. Snoring in a Hispanic-American population. Arch Intern Med 1990; 150:597–601.
17. Jennum P, Sjol A. Snoring, sleep apnoea and cardiovascular risk factors: the Monica II Study. Int J Epidemiol 1993; 22:439–44.
18. Olson LG, King MT, Hensley MJ, et al. A community study of snoring and sleep-disordered breathing: health outcomes. Am J Respir Crit Care Med 1995;152:717–20.
19. Stradling JR, Crosby JH. Relation between systemic hypertension and sleep hypoxaemia or snoring: analysis in 748 men drawn from general practice. BMJ 1990;300:75–8.
20. Colditz GA, Martin P, Stampfer MJ, et al. Validation of questionnaire information on risk factors and disease outcomes in a prospective cohort study of women. Am J Epidemiol 1986; 123:894–900.
21. Fiebach NH, Hebert PR, Stampfer MJ, et al. A prospective study of high blood pressure and cardiovascular disease in women. Am J Epidemiol 1989;130:646–54.
22. Rimm EB, Stampfer MJ, Colditz GA, et al. Validity of self-reported waist and hip circumferences in men and women. Epidemiology 1990;1:466–73.
23. Wolf A, Hunter D, Colditz GA, et al. Reproducibility and validity of a self-administered physical activity questionnaire. Int J Epidemiol 1994;23:991–9.
24. D'Agostino RB, Lee M-L, Belanger AJ, et al. Relation of pooled logistic regression to time dependent Cox regression analysis: the Framingham Heart Study. Stat Med 1990;9: 1501–15.
25. Bloom JW, Kaltenborn WT, Quan SF. Risk factors in a general population for snoring. Chest 1988;93:678–83.
26. Hongsberg AE, Dodge RR, Cline MG, et al. Incidence and remission of habitual snoring over a 5- to 6-year period. Chest 1995;108:604–9.
27. Lindberg E, Taube A, Janson C, et al. A 10-year follow-up of snoring in men. Chest 1998;114:1048–55.
28. Smith PL, Gold AR, Meyers DA, et al. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. Ann Intern Med 1985;103:850–5.
29. Krol RC, Knuth SL, Bartlett DJ. Selective reduction of genioglossal muscle activity by alcohol in normal human subjects. Am Rev Respir Dis 1984;129:247–50.
30. Clark RW, Boudoulas H, Schaal SF, et al. Adrenergic hyperactivity and cardiac abnormality in primary disorders of sleep. Neurology 1980;30:113–19.
31. Hung J, Whitford EG, Parsons RW, et al. Association of sleep apnoea with myocardial infarction in men. Lancet 1990;336: 261–4.
32. Stradling J, Davis RJO. Sleep apnea and hypertension—what a mess. Sleep 1997;20:789–93.
33. Hla KM, Young TB, Bidwell T, et al. Sleep apnea and hypertension. Ann Intern Med 1994;120:382–8.
34. Young T, Peppard P, Palta M, et al. Population-based study of sleep-disordered breathing as a risk factor for hypertension. Arch Intern Med 1997;157:1746–52.
35. Young T, Finn L, Hla M, et al. Snoring as part of a dose-response relationship between sleep-disordered breathing and blood pressure. Sleep 1996;19:S202–5.
36. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993;328:1230–5.
37. Koskenvuo M, Kaprio J, Telakivi T, et al. Snoring as a risk factor for ischemic heart disease and stroke in men. BMJ 1987; 294:16–19.
38. Palomaki H. Snoring and the risk of ischemic stroke. Stroke 1991;22:1021–5.
39. Wittman JC, Willett WC, Stampfer MJ, et al. A prospective study of nutritional factors and hypertension among US women. Circulation 1989;80:1320–7.
40. Ascherio A, Hennekens C, Willett WC, et al. Prospective study of nutritional factors, blood pressure, and hypertension among US women. Hypertension 1996;27:1065–72.
41. Huang Z, Willett WC, Manson JE, et al. Body weight, weight change, and risk for hypertension in women. Ann Intern Med 1998;128:81–8.
42. Rosner B, Polk BF. Predictive values of routine blood pressure measurements in screening for hypertension. Am J Epidemiol 1983;117:429–42.
43. Telakivi T, Partinen M, Koskenvuo M, et al. Periodic breathing and hypoxia in snorers and controls: validation of snoring history and association with blood pressure and obesity. Acta Neurol Scand 1987;76:69–75.