

Chronic Nasal Congestion at Night Is a Risk Factor for Snoring in a Population-Based Cohort Study

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Background: Nasal congestion at night is thought to have a role in snoring and sleep apnea, but this hypothesis has not previously been tested in a population-based study.

Methods: Baseline and 5-year follow-up data on self-reported nocturnal nasal congestion and snoring frequency were collected from a population-based sample of 4916 men and women (age range, 30-60 years at baseline) enrolled in the ongoing Wisconsin Sleep Cohort Study. In-laboratory polysomnography was performed on a subset (n=1032) of the study population to determine the frequency of apnea and hypopnea episodes during sleep. Logistic regression was used to estimate odds ratios for snoring with chronic nasal congestion at night.

Results: Nocturnal nasal congestion frequency was independently associated with snoring frequency in cross-sectional analyses. The odds ratios (adjusted for sex, age, body habitus, and smoking) for habitual snoring with

severe (always or almost always) nasal congestion vs none was 3.0 (95% confidence interval, 2.2-4.0). This association was not explained by habitual snorers with frank sleep apnea (ie, ≥ 5 apnea and hypopnea episodes per hour of sleep). Prospective analyses showed that persons with chronic severe nasal congestion had a high risk of habitual snoring according to the data from the 5-year follow-up survey: the odds ratio for habitual snoring and reporting congestion always or almost always at both baseline and follow-up was 4.9 (95% confidence interval, 2.8-8.8).

Conclusions: Nocturnal nasal congestion is a strong independent risk factor for habitual snoring, including snoring without frank sleep apnea. Intervention studies are needed to determine if snoring can be reduced with treatment of nasal congestion.

Arch Intern Med. 2001;161:1514-1519

SEVERAL population-based studies have linked self-reported snoring to adverse health outcomes, including hypertension and ischemic heart disease,¹ daytime sleepiness,²⁻⁴ and accidents.⁵ Most recently, the role of snoring in the development of cardiovascular conditions was investigated prospectively in the Nurses Health Study, a study of 72 231 nurses who have been followed up repeatedly since 1976.⁶ The significant associations of snoring with incident hypertension and cardiovascular disease found in this study add strong support to a causal role of snoring in adverse health outcomes.

Snoring is the audible result of upper airway dynamics that underlie sleep-disordered breathing (SDB), including increased sleep-induced compliance and narrowing of the upper airway, turbulent airflow, and fluctuating pressure differentials between the atmosphere and the intratho-

racic space.⁷ The severity of SDB can range from inspiratory breaths with increased resistance requiring greater respiratory effort to the frequently repeated episodes of partial or complete airway closures characteristic of sleep apnea syndrome.⁸ Thus, a person reporting snoring may lie anywhere on the SDB severity continuum. However, most self-reported snorers have few detectable breathing pauses and are at the milder end of the SDB spectrum. This level of SDB is often referred to as *simple snoring*.⁸

The public health importance of snoring as a potential source of morbidity is enormous because its prevalence is so high. Prevalence estimates for habitual snoring vary according to population characteristics, such as age and body habitus, but several studies of fairly similar populations in the United States, Australia, Great Britain, and Poland have indicated that roughly 40% and 20% of middle-aged men and women, respectively, report habitual snor-

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PARTICIPANTS AND METHODS

The study protocol and informed consent procedure were approved by the Committee for the Protection of Human Subjects, University of Wisconsin School of Medicine.

SAMPLE

A population-based random sample of men and women aged 30 to 60 years (n=6569) was drawn from a sampling frame of employee payroll records of 4 state of Wisconsin agencies. This sample served as the source of participants for 2 mailed questionnaires, conducted 5 years apart, and for a subsample that was studied by in-laboratory polysomnography (n=804). Seventy-five percent of the target sample completed the first questionnaire (n=4916), and of these, 3446 completed the second questionnaire as well. The participants did not differ from the target sample on characteristics available from the payroll records, including sex, age, salary, and job classification.

From the sample of baseline survey participants, a weighted probability sample was selected for extensive overnight studies to be conducted every 4 years. Recruitment is ongoing. To date, 1400 men and women from this sample have completed an overnight study at least once. The response rate for the overnight studies has averaged 50% for the first study, 75% for the 4-year follow-up, and 85% for the 8-year follow-up. We were able to assess potential bias by comparing participants with the recruitment sample on numerous items taken from the first questionnaire. The items included lifestyle, sleep problems, medical history, and demographics. A healthy volunteer bias was seen; the participants, on average, had attained a higher educational level and salary, had less hypertension, and reported slightly more insomnia. When necessary, care is taken in analyses to account for a potential bias due to these factors. The characteristics of the baseline sample are given in **Table 1**.

DATA COLLECTION

Snoring Status

Snoring status was derived from participants' reported snoring frequency reported on the questionnaire and from the number of apnea and hypopnea episodes per hour of sleep determined by polysomnography. The question was phrased as follows: "According to what others have told you, please estimate how often you snore." Response categories included "never," "rarely," "sometimes, a few nights per month," "irregular but at least once a week," "several nights (3-5 nights per week)," "every night or almost every night," and "do not know."

Sleep-disordered breathing was measured by 18-channel polysomnography. The polysomnography consisted of a standard montage of surface leads to record analog data on cardiopulmonary function during sleep. Sleep state was monitored by electroencephalography, electro-oculography, and submental electromyography. Airflow was detected at the nares by end-tidal carbon dioxide detection (capnograph) and at the mouth by thermistry (Thermister; Pro-Tec, Henderson, Tenn). Respiratory effort was measured by chest and abdominal excursions using calibrated inductance plethysmography (Respirace; Ambulatory Monitoring, Ardsley, NY). Arterial oxygenation was recorded by finger pulse oximetry (Ohmeda 3740; Ohmeda, Englewood, Colo).

The polysomnography records were manually scored using conventional criteria.²² Each 30-second epoch of the recordings was scored for sleep stage, presence of an apnea (absence of airflow for 10 seconds or more with either no respiratory effort or opposing chest and abdomen excursions indicating attempt to breathe against a closed airway), and presence of a hypopnea (reduction in respiratory effort with a $\geq 4\%$ dip in oxygen saturation). A polysomnographic study of acceptable quality was defined by adequate sleep and breathing signals throughout the

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ing.^{2,9-11} With mounting evidence that snoring is a source of morbidity, there is a clear need to reduce this prevalence. However, the number of people who snore and the lack of effective therapy make this a daunting task.

Currently, concerned snorers are advised to seek polysomnographic evaluation for sleep apnea. Snorers found to have frequent apnea and hypopnea events would be candidates for nasal continuous positive air pressure, an effective treatment for sleep apnea, which splints open the upper airway to prevent breathing pauses. This therapy, however, is not appropriate for the large remainder of individuals with simple snoring, for whom there is little in the way of effective treatment. Consequently, the most rational strategy to reduce the prevalence of simple snoring must be risk factor reduction.

Several population-based investigations indicate that common risk factors for sleep apnea of obesity, middle age, male sex, and smoking are also independent correlates of self-reported snoring. Identification of risk factors that could be modified by intervention may hold the best promise of reducing the prevalence of

snoring. Unfortunately, most of the hypothesized risk factors for snoring cannot be eliminated, such as sex and aging, or are difficult to modify, such as smoking and obesity.

One potential modifiable risk factor is chronic nasal congestion during sleep. A biological basis for nasal congestion as a cause of SDB lies in the importance of nasal breathing to an adequate pressure differential between the atmosphere and the intrathoracic space.^{12,13} Nasal congestion leads to breathing with increased resistance and lower flow velocity, which in turn may cause an increased pressure differential and a tendency for airway collapse.¹⁴

Most previous research has focused on nasal congestion as a predictor of SDB, rather than of snoring in general. Some, but not all, of these studies have shown that nasal congestion is related to the frequency and duration of breathing pauses in patients with sleep apnea.¹⁵⁻¹⁷ Also, breathing pauses have been provoked in persons without SDB by experiments in which nasal congestion is simulated by packing or taping the nose.^{18,19} Limited analyses secondary to other study aims from 2

night, at least 4 hours of objectively measured sleep, and at least 1 period of rapid eye movement sleep.

The total number of scored apneas and hypopneas, divided by the number of hours of sleep (apnea-hypopnea index [AHI]) was determined for each participant as the summary measure of SDB.

Nighttime Nasal Congestion

Nighttime nasal congestion was assessed by questions on its frequency and cause on the mailed questionnaire and in an interview conducted during the overnight study. One question asked how often nocturnal nasal congestion, obstruction, or discharge is experienced. Response categories included "never," "rarely," "sometimes," "often" and "always or almost always." A different set of questions was asked to determine the cause of regular nocturnal nasal stuffiness, if present. For this, participants were asked (yes or no) if they experienced nighttime nasal stuffiness regularly. If yes, the causes of the stuffiness were requested. The causes were grouped into allergy, structural, or other.

Covariates

Smoking and asthma were assessed by questionnaire and by interview. Persons who reported that they currently smoked at least 1 pack of cigarettes per week were coded as current smokers. Asthma was considered present if the participant reported physician-diagnosed asthma. Standard anthropomorphic techniques were used to measure weight, height, skin folds, and circumferences of waist, hips, and neck.

ANALYSIS

Definitions

Habitual snoring was defined as a response to the snoring frequency question of "several nights per week to every

night." Simple snoring was defined as habitual snoring without frequent apnea or hypopnea events (ie, <5 polysomnographically determined apnea and hypopnea episodes per hour of sleep, or AHI <5).

Nighttime nasal congestion was used as a categorical variable with all the response categories ("never," "rarely," "sometimes," "often," and "always or almost always"). For the longitudinal analysis, severe nasal congestion was defined as nasal congestion at night always or almost always. Chronic nasal congestion was severe congestion reported at both baseline and 5-year follow-up; intermittent congestion was severe congestion at only 1 time point.

A binary variable was used for current smoking (yes, at least 1 pack per week; no, all others) and for physician-diagnosed asthma (yes, no). Body habitus was indicated by a continuous variable for body mass index (BMI).

Statistical Procedures

Data were analyzed with SAS procedures for descriptive statistics, contingency tables, and logistic regression.²³ Multiple logistic regression was used to assess the strength of association of nasal congestion and snoring. This technique allows multiple regression of conditions with a binary outcome (eg, habitual snoring present vs habitual snoring not present). The odds ratio (OR) estimated by this technique expresses how likely it is that individuals who have the risk factor being investigated (eg, nasal congestion), relative to those who do not have the risk factor (eg, the reference category of no nasal congestion), have the outcome of interest (eg, habitual snoring). Age, sex, BMI, and smoking were added to the regression models as potential confounding factors or effect modifiers. Statistical significance for differences between proportions was assessed by χ^2 tests and that for logistic regression coefficients by the Wald χ^2 test. Two-tailed *P* values of less than .05 indicated statistical significance.

population-based studies have indicated that nasal congestion is linked to snoring. Stradling and Crosby,²⁰ in a study of 1001 men, found that habitual snorers were significantly more likely to report nasal congestion. In a previous investigation of the association of nasal resistance and SDB, we also found evidence for the congestion-snoring link.²¹ To date, a study specifically designed to test the hypothesis that nasal congestion is related to snoring has not been conducted, to our knowledge.

We used data from the Wisconsin Sleep Cohort Study, an ongoing population-based prospective study of the natural history of SDB, to investigate the association of nocturnal nasal congestion with snoring. Data from baseline (n=4916) and 5-year follow-up surveys (n=3446) on a sample of middle-aged men and women and in-laboratory overnight studies on a subsample (n=1032) of the study population provided a unique opportunity to investigate the role of nocturnal nasal congestion in the occurrence of self-reported habitual snoring. Also, objective data from polysomnography permitted us to assess the role of nasal congestion across the SDB spectrum, from simple snoring to sleep apnea.

RESULTS

ANALYSES OF CROSS-SECTIONAL DATA

Adjusted ORs for nasal congestion and habitual snoring estimated by logistic regression are given in **Table 2**. The cross-sectional analysis shows a dose-response relationship between the frequency of nocturnal nasal congestion and habitual snoring, independent of sex, age, smoking, BMI, and asthma. Persons with severe nasal congestion (occurring every night or almost every night) compared with those with no congestion had 3 times the odds of being a habitual snorer (OR, 3.00; 95% confidence interval [CI], 2.2-4.0). Concordant with previous studies of self-reported snoring, smoking, obesity, age, and male sex were also significantly related to habitual snoring. Interestingly, asthma was also associated with snoring. Persons reporting physician-diagnosed asthma, compared with those without asthma, had 40% greater odds of being a habitual snorer.

Table 3 shows the results of more detailed analyses to determine if nasal congestion is associated with

Table 1. Sample Characteristics at Baseline (N = 4916)

Characteristic	Value*
Sex	
Male	53.9
Female	46.1
Age, mean (SD), y	42.6 (7.8)
Snoring	
Never	23.3
Rarely	12.1
Sometimes	20.5
At least once a week	10.4
Several nights a week	10.7
Every or almost every night	15.4
Don't know	7.5
Nocturnal nasal congestion	
Never	30.8
Rarely	31.5
Sometimes	20.3
Often	11.7
Always or almost always	5.7
Asthma	7.9
Current smoking	21.7
Allergies that cause nasal congestion	
Yes	36.1
Do not take medication	15.0
Take medication	21.1
No	63.9
Regular nocturnal nasal congestion†	
None	80.5
Due to allergy	10.7
Due to other	8.9

*All values other than age are percentages.

†Subsample, n = 524.

simple snoring. From the total of 1032 participants on whom polysomnography was performed, those with an AHI of 5 or greater were excluded. The logistic regression model using this subsample (n=804) compares simple snorers (ie, habitual snorers with an AHI <5) with those with no SDB (nonhabitual snorers and nonsnorers and an AHI <5). The results show a strong association between nasal congestion and simple snoring (OR, 3.33; 95% CI, 1.79-6.21), with a dose-response relationship suggested by the increasing OR as the frequency of nasal congestion increases. Thus, the association between congestion and self-reported habitual snoring is not explained by those snorers with sleep apnea.

To determine if the risk of habitual snoring with nasal congestion varied by cause, we used the variable for regular nasal stuffiness at night, with 3 categories: "yes due to allergies," "yes due to other causes" (eg, deviated septum), and "no regular congestion." There was no difference in the ORs for congestion due to allergy or congestion due to other causes. Similarly, we tested an interaction of the nocturnal nasal congestion variable and a variable for the presence of allergies, but found no evidence for a difference in associations by allergy status.

ANALYSES OF LONGITUDINAL DATA

The data on nasal congestion and snoring at baseline and follow-up were first examined to determine the degree to which responses changed over the 5-year interval. The

Table 2. Logistic Regression Model of Habitual Snoring (N = 4557)*

Variable	Odds Ratio	95% Confidence Interval
Sex, male	2.44	2.13-2.86
Age, per year	1.05	1.04-1.06
Body mass index, per 1 kg/m ²	1.12	1.11-1.14
Nighttime nasal congestion		
Never	1.00	Reference category
Rarely	1.10	0.91-1.33
Sometimes	1.44	1.17-1.77
Often	1.61	1.26-2.05
Always or almost always	3.00	2.18-4.01
Asthma	1.40	1.09-1.81
Current smoking	1.89	1.57-2.20

*Habitual snoring is defined as a self-reported snoring frequency of 3 to 7 nights per week.

Table 3. Logistic Regression Model of Simple Snoring (N = 804)*

Variable	Odds Ratio	95% Confidence Interval
Sex, male	1.92	1.41-2.63
Age, per 1 year	1.01	0.99-1.03
Body mass index, per 1 kg/m ²	1.09	1.06-1.12
Nighttime nasal congestion		
Never	1.00	Reference category
Rarely	1.14	0.77-1.69
Sometimes	1.52	0.98-2.36
Often	2.39	1.43-4.00
Always or almost always	3.33	1.79-6.21
Asthma	1.53	0.89-2.64
Current smoking	2.58	1.76-3.79

*Simple snoring is defined as self-reported snoring 3 to 7 nights per week and fewer than 5 apnea and hypopnea events per hour of sleep.

report of habitual snoring appeared to be quite stable over time. Of those reporting habitual snoring at baseline, fewer than 2% reported snoring never or rarely at follow-up, while 80% reported habitual snoring at follow-up. However, there was more change over this period in the frequency categories for nasal congestion.

Only 39% of those reporting congestion "always or almost always" at baseline remained in this category at follow-up; 50% reported less frequent congestion (sometimes, often). Overall, the longitudinal data indicate that while self-reported snoring status is quite stable, nasal congestion frequency among those reporting any congestion at baseline changed appreciably.

The changes in congestion frequency over time are not unexpected, as they are likely to reflect seasonal exposure to allergens or changes in other exposures. However, the changes may also reflect a lack of validity of the congestion question as an indicator of a chronic condition. To address this, we created a variable for chronic nasal congestion. We thought that individuals who reported severe congestion (always/almost always) at both time points were most likely to have a chronic congestion condition, while those with no congestion at both time points were a more specific comparison group of

Table 4. Logistic Regression Model of Chronic Severe Nocturnal Nasal Congestion and Habitual Snoring at Baseline and 5-Year Follow-up*

Nighttime Nasal Congestion	Habitual Snoring			
	Baseline		5-Year Follow-up	
	Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval
None (no severe congestion at baseline and 5-year follow-up)	1.00	Reference category	1.00	Reference category
Intermittent congestion (severe congestion reported at either baseline or follow-up, but not both)	1.6	1.26-2.08	1.6	1.27-2.10
Chronic congestion (severe congestion at baseline and 5-year follow-up)	3.6	2.06-6.25	4.9	2.78-8.78

*Severe congestion is defined as a self-reported frequency of always or almost always having nocturnal congestion, and habitual snoring is defined as a self-reported frequency of 3 to 7 nights per week.

persons who were free of nocturnal congestion. **Table 4** shows the results of the logistic regression model estimating the association of chronic severe vs no severe congestion and intermittent severe congestion vs no severe congestion with habitual snoring based on the longitudinal data. Controlling for age, sex, smoking, BMI, and asthma, the ORs suggest that individuals with chronic severe nocturnal nasal congestion have a 3.6-fold greater odds of being a habitual snorer at baseline and that the odds increase over time, with a 4.9-fold greater odds of being a habitual snorer at follow-up.

COMMENT

The results of this investigation identify nocturnal nasal congestion as a risk factor for simple snoring, as well as for habitual snoring (which includes snorers with sleep apnea). Nocturnal nasal congestion occurring always or almost always was associated with a 3-fold increase in the likelihood of habitual snoring for persons with or without sleep apnea, independent of sex, age, BMI, smoking, and asthma. The ORs for habitual and simple snoring increased with increasing category of nasal congestion frequency. We did not find that congestion due to allergies, compared with other causes, was a stronger predictor of snoring. Most importantly, chronic severe nocturnal nasal congestion (ie, nocturnal nasal congestion occurring always or almost always at both baseline and 5-year follow-up) was most strongly related to snoring, and the strength of the association increased from an OR of 3.60 (95% CI, 2.1-6.3) (to an OR ratio of 4.9 (95% CI, 2.8-8.8) over time.

We found no evidence that this association was driven by a strong relationship between nasal congestion and more severe SDB, eg, snorers with 5 or more apnea and hypopnea episodes per hour of sleep. The equivalent OR for snorers with and without frank SDB is compatible with a natural history scheme whereby simple snoring is an early manifestation of SDB: exposure to factors that cause SDB leads first to episodic upper airway resistance overtly manifested as snoring. With worsening airway dynamics over time, simple snoring may, in a significant proportion of people, progress to more severe SDB.²⁴

If snoring is an early marker for progressive SDB, risk factor intervention that reduces or prevents the onset and progression of simple snoring could prevent the develop-

ment of clinically significant SDB. However, in considering the natural history path of SDB from early stage to severe sleep apnea, it is important to note that not all significant episodic upper airway resistance results in snoring. "Silent" breathing events defined by increasing airway resistance and terminating in brief arousals from sleep, termed *respiratory effort related arousals*, may lead to upper airway resistance syndrome (UARS), characterized by the frequent occurrence of these events, in combination with resultant daytime sleepiness.^{3,25} As part of the SDB spectrum, it is likely that respiratory effort-related arousals and UARS are associated with the same risk factors as is snoring. Unfortunately, very little is known about the epidemiology of UARS, and without having the necessary measure of esophageal pressure to identify UARS events in our participants, we are not able to address this hypothesis.

To our knowledge, this is the only study in which risk factors for simple snoring have been identified. Similarly, the role of chronic nasal nocturnal congestion over several years has not been examined as a risk factor for snoring. Thus, we are not able to compare these associations with existing reports. However findings from the few studies of other risk factors, eg, BMI, smoking, age, sex, and general snoring, are consistent with our results, providing some confidence in the validity of our methodology.^{2,6,10,11,26}

It is possible that our findings are attributable to study limitations, including sample bias or incomplete control for confounding factors. However, it is difficult to explain how limitations in our study could account for the strong, persistent associations of congestion and snoring that we found. Sample bias would require the overrepresentation of habitual snorers with nasal congestion. However, the longitudinal analyses yielded even stronger findings than those from cross-sectional analyses. Participation bias can cause spurious findings in cross-sectional analyses, but this is less of a concern in longitudinal analyses. For this type of bias to occur, participation would have to be differentially greater in habitual snorers with nasal congestion who were also destined to have nasal congestion 4 years into the future. Finding an even stronger relationship with the longitudinal data further reduces this concern with participation bias.

We were able to account for several potential confounding factors, but it is possible that an unknown fac-

tor related to both snoring and nasal congestion is responsible for part or all of the associations reported herein. However, such a factor would have to be extremely influential to account for the relatively high ORs found, particularly in the longitudinal analyses.

Misclassification of nocturnal nasal congestion and of snoring is possible, as both of these measures relied on self-report. Although there have been a few very small validation studies that show self-reported snoring to be of variable accuracy,²⁷ there have been no studies on reported nasal congestion, to our knowledge. Our classification of the cause of congestion is subject to variation in participants' knowledge as well as to the severity of the congestion. We had data on allergy medication use, but the interpretation of findings using this variable is limited: medication may reduce congestion, but it is also likely to be used by the persons with the most severe congestion. Misclassification of nasal congestion or snoring, if random, would result in reducing the magnitude of true ORs. However, if misclassification was differential to one of the exposure or outcome variables with respect to the other, then bias could result. Such a situation would arise if there were a tendency of individuals who snore to exaggerate any other nocturnal condition, including nocturnal nasal congestion. Unfortunately, we were not able to address this possible bias.

The particular strengths of this study are the large, population-based sample, longitudinal data on congestion and snoring over a 5-year interval, and the ability to identify simple snorers using data from in-laboratory polysomnography. Prevalences of common conditions such as smoking, seasonal allergic rhinitis, hypertension, and asthma indicate that our sample is representative of the general middle-aged population. However, our sample lacks racial heterogeneity, and thus the findings may not apply to races other than white.

Recent population-based studies indicate that simple snoring, in addition to snoring accompanied by episodes of apnea and hypopnea, is linked to significant morbidity. If associations with morbidity are causal, the highly prevalent condition of habitual snoring could account for a significant proportion of adverse health outcomes, including pathologic sleepiness, accidents, and cardiovascular disease. Furthermore, habitual snoring can impair the sleep of bed partners and cause interpersonal stress. We found that regular nocturnal nasal congestion, particularly that which is chronic over several years, is a strong risk factor for snoring with and without frank SDB. This relationship may be particularly important because nasal congestion can be controlled pharmacologically. If this association is causal, the substantial magnitude of the OR, in conjunction with the high prevalence of nasal congestion, suggests that intervention may reduce the prevalence of snoring significantly. At present, there are no firm data from a randomized controlled clinical trial testing the efficacy of pharmacological intervention to reduce snoring. The findings of the present investigation underscore the importance of continuing to investigate this modifiable risk factor for snoring.

Accepted for publication November 8, 2000.

This study was supported in part by grants R01HL62252 and RR03186 from the National Institutes of

Health, Bethesda, Md, and a grant from Integrated Therapeutics Group Inc, Kenilworth, NJ.

We are grateful for the technical expertise and contributions of Linda Evans, Katherine Pluff, Katherine Kennison, Leah Steinberg, Anthony Jacques, Steven Weber, PhD, James Skatrud, MD, Jerome Dempsey, PhD, and Safwan Badr, MD.

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REFERENCES

1. Young T, Peppard P. Epidemiological evidence for an association of sleep-disordered breathing with hypertension and cardiovascular disease. In: Bradley TD, Floras JS, eds. *Sleep Apnea: Implications in Cardiovascular and Cerebrovascular Disease*. New York, NY: Marcel Dekker Inc; 2000:261-283.
2. Zielinski J, Zgierska A, Polakowska M, et al. Snoring and excessive daytime somnolence among Polish middle-aged adults. *Eur Respir J*. 1999;14:946-950.
3. Guilleminault C, Stoohs R, Clerk A, Simmons J, Labanowski M. From obstructive sleep apnea syndrome to upper airway resistance syndrome: consistency of daytime sleepiness. *Sleep*. 1992;15(suppl):S13-S16.
4. Gottlieb DJ, Whitney CW, Bonekat WH, et al. Relation of sleepiness to respiratory disturbance index: the Sleep Heart Health Study. *Am J Respir Crit Care Med*. 1999;159:502-507.
5. Young T, Blustein J, Finn L, Palta M. Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults. *Sleep*. 1997;20:608-613.
6. Hu FB, Willet W, Colditz GA, et al. Prospective study of snoring and risk of hypertension in women. *Am J Epidemiol*. 1999;150:806-816.
7. Hoffstein V. Snoring. *Chest*. 1996;109:201-222.
8. Lugaresi E, Plazzi G. Heavy snorers disease: from snoring to the sleep apnea syndrome: an overview. *Respiration*. 1997;64(suppl 1):11-14.
9. Young T, Palta M, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. 1993;328:1230-1235.
10. Ohayon M, Guilleminault C, Priest RG, Caulet M. Snoring and breathing pauses during sleep: telephone interview survey of a United Kingdom population sample. *BMJ*. 1997;314:860-863.
11. Bearpark H, Elliot L, Grunstein R, Schneider H, Althaus W, Sullivan C. Snoring and sleep apnea: a population study in Australian men. *Am J Respir Crit Care Med*. 1995;151:1459-1465.
12. Olsen KD, Kern EB. Nasal influences on snoring and obstructive sleep apnea. *Mayo Clin Proc*. 1990;65:1095-1105.
13. Proctor DF. The upper airways: nasal physiology and defense of the lungs. *Am Rev Respir Dis*. 1977;115:97-129.
14. Shepard JW, Burger CD. Nasal and oral flow-volume loops in normal subjects and patients with obstructive sleep apnea. *Am Rev Respir Dis*. 1990;142:1288-1293.
15. Metes A, Ohki M, Cole P, et al. Snoring, apnea and nasal resistance in men and women. *J Otolaryngol*. 1991;20:57-61.
16. McNicolas WT, Tarlo S, Cole P, et al. Obstructive apneas during sleep in patients with seasonal allergic rhinitis. *Am Rev Respir Dis*. 1982;126:625-628.
17. Miljeteig H, Hoffstein V, Cole P, et al. Snoring and nasal resistance during sleep. *Laryngoscope*. 1993;103:918-23.
18. Millman RP, Acebo C, Rosenberg C, Carskadon MA. Sleep breathing and cephalometrics in older children and young adults. II: response to nasal occlusion. *Chest*. 1996;109:109:673-679.
19. Zwilllich C, Pickett C, Hanson F, Weil J. Disturbed sleep and prolonged apnea during nasal obstruction in normal men. *Am Rev Respir Dis*. 1981;124:158-160.
20. Stradling JR, Crosby JH. Predictors and prevalence of obstructive sleep apnea in 1001 middle-aged men. *Thorax*. 1991;46:85-90.
21. Young T, Finn L, Kim H. Nasal obstruction as a risk factor for sleep-disordered breathing. *J Allergy Clin Immunol*. 1997;99(suppl):S757-S762.
22. Rechtschaffen A, Kales AA, eds. *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*. Washington, DC: US Government Printing Office; 1968. NIH publication 204.
23. SAS Institute Inc. *SAS/STAT Software: Changes and Enhancements Through Release 6.11*. Cary, NC: SAS Institute Inc; 1996.
24. Lugaresi E, Cirignotta F, Gerardi R, Montagna P. Snoring and sleep apnea: natural history of snorers disease. In: Guilleminault C, Partinen M, eds. *Obstructive Sleep Apnea Syndrome: Clinical Research and Treatment*. New York, NY: Raven Press; 1990:25-36.
25. American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definitions and measurement techniques in clinical research: the report of an American Academy of Sleep Medicine Task Force. *Sleep*. 1999;22:667-689.
26. Lindberg E, Taube A, Janson C, et al. A 10-year follow-up of snoring in men. *Chest*. 1998;114:1048-1055.
27. Hoffstein V. Is snoring dangerous to your health? *Sleep*. 1996;19:506-516.