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Among middle-aged adults, snoring predicted hypertension independently of sleep apnoea

Habibolah Khazaie[1], Saeedeh Negahban[1], Mohammad R Ghadami[1], Dena Sadeghi Bahmani[2], Edith Holsboer-Trachsler[2] and Serge Brand[1,2,3]

Abstract

Objective: While the link between obstructive sleep apnoea (OSA) and hypertension is well established, the relationships between snoring, OSA, and hypertension remain unclear. This study aimed to evaluate the association between hypertension and snoring independently of OSA.

Methods: Adults with sleep difficulties underwent a one-night polysomnographic sleep assessment, including a thorough assessment of apnoea and snoring. Upon waking, blood pressure was measured, the measurement repeated after 15 min, in a resting position. Anthropometric data were recorded. Hypertension was defined as blood pressure $\geq$140/90 mmHg or the use of antihypertensive medications.

Results: The study enrolled 181 adults (mean age 48.8 years; 119 males). Snoring, apnoea, blood pressure and anthropometric dimensions were highly associated. Patients with hypertension had higher levels of snoring and apnoea, as well as indicators of excess weight. Snoring was the most robust predictor of hypertension.

Conclusions: Snoring is a risk factor for hypertension independently of apnoea and anthropometric dimensions. While the presence of snoring is not able to replace a thorough

[1]Sleep Disorders Research Centre, Kermanshah University of Medical Sciences, Kermanshah, Iran
[2]Psychiatric Clinics, Centre for Affective, Stress and Sleep Disorders, Psychiatric University Hospitals, University of Basel, Basel, Switzerland
[3]Department of Sport, Exercise and Health, Division of Sport and Psychosocial Health, University of Basel, Basel, Switzerland

Corresponding author:
Serge Brand, Psychiatric Clinics (UPK), Centre for Affective, Stress and Sleep Disorders (ZASS), University of Basel, Wilhelm Klein-Strasse 27, 4002 Basel, Switzerland.
Email: serge.brand@upkbs.ch

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polysomnographic evaluation of the apnoea-hypopnoea index and OSA, snoring as an acoustic signal is easily detectable. The early identification and management of snoring may reduce cardiovascular risk.

Keywords
Snoring, sleep apnoea, cardiovascular disease, blood pressure

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Introduction

Sleep-related breathing disorders (SRBD) are among the most common sleep disorders, which have a major impact on health and quality of life.[1,2] SRBD is an umbrella term for a range of syndromes from upper airway resistance syndrome to obstructive sleep apnoea (OSA). SRBDs usually occur as a result of a partial or complete obstruction of the airway. The estimated prevalence of OSA, which is defined by an apnoea-hypopnoea index (AHI) of more than 5 per hour measured by polysomnography (PSG), is approximately 20–30% for males and 10–15% for females, and it is more prevalent in older people.[3,4] In people with OSA, during sleep the air stream in the upper airway is reduced, especially at the soft palate level. Reduction of air entering into the lungs leads to an abnormal reduction in blood oxygen level. Subsequently, diaphragm and other chest wall muscle reactions lead to obstruction elimination in the upper airway leading the individual with OSA to wake with loud snoring.[4] Although people with OSA present a long history of snoring as a main symptom, several studies have demonstrated that some medical problems are related to snoring independently of the AHI, [5–7] which is the main index for OSA diagnosis.[8]

With regard to the association between OSA and hypertension, several studies have been performed, though there is an inconsistency in the findings.[9–13] In addition, several studies have examined the association between snoring and hypertension and some of these have shown that the prevalence of hypertension among participants with habitual snoring was significantly higher than among those who did not snore, suggesting that snoring may be an independent risk factor for hypertension.[14–17] Hypertension is particularly critical, as the median prevalence of total hypertension in the USA in 2009 was 37.6% for men and 40.1% for women.[18] Furthermore, hypertension is associated with a broad range of both somatic[4,18] and psychiatric problems.[19–21] Surprisingly, however, it appears that there has been little investigation of the relationships between snoring, hypertension and apnoea. Accordingly, the present study was designed to evaluate these relationships. Specifically, the study explored the associations between snoring, apnoea, blood pressure and anthropometric dimensions including neck and waist circumference and body mass index (BMI).

Patients and methods

Patient population

This prospective study enrolled consecutive patients who were referred to the Sleep Disorders Research Centre of the Kermanshah University of Medical Sciences, Kermanshah, Iran due to their difficulties in sleeping and breathing

during sleep between November 2014 and August 2015. Inclusion criteria were as follows: (i) aged between 40 and 60 years; (ii) difficulties in sleeping and breathing, as reported by the patients and as taken from medical records; (iii) willing and able to participate in the study; (iv) provided written informed consent. Exclusion criteria were as follows: (i) current psychiatric disorders as assessed by a psychiatrist based on the Structured Clinical Interview for Diagnostic and Statistical Manual of Manual of Psychiatric Disorders-IV diagnoses;[22] (ii) a history of underlying diseases such as neurodegenerative or respiratory diseases; (iii) pregnancy; (iv) using sedative drugs or opioids; (v) tobacco dependency.

Patients underwent an overnight PSG sleep assessment. The following morning, blood pressure was measured in a resting condition. Blood pressure measurements were repeated twice, the measurements made 15 min apart, at rest in a supine position using a mercury sphygmomanometer. Participants also completed sociodemographic questionnaires and anthropometric measurements were taken.

All participants gave their written informed consent. All procedures performed in the present study were in accordance with the ethical standards of the institutional research committee of the Kermanshah University of Medical Sciences and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Sleep and breathing assessment

An overnight PSG (SOMNOscreen^{TM} plus; SOMNOmedics GmbH, Randersacker, Germany) was performed with all participants to determine the presence and severity of OSA as well as snoring. PSG recordings were started based on the patient's usual sleeping habits. Patients were recorded for a minimum of 7 h. The PSG recording and scoring of sleep stages and respiratory events were performed as per guidelines of the American Academy of Sleep Medicine (AASM 2012) by trained sleep physicians (H.K., S.N. and M.R.G.).[23] Following the AASM 2012 recommendations, hypopnoea was defined as a $\geq$30% reduction in airflow from baseline accompanied by a $\geq$3% decrease in oxygen desaturation index. Further, AHI, a commonly used index for OSA, was applied according to the AASM 2012 criteria. According to these criteria, hypopnoea, a partial cessation of breathing, can be defined as a decrease from baseline in the amplitude of a valid measure of breathing during sleep that either reaches $>$50% with an oxygen desaturation of 3% or an arousal, or alternatively a 30% reduction with 3% oxygen desaturation. Apnoea is defined as complete cessation of breathing. Obstructive apnoea/hypopnoea was defined as an event that lasts for $\geq$10 sec. The AHI was calculated as the mean number of apnoeas and hypopnoeas per hour of recording in the sleep period. An AHI $\geq$5 was used to diagnose OSA.[4] While there is no strict consensus about the definition and assessment of snoring, this study followed the Guidelines of the German Society of Otorhinolaryngology, Head and Neck Surgery[24] and defined snoring as follows. Snoring was detected by a nasal cannula in conjunction with a pressure sensor. This pressure sensor monitored the pressure fluctuations produced by snoring. Snoring episodes with values more than 20 microbar were recorded after excluding artefacts of $\geq$10000 microbar. The cumulative duration of snoring episodes was obtained from the data record and then these data were used to calculate the percentage of time spent snoring during sleep by dividing the cumulative duration of snoring episodes by the total sleep time. Using the PSG-derived snoring information, patients were defined as 'snorers' if they snored for more than 3% of the total

sleep time. A snore index (SI) was calculated as the mean amount of snoring per hour of recorded sleep. Primary snoring was diagnosed as a complaint of snoring with an AHI of <5. The number of physiological awakenings per hour was recorded as the arousal index.

Blood pressure

Brachial systolic and diastolic blood pressure was measured on the morning after carrying out the PSG and after 5 min in a resting condition. A mean of two measurements was calculated and used to determine systolic and diastolic blood pressure. Measurements were taken twice, with a 15-min interval between measurements, at rest in a supine position using a mercury sphygmomanometer. Patients were not allowed to exercise, drink tea or coffee, or eat 30 min before or during the measurement of blood pressure. Mean arterial pressure (MAP) was calculated using the following formula: MAP $= [(2 \times \text{diastolic}) + \text{systolic}]/3$. Hypertension was defined as blood pressure $\geq$140/90 mmHg or the use of antihypertensive medications.[13]

Anthropometric measurements

Study nurses measured the neck and waist circumference and height of all study participants. Weight was measured with a commercially available electronic balance. The BMI was calculated using the following formula: BMI = body mass/square of the height (kg/m^2).

Statistical analyses

All statistical analyses were performed using SPSS®, version 23.0 (IBM Corporation, Armonk, NY, USA) for Windows®. Three statistical approaches were adopted. First, apnoea and snoring scores were correlated with blood pressure indices and anthropometric dimensions using Pearson's correlation coefficient. Secondly, the sample was split into patients with and without hypertension, and indices of apnoea and snoring together with the anthropometric dimensions were compared using Student's *t*-test. Thirdly, a multiple regression analysis was performed with hypertension as the outcome variable and apnoea and snoring indices and anthropometric dimensions as predictors. A P-value < 0.05 was considered statistically significant.

Results

This study enrolled 181 patients with a mean ± SD age of 48.8 ± 5.4 years (119 [65.7%] males). Of the 181 patients, 90 (49.7%) had apnoea and snoring, 18 (9.9%) had apnoea without snoring, 41 (22.7%) had snoring without apnoea, and 32 (17.7%) had neither apnoea nor snoring. The overall mean ± SD arousal index was 25.76 ± 7.64 (median 25.20); and there were no significant differences between those patients with apnoea and snoring (27.32 ± 8.54), patients with apnoea without snoring (25.35 ± 7.44), patients with snoring without apnoea (24.24 ± 7.41), and patients with neither apnoea nor snoring (24.78 ± 5.47).

Table 1 presents the full descriptive statistics and correlation coefficients between age, SI, AHI, blood pressure-related measures (systolic blood pressure, diastolic blood pressure, MAP) and anthropometric dimensions (BMI, neck and waist circumference). Greater age was associated with higher SI and AHI, higher blood pressure-related measures and higher anthropometric dimensions. Higher SI and AHI were associated with higher blood pressure-related measures and higher anthropometric dimensions. Higher blood pressure-related indices were associated with higher anthropometric dimensions.

Table 1. Overview of correlations and descriptive statistics for age, snoring index, apnoea-hypopnoea index, blood pressure-related measures and anthropometric dimensions in patients ($n = 181$) with difficulties in sleeping and breathing during sleep.

	Dimensions									
	Age	Snoring and apnoea		Blood pressure			Anthropometrics			
Dimensions	1	2	3	4	5	6	7	8	9	Mean (SD)
1 Age, years	–	0.17*	0.23**	0.36**	0.27**	0.35**	0.17*	0.23**	0.34***	48.83 (5.40)
2 Snoring index		–	0.42***	0.33***	0.31***	0.36***	0.23***	0.22**	0.25***	151.23 (148.87)
3 Apnoea-hypopnoea index			–	0.35***	0.26***	0.34***	0.45***	0.34***	0.40***	15.07 (18.06)
4 Systolic blood pressure, mmHg				–	0.70***	0.92***	0.47***	0.27***	0.31***	116.73 (16.90)
5 Diastolic blood pressure, mmHg					–	0.92***	0.36***	0.14	0.25**	75.52 (8.96)
6 Mean arterial pressure, mmHg						–	0.47***	0.24**	0.30***	89.62 (8.74)
7 Neck circumference, cm							–	0.60***	0.65***	38.19 (4.07)
8 Waist circumference, cm								–	0.64***	96.50 (14.45)
9 Body mass index									–	26.92 (4.76)

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$; Pearson's correlation coefficients.

The differences in SI, age, AHI and anthropometric dimensions between patients without and with hypertension are presented in Table 2. Patients with hypertension were defined as those having a blood pressure $\geq$140/90 mmHg or those using antihypertensive medications. Compared with patients without hypertension ($n = 153$), patients with hypertension ($n = 28$) were significantly older, had higher snoring and apnoea-hypopnoea indices, higher blood pressure indices and higher anthropometric indices ($P < 0.05$ for all comparisons).

Given the significant differences in age, SI, AHI and anthropometric dimensions between patients with or without hypertension, the study then investigated which of these dimensions could accurately predict the MAP (as an index of cardiac load) using a multiple regression analysis (stepwise backward) with MAP as the dependent variable and age, SI, AHI, and anthropometric dimensions as predictors. MAP ($R = 0.544$; $R^2 = 0.296$; Durbin–Watson coefficient: 2.24; intercept: standard coefficient: 53.23; standard error: 6.59, $t = 8.11$; $P < 0.001$) was predicted by the SI (beta = 0.32, $t = 4.24$, $P < 0.01$) and neck circumference (beta = 0.36, $t = 4.85$, $P < 0.001$), while age, AHI, BMI and waist circumference had no predictive value. If the Durbin–Watson coefficient is between 1.5 and 2.5, it shows that the residuals of the independent variables are independent.[25]

Discussion

The key findings of the present study were that: (i) age, SI, AHI, blood pressure, and anthropometric measurements were

Table 2. Comparison of age, snoring index, apnoea-hypopnea-index, blood pressure-related measures and anthropometric dimensions in patients stratified according to the presence of hypertension.

Characteristics	Hypertension		Statistical significance[a]
	Yes $n = 28$	No $n = 153$	
Age, years	50.25 (3.54)	41.60 (4.26)	$P < 0.01$
Snoring index	256.80 (189.46)	132.74 (132.20)	$P < 0.001$
Apnoea-hypopnoea index	24.61 (23.54)	13.67 (16.43)	$P < 0.01$
Systolic blood pressure, mmHg	143.93 (17.29)	111.79 (11.20)	$P < 0.001$
Diastolic blood pressure, mmHg	87.68 (7.76)	73.13 (7.24)	$P < 0.001$
Mean arterial pressure, mmHg	106.67 (7.73)	86.19 (7.91)	$P < 0.001$
Neck circumference, cm	40.56 (4.15)	37.75 (3.56)	$P < 0.01$
Waist circumference, cm	102.88 (12.44)	95.62 (14.56)	$P < 0.05$
Body mass index	28.45 (4.32)	26.64 (4.79)	$P < 0.05$

Data presented as mean (SD).
[a]Student's *t*-test; $t(179)$.

associated; (ii) patients with hypertension, compared with those who were normotensive, had a higher SI, AHI and anthropometric measures (indicating excess weight); and (iii) higher MAP was predicted by the SI independently of AHI or the anthropometric measurements. Most importantly, in the regression analysis, increased snoring was the strongest robust variable to predict higher MAP. Further, at least statistically, the arousal index was not associated with snoring or apnoea.

This present study indicated that snoring is a risk factor for hypertension independently of BMI, AHI, age, waist or neck circumference. A population-based study showed persistent snoring to be an independent risk factor for the development of hypertension among males aged <50 years.[14] The authors suggested that further prospective studies including whole night sleep recordings are needed to establish whether this is due to a higher prevalence of OSA syndrome among snorers or whether snorers without apnoea and with increased upper airway resistance are also at increased risk of developing hypertension.[14] In a separate study, the same research group applied a multivariate model adjusting for age, BMI, smoking, physical activity and alcohol dependency, and found that snoring and excessive daytime sleepiness were risk factors for hypertension.[26] Snoring and excessive daytime sleepiness were more closely related to hypertension among women aged <50 years.[27]

Snoring appears to be an indicator of 'poor health behaviour' such as increased weight as reported in Tables 1 and 2. It is possible that the motivation of snorers for hypertension screening is lower than that of non-snorers. Snoring and hypertension are both prevalent in the community and many individuals suffer from both.[17] Several studies have reported an association between snoring and hypertension that is independent of other factors.[16,17,28–30] A study of 169 patients reported that snoring but not sleepiness was associated with increased aortic root diameter in hypertensive patients.[31] Another study found that snoring intensity was associated with high blood pressure after adjusting for several confounding factors (age, sex, BMI, AHI, alcohol consumption, and smoking).[17] A study

in Chinese patients found that the prevalence of hypertension increased with greater severity of snoring.[32] The severity of snoring was positively correlated with diastolic and systolic blood pressures.[32] The authors also found snoring, age, BMI and alcohol consumption all to be risk factors for hypertension.[32] A multivariate regression analysis demonstrated a significant association between snoring sound intensity and systolic/diastolic blood pressure after adjusting for AHI and other confounding factors but only in the non-to-mild OSA group.[33] Snoring sound intensity may be independently associated with daytime blood pressure in patients with either primary snoring or mild OSA.[33]

There is also clear evidence from epidemiological and pathophysiological research for a causal link between OSA and hypertension.[9–13] OSA is a recognized cause of secondary hypertension and independently associated with target organ damage in hypertensive patients. The acute haemodynamic and autonomic disturbance that results from OSA, with associated repeated arousals and intermittent hypoxaemia, appears to result in sustained hypertension.[9–13] In addition to the metabolic and humoral effects of obesity, OSA appears to predispose individuals to autonomic imbalance characterized by sympathetic over-activity and altered baroreflex mechanisms as well as alterations to vascular function.[9–13] OSA episodes produce surges in systolic and diastolic blood pressure that keep mean blood pressure levels elevated at night. In many patients, blood pressure remains elevated during the daytime, even when breathing is normal.[9–13] Contributors to this diurnal pattern of hypertension include sympathetic nervous system over-activity and alterations in vascular function and structure caused by oxidant stress and inflammation.[9–13] A study in children with sleep apnoea syndrome found that they exhibited a higher 24-h blood pressure than those with primary snoring.[34] OSA in children seems to be associated with the development of hypertension and other cardiovascular diseases.[34] Another study found that a significant proportion of hypertensive patients suffered from excessive daytime sleepiness and presented a high risk of sleep apnoea.[35] However, there are discrepancies in the findings regarding the correlation between hypertension and sleep apnoea. For example, another study found no significant association between sleep apnoea/non-sleep apnoea and hypertension prevalence.[36]

In our opinion, elevated blood pressure should be considered in the diagnosis and treatment of sleep problems, particularly apnoea and snoring. Increasing awareness of sleep apnoea and snoring among healthcare providers appears to be relevant to the primary care of patients with sleep disorders; and it has the potential benefits for the identification and treatment of sleep apnoea and snoring.[31] The clinical findings from this present study suggest that hypertensive patients should to be questioned about snoring, a symptom that is strongly associated with not only OSA but also with elevated blood pressure.

Despite these encouraging results, the following issues caution against overgeneralization of the present findings. First, assessing daytime sleepiness and cognitive performance could have provided a deeper understanding regarding the extent to which snoring and apnoea impact on daily functioning and intellectual performance. Secondly, there is no strict consensus on how to define snoring,[24] and one limitation might be that this present study did not assess snoring intensity. However, to the best of our knowledge, there is no standardized tool or instrument to thoroughly assess snoring intensity. Thirdly, blood pressure was assessed in the morning, while close monitoring of blood pressure throughout the night would have allowed a more

detailed analysis of relationships between blood pressure mechanisms, sleep, snoring, and breathing. Fourthly, the present findings might have emerged due to latent and unassessed variables, which might have biased two or more dimensions in the same or opposite direction. This issue might relate to renal function, but alcohol use, which might be an issue in Western countries, was not determined in this current study because, for cultural and religious reasons, alcohol use is highly discouraged. Fifthly, unfortunately pulse oximetry data (oxygen desaturation index; basal O_2; nocturnal nadir) were not assessed, because intermittent hypoxaemia appears to be a key mechanism for the development of high blood pressure. Sixthly, the arousal index was not statistically associated with the presence or absence of apnoea, or snoring, or the combination of them. Finally, the study did not allow for the possibility that some patients had undiagnosed upper airway resistance syndrome (UARS),[37] which requires oesophageal pressure monitoring for diagnosis. The treatment of UARS is challenging and the application of nasal continuous positive airway pressure, the mainstay of treatment, has limited effectiveness.[38] However, future studies should make every effort to diagnose UARS.

In conclusion, while sleep apnoea has an important role to play in hypertension, the present data demonstrate that snoring is also a risk factor for hypertension independent of BMI, AHI and age. One novel aspect of this present study was the use of overnight PSG to provide objective measures of both snoring and apnoea. While it would not be our intention to replace a thorough routine PSG evaluation of AHI and OSAs with a measurement of snoring, at a behavioural level at least, snoring is an acoustic signal that is easily detectable. Therefore, early identification and management of snoring may reduce cardiovascular risk. Accordingly, appropriate intervention strategies are indicated to reduce the burden of hypertension associated with sleep apnoea and snoring in the population.

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Declaration of conflicting interest

The authors declare that there are no conflicts of interest.

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