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ORIGINAL RESEARCH

Association Between Sleep Efficiency and Hypertension in Chinese Obstructive Sleep Apnea Patients

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Objective: We aimed to explore the relationship of sleep efficiency (SE) with the prevalence of hypertension in Chinese obstructive sleep apnea (OSA) patients based on polysomnography (PSG) records.

Methods: We studied 2360 patients with OSA and 764 primary snorers who underwent PSG in our hospital. SE was divided into three grades, including ≥85%, 80%~84.9%, and <80%. Hypertension was defined based either on direct blood pressure measurements, under anti-hypertensive treatments or on physician diagnosis. Multivariate logistic regression models were conducted to investigate the association between SE and hypertension.

Results: After adjusting for potential confounding factors, OSA patients with <80% SE and those with 80% to 84.9% SE were significantly associated with the prevalence of hypertension (OR = 1.248, 95% CI 1.018~1.531, *P*=0.033; OR = 1.380, 95% CI 1.040~1.832, *P*=0.026). Compared to primary snorers, OSA combined with <85% SE increased the odds of hypertension. In stratified analysis by SE, risk of hypertension only in those with <80% SE was significantly different between OSA and primary snorers. Furthermore, this relationship between reduced SE and hypertension was evident especially in female, younger ages, obese, moderate and severe OSA patients. No significant relationship between reduced SE and hypertension was found in primary snores group.

Conclusion: We found that poor SE was correlated with the prevalence of hypertension in Chinese OSA patients, but not in those with primary snoring. Moreover, this relationship was evident especially in female, younger ages, obese, moderate and severe OSA patients.

Keywords: sleep efficiency, hypertension, obstructive sleep apnea, polysomnography

Plain Language Summary

Former studies found that both OSA and reduced SE were associated with hypertension. However, the relationship between low SE and hypertension in Chinese OSA patients has not been determined. Our results first time demonstrated that poor SE was correlated with the prevalence of hypertension in Chinese OSA patients. This study provided evidences indicating that in addition to AHI and nocturnal hypoxemia, reduced SE may be an important factor of hypertension risk in patients with OSA.

Introduction

Obstructive sleep apnea (OSA) is a common sleep disorder characterized by complete or partial upper airflow cessation during sleep, which affects approximately 17% of men and 9% of women aged 50 to 70 years.[1] Previous studies found that approximately 30~50% of OSA patients reported hypertension and OSA was an important cause of hypertension.[2–4] Hypertension increased the risk of cardiovascular disease and cardiovascular mortality observed in patients with OSA.[5]

There are some evidences indicating a close relationship between sleep quality and hypertension. Previous studies have found that poor sleep quality was associated with higher risk of hypertension.[6–8] However, the majority of these

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studies assessed sleep quality only based on subjective measurements,[6–8] which are more variable than objective measurements. Sleep efficiency (SE), which is an objective indicator, has been used to evaluate sleep quality. Former studies have already confirmed that poor SE was associated with diabetes, metabolic syndrome, and hypertension.[9–12]

Because both OSA and SE are separately associated with hypertension, the co-occurrence of poor SE in OSA patients may further increase the risk of hypertension in this population. However, up to now, the relationship between low SE and hypertension in OSA patients has not been determined. The subjects of previous studies only came from European community.[12–15] To our knowledge, there is no evidence on the association between SE and hypertension in Chinese OSA patients based on PSG records. Therefore, in this study, we aimed to explore the relationship between SE and the prevalence of hypertension in a large cohort of Chinese OSA patients.

Methods

Subjects

This cross-sectional study included patients from Sleep Medical Center, XuanWu hospital. The study procedure was approved by the ethics committee of XuanWu hospital of Capital Medical University (protocol No. Clinical research 2021–185). As the study was performed retrospectively for reviewing participant PSG records, and involved no manipulation by drug or device, the institutional review board specifically approved the informed consent waiver because of the anonymous and purely observational nature of this study. Patient data confidentiality was covered by compliance with the Declaration of Helsinki.

All participants were Chinese adults (age >18 years) in our study. A comprehensive questionnaire was used for all research subjects to collect information, including general health, tobacco use, alcohol drinking, sleep complaints and medical history. The diagnosis of diabetes mellitus (DM)[16] and coronary heart disease (CHD)[17] were determined by specialists according to clinical manifestations and auxiliary examinations.

In this study, patients with an apnea hypopnea index (AHI) ≥5 events/hour were classified as OSA group, whereas individuals with an AHI <5 events/hour were regarded as primary snoring group. Patients with OSA were further stratified according to disease severity as follows: mild OSA (AHI 5~15 events/hour), moderate OSA (AHI 15~30 events/hour), and severe OSA (AHI >30 events/hour). The exclusion criteria are as follows: (1) subjects were diagnosed or treated OSA before; (2) individuals were taking sleep-disrupting medical condition; (3) subjects were diagnosed other comorbid sleep disorders; (4) participants slept fewer than 3 hours during PSG or missed BP data.

Blood Pressure Measures

Blood pressure was measured manually in triplicate with a 5-minute interval in the evening before PSG and in the morning after PSG. Recorded BP was the average of the second and third measurements. Hypertension was defined as (1) diastolic BP (DBP)≥90 mmHg and/or systolic BP (SBP)≥140 mmHg at either evening or morning measurement; (2) participants have used anti-hypertensive medication before; (3) subjects were diagnosed hypertension as a clinical history. We used the average of evening and morning BP for analysis.

Polysomnography

All participants underwent overnight PSG monitoring in the sleep medical center of XuanWu Hospital (Australia Compumedics Grael). Sleep data were automatically collected on the computer to analysis (ProFusion PSG Software). SE was defined as the percentage of the time spent asleep to total time in bed and categorized into three grades (≥85%, 80%~84.9%, and <80%). The highest SE percentage group in OSA and primary snoring group was considered as a reference. Wake after sleep onset (WASO) was defined as the total arousal time from the beginning of sleep to the time of waking. Sleep latency (SL) was calculated from lying in bed to sleeping. Total sleep duration was defined as the total sleep time. Total arousal events were calculated as the total number of arousals to the total sleep time. The percentage of rapid eye movement percentage (REM%) was described as percentage of total sleep time. Slow-wave sleep (SWS) was expressed as the third stage of non-REM sleep. AHI was defined as all apnea (more than 90% reduction of airflow for at

least 10s) and hypopnea (≥50% reduction of airflow for at least 10s accompanied with more than 3% decrease in arterial oxygen saturation (SaO_2)) occurrences per hour of sleep.

Statistical Analysis

Data were presented as mean ± standard deviation (SD) for continuous variables, and categorical variables were presented as percentages. Comparisons between hypertensive and non-hypertensive patients were conducted using ANOVA, independent-sample t-tests or Mann–Whitney *U*-tests for normally distributed and skewed continuous variables, respectively. Chi-square tests were performed for categorical variables.

Logistic regression models were used to examine the associations of SE and hypertension in all participants, OSA and primary snoring group, respectively. Next, considering the joint effect of OSA and SE, we assessed the association of SE and OSA with hypertension by using primary snoring as a reference group. Moreover, we examined the SE×OSA interaction by comparing OSA to primary snoring in the same SE grade. Model 1 was adjusted for age, sex, BMI (body mass index); Model 2 was adjusted for Model 1 and CHD, DM, tobacco use, and alcohol drinking; Model 3 was adjusted for Model 2, heart rate, oxygen desaturation index, time with sat <90%, daytime sleepiness, lowest oxygen saturation during sleep (Lowest SpO_2) and AHI. The unadjusted and adjusted odds ratio (OR) and 95% CI from logistic regression models were used to present the relationship between SE and hypertension in OSA and primary snoring group.

In this study, we divided age into two groups (<60 years and ≥60 years) to assess the relationship between SE and hypertension in OSA patients. Besides, BMI (<30 vs ≥30), sex (male vs female) and AHI (≥15 vs <15) were divided into subgroups to examine the relationship between SE and hypertension in OSA group.

Data were analyzed using SPSS 26.0, and comparisons with P values <0.05 were considered statistically significant.

Results

Basic Characteristics

A total 3124 participants (2360 OSA and 764 primary snoring patients, 46.48±13.25 years) were included in our study. The baseline characteristics of participants are shown in Table 1. The proportion of hypertension in OSA group was significantly higher than primary snoring group (61.6% vs 32.3%). In OSA and primary snoring group, hypertension was more prevalent among subjects who were older, with higher BMI, with more CHD and DM. Furthermore, participants with hypertension had significantly elevated WASO, oxygen desaturation index and AHI compared to controls both in OSA and primary snoring group. On the other hand, SE, total sleep duration, and the lowest SpO_2 were significantly lower in individuals with hypertension than in those without hypertension.

SE and Hypertension

Figure 1 illustrates the frequency of hypertension and BP levels in all patients, OSA and primary snoring group stratified by SE. Compared with the higher percentages of SE (≥85% and 80%~84.9%), those with the lowest percentages of SE (<80%) exhibited higher odds of hypertension and higher levels of SBP and DBP in all participants and OSA patients (Figure 1A-F). In primary snoring group, hypertension was more frequent and SBP was higher in those with <80% SE than those with 80% to 84.9% SE and those with ≥85% SE, not DBP (Figure 1G-I).

As shown in Table 2, SE presented a significant association with hypertension only in OSA group. In OSA group, patients with <80% SE and those with 80% to 84.9% SE had significantly higher likelihood of having hypertension than those with ≥85% SE, respectively (OR = 1.248, 95% CI 1.018~1.531, *P*=0.033; OR = 1.380, 95% CI 1.040~1.832, *P*=0.026). However, no significant relationship between SE and hypertension was found in the primary snoring group.

In Table 3, we examined the joint effect of OSA and SE on hypertension. Compared to primary snoring, patients with OSA with <80% SE and those with 80% to 84.9% SE had significantly increased odds of hypertension (OR = 1.295, 95% CI 1.003~1.671, *P*=0.047; OR = 1.405, 95% CI 1.017~1.940, *P*=0.039). In contrast, OSA patients with ≥85% SE did not have significantly higher ratio of hypertension than primary snorers (OR = 1.016, 95% CI 0.783~1.318, *P*=0.905). We further conducted logistic regression analysis to examine the SE×OSA interaction by comparing OSA to primary snoring in the same SE grade. As compared to primary snoring within the same SE grade, odds of prevalent hypertension in those with OSA with <80%

Table 1 Clinical Characteristics of 3124 Study Subjects (n = 3124)

Variables	Total (n=3124)	OSA(n=2360)		*P* value	Primary Snoring(n=764)		*P* value
		HT(n=1453)	Non-HT (n=907)		HT(n=247)	Non-HT (n=517)	
Age(y)	46.48±13.25	50.17±12.54	43.64±12.66	<0.001	49.37±13.22	39.70±12.40	<0.001
Gender, n(%)				0.965			0.127
Male	2354 (75.40)	1178 (81.10)	736 (81.10)	-	152 (61.50)	288 (55.70)	-
Female	770 (24.60)	275 (18.90)	171 (18.90)	-	95 (38.50)	229 (44.30)	-
BMI, n(%)				<0.001			<0.001
≤24.9(kg/m^2)	939 (30.10)	238 (16.40)	259 (28.60)	-	101 (40.90)	341 (66.00)	-
25~29.9(kg/m^2)	1459 (46.70)	724 (49.80)	470 (51.80)	-	116 (47.00)	149 (28.80)	-
≥30(kg/m^2)	726 (23.20)	491 (33.80)	178 (19.60)	-	30 (12.10)	27 (5.20)	-
Tobacco use, n(%)	1067 (34.20)	566 (39.00)	326 (35.94)	0.142	69 (27.90)	106 (20.50)	0.022
Alcohol drinking, n(%)	953 (30.50)	525 (36.10)	280 (30.90)	0.009	67 (27.10)	81 (15.70)	<0.001
Diabetes, n(%)	349 (11.20)	202 (13.90)	64 (7.05)	<0.001	49 (19.80)	34 (6.60)	<0.001
CHD, n(%)	216 (6.90)	147 (10.12)	35 (3.86)	<0.001	22 (8.90)	12 (2.30)	<0.001
SBP, mmHg	123.53±15.05	132.15±14.17	115.58±9.12	<0.001	128.35±12.98	110.95±9.44	<0.001
DBP, mmHg	84.17±12.56	90.59±10.10	78.20±6.21	<0.001	85.97±9.72	74.98±6.81	<0.001
HR, times/min	66.23±9.37	67.46±10.02	66.57±9.04	0.037	63.54±7.49	63.44±7.93	0.861
Sleep characteristics							
Sleep efficiency(%)	79.10±14.26	76.89±15.03	80.96±13.56	<0.001	78.24±14.39	82.45±11.89	<0.001
WASO(min)	81.95±55.57	90.46±58.24	75.42±53.02	<0.001	84.10±55.46	68.45±47.73	<0.001
Sleep latency(min)	5.05±14.25	5.44±17.48	4.40±10.10	0.126	5.76±12.28	4.76±10.81	0.256
Total sleep duration(min)	346.08±66.09	334.83±69.97	356.42±62.27	<0.001	341.05±65.97	361.99±54.45	<0.001
Total arousal events	48.95±27.74	55.16±30.50	48.66±27.55	<0.001	38.97±17.35	36.81±16.14	0.091
REM(%)	9.49±8.89	8.86±9.40	9.60±8.62	0.056	10.87±7.57	10.43±8.29	0.485
SWS(%)	26.94±18.06	24.98±18.97	28.35±17.52	<0.001	26.71±16.79	30.11±16.23	0.008
AHI, events/h	28.96±26.99	41.19±25.97	32.11±23.78	<0.001	2.34±1.49	1.80±1.49	<0.001
Lowest SpO_2	79.23±11.57	74.48±11.64	78.04±10.13	<0.001	88.90±4.26	90.05±3.47	<0.001
Oxygen desaturation index	7.51±4.91	9.12±5.26	7.77±4.68	<0.001	4.60±1.86	3.94±1.87	<0.001
Time with Sat<90%(min)	38.84±65.09	57.48±73.23	39.65±63.69	<0.001	2.02±10.93	2.63±20.69	0.663
Daytime sleepiness(%)	1504 (48.14)	759 (52.24)	437 (48.18)	0.055	97 (39.27)	211 (40.81)	0.685

Note: Values are expressed as number (%) or as mean±SD, The *P* values represent the difference between two groups.
Abbreviations: AHI, apnea-hypopnea index; Lowest SpO_2, lowest oxygen saturation during sleep; OSA, obstructive sleep apnea; REM, rapid eye movement sleep; SWS, slow-wave sleep; CHD, coronary heart disease; WASO, wake after sleep onset; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate.

SE were significant (OR = 1.532, 95% CI 1.095~2.143, *P*=0.013), while odds were not significant in the other two grades (≥85% and 80%~84.9%) (Table 4). These results indicated that in addition to AHI and nocturnal hypoxemia, reduced SE may be an important factor of hypertension risk in patients with OSA.

Subgroup Analysis

We investigated the association between SE and hypertension risk in subgroups analyses with OSA group stratified by age (≥60 years vs <60 years), sex (male vs female), BMI (≥30 kg/m^2 vs <30 kg/m^2) and AHI (≥15 events/h vs <15 events/h). The association between hypertension and SE was present in female, younger ages (<60 years old), obese, moderate and severe OSA patients (Table 5).

Discussion

We examined the association between SE and the prevalence of hypertension in a large population of OSA and primary snoring patients. Our results found that poor SE (<85%) measured by PSG was associated with hypertension risk only in OSA subjects. This relationship was evident especially in female, younger ages (<60 years old), obese, moderate and severe OSA patients.

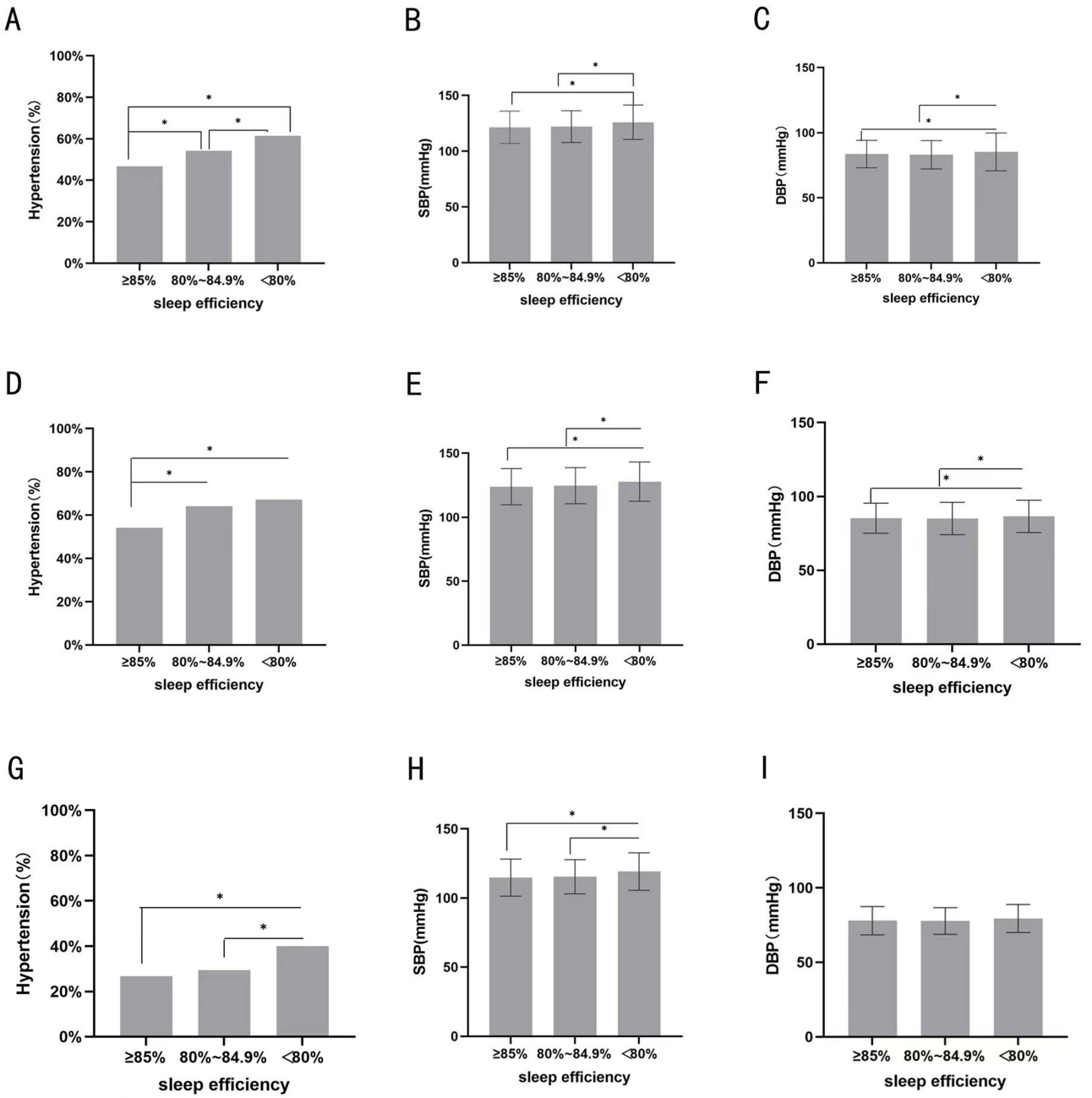

Figure 1 Frequency of hypertension and SBP and DBP levels across different grades of SE in all participants, OSA and primary snoring groups. (**A**) The proportion of hypertension in different SE categories in all participants. (**B**) SBP in different SE categories in all participants. (**C**) DBP in different SE categories in all participants. (**D**) The proportion of hypertension in different SE categories in OSA group. (**E**) SBP in different SE categories in OSA group. (**F**) DBP in different SE categories in OSA group. (**G**) The proportion of hypertension in different SE categories in primary snoring. (**H**) SBP in different SE categories in primary snoring. (**I**) DBP in different SE categories in primary snoring.

Notes: Error bars indicate standard deviation. *$P < 0.05$.

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure.

SE is an objective indicator to evaluate sleep quality. Multiple cross-sectional and longitudinal studies have demonstrated a significant relationship between SE and hypertension.[12–15,18–21] Four cross-sectional studies found that reduced SE, as measured by actigraphy or a contactless biomotion sleep sensor, was significantly related to an increased prevalence of hypertension[12–14,20] and impaired flow-mediated dilation,[21] which implicating poor sleep as a CVD risk factor. Furthermore, two longitudinal studies reported that lower SE was independently associated with higher risk of hypertension[18] and BP nondipping[19] after a mean follow-up of 7 and 7.4 years, respectively. Nevertheless, in the majority

Table 2 ORs and 95% CIs for Sleep Efficiency Associated with Hypertension in All Subjects and Subgroup Analysis with or Without OSA

		OR(95% CI)					
Predictors	**n**	**Model 1**	***P* value**	**Model 2**	***P* value**	**Model 3**	***P* value**
All subjects	3124						
Sleep efficiency							
<80%	1391	1.233 (1.034,1.469)	0.019	1.206 (1.011,1.439)	0.038	1.177 (0.982,1.411)	0.078
80%~84.9%	449	1.220 (0.961,1.549)	0.103	1.199 (0.943,1.524)	0.138	1.222 (0.957,1.561)	0.108
≥85%	1284	Reference		Reference		Reference	
OSA	2360						
Sleep efficiency							
<80%	1096	1.309 (1.074,1.595)	0.008	1.275 (1.045,1.557)	0.017	1.248 (1.018,1.531)	0.033
80%~84.9%	323	1.376 (1.043,1.816)	0.024	1.354 (1.025,1.788)	0.033	1.380 (1.040,1.832)	0.026
≥85%	941	Reference		Reference		Reference	
Primary snoring	764						
Sleep efficiency							
<80%	295	0.991 (0.672,1.463)	0.965	1.002 (0.676,1.483)	0.993	0.993 (0.668,1.476)	0.971
80%~84.9%	126	0.872 (0.527,1.444)	0.596	0.885 (0.530,1.477)	0.640	0.878 (0.524,1.470)	0.620
≥85%	343	Reference		Reference		Reference	

Notes: Model 1 was adjusted for age, sex, BMI; Model 2 was adjusted for Model 1 and CHD, diabetes, tobacco use, and alcohol drinking; Model 3 was adjusted for Model 2, heart rate, oxygen desaturation index, time with sat<90%, daytime sleepiness, lowest oxygen saturation during sleep and AHI.
Abbreviations: OSA, obstructive sleep apnea; 95% CI, 95% confidence interval.

Table 3 Adjusted ORs and 95% CIs for the Joint Effect of OSA and Sleep Efficiency on Hypertension

Predictors	**n**	OR(95% CI)					
		Model 1	***P* value**	**Model 2**	***P* value**	**Model 3**	***P* value**
Primary snoring	764	Reference		Reference		Reference	
OSA-SE							
<80%	1096	1.940 (1.549,2.430)	<0.001	1.930 (1.539,2.421)	<0.001	1.295 (1.003,1.671)	0.047
80%~84.9%	323	2.061 (1.528,2.780)	<0.001	2.071 (1.533,2.798)	<0.001	1.405 (1.017,1.940)	0.039
≥85%	941	1.502 (1.198,1.883)	<0.001	1.537 (1.224,1.930)	<0.001	1.016 (0.783,1.318)	0.905

Notes: Model 1 was adjusted for age, sex, BMI; Model 2 was adjusted for Model 1 and CHD, diabetes, tobacco use, and alcohol drinking; Model 3 was adjusted for Model 2, heart rate, oxygen desaturation index, time with sat<90%, daytime sleepiness, lowest oxygen saturation during sleep and AHI.
Abbreviations: OSA, obstructive sleep apnea; SE, sleep efficiency; 95% CI, 95% confidence interval.

Table 4 Adjusted ORs and 95% CIs for the Association Between OSA and Hypertension Across Different Sleep Efficiency

Sleep Efficiency	**n**	OR(95% CI)					
		Model 1	***P* value**	**Model 2**	***P* value**	**Model 3**	***P* value**
≥85%							
Primary snoring	343	Reference		Reference		Reference	
OSA	941	1.470 (1.069,2.022)	0.018	1.516 (1.100,2.089)	0.011	1.132 (0.790,1.621)	0.500
80%~84.9%							
Primary snoring	126	Reference		Reference		Reference	
OSA	323	2.156 (1.294,3.594)	0.003	2.358 (1.385,4.015)	0.002	1.455 (0.798,2.654)	0.221
<80%							
Primary snoring	295	Reference		Reference		Reference	
OSA	1096	1.883 (1.397,2.539)	<0.001	1.844 (1.363,2.495)	<0.001	1.532 (1.095,2.143)	0.013

Notes: Model 1 was adjusted for age, sex, BMI; Model 2 was adjusted for Model 1 and CHD, diabetes, tobacco use, and alcohol drinking; Model 3 was adjusted for Model 2, heart rate, oxygen desaturation index, time with sat<90%, daytime sleepiness and lowest oxygen saturation during sleep.
Abbreviations: OSA, obstructive sleep apnea; 95% CI, 95% confidence interval.

Table 5 ORs and 95% CIs for Sleep Efficiency Associated with Hypertension in Subgroup OSA Patients

	OR(95% CI)					
Predictors	**Model 1**	***P* value**	**Model 2**	***P* value**	**Model 3**	***P* value**
Male						
<80%	1.192 (0.958,1.484)	0.115	1.151 (0.923,1.436)	0.212	1.139 (0.908,1.429)	0.262
80%~84.9%	1.344 (0.989,1.828)	0.059	1.309 (0.962,1.782)	0.087	1.336 (0.975,1.830)	0.071
≥85%	Reference		Reference		Reference	
Female						
<80%	1.990 (1.251,3.167)	0.004	2.069 (1.291,3.314)	0.003	1.928 (1.181,3.145)	0.009
80%~84.9%	1.560 (0.815,2.987)	0.180	1.609 (0.835,3.100)	0.155	1.629 (0.832,3.189)	0.155
≥85%	Reference		Reference		Reference	
Age≥60						
<80%	1.057 (0.612,1.827)	0.842	1.030 (0.593,1.789)	0.916	1.078 (0.607,1.916)	0.797
80%~84.9%	0.927 (0.428,2.008)	0.847	0.894 (0.411,1.942)	0.777	0.901 (0.406,2.000)	0.797
≥85%	Reference		Reference		Reference	
Age<60						
<80%	1.317 (1.061,1.635)	0.013	1.292 (1.040,1.607)	0.021	1.286 (1.032,1.604)	0.025
80%~84.9%	1.423 (1.054,1.921)	0.021	1.415 (1.047,1.911)	0.024	1.447 (1.066,1.964)	0.018
≥85%	Reference		Reference		Reference	
BMI≥30 kg/m^2						
<80%	1.617 (1.085,2.410)	0.018	1.579 (1.056,2.360)	0.026	1.573 (1.044,2.371)	0.030
80%~84.9%	1.643 (0.906,2.980)	0.102	1.601 (0.878,2.921)	0.125	1.648 (0.895,3.036)	0.109
≥85%	Reference		Reference		Reference	
BMI<30 kg/m^2						
<80%	1.235 (0.982,1.553)	0.071	1.216 (0.965,1.533)	0.097	1.227 (0.970,1.553)	0.088
80%~84.9%	1.303 (0.950,1.787)	0.101	1.285 (0.936,1.765)	0.121	1.301 (0.942,1.796)	0.111
≥85%	Reference		Reference		Reference	
AHI≥15 events/h						
<80%	1.380 (1.098,1.734)	0.006	1.338 (1.062,1.685)	0.013	1.330 (1.049,1.687)	0.019
80%~84.9%	1.398 (1.008,1.939)	0.045	1.365 (0.982,1.895)	0.064	1.426 (1.020,1.995)	0.038
≥85%	Reference		Reference		Reference	
AHI<15 events/h						
<80%	1.069 (0.710,1.610)	0.748	1.008 (0.665,1.527)	0.970	1.023 (0.672,1.558)	0.914
80%~84.9%	1.281 (0.752,2.182)	0.363	1.232 (0.721,2.108)	0.445	1.159 (0.671,2.003)	0.597
≥85%	Reference		Reference		Reference	

Notes: Model 1 was adjusted for age, sex, BMI; Model 2 was adjusted for Model 1 and CHD, diabetes, tobacco use, and alcohol drinking; Model 3 was adjusted for Model 2, heart rate, oxygen desaturation index, time with sat <90%, daytime sleepiness, lowest oxygen saturation during sleep and AHI.
Abbreviations: BMI, body mass index; 95% CI, 95% confidence interval.

of these studies, participants almost came from community. The effect of decreased SE on hypertension in OSA patients has not been fully explored.

In a retrospective study of 151 OSA patients who underwent PSG, Friedman et al[11] found that subjects with resistant hypertension were associated with a reduction in sleep efficiency of 7.9% and 10.2% compared to subjects with controlled hypertension and normotension, respectively. Another cross-sectional study[13] found that individuals with reduced SE had a higher level of SBP/DBP and a higher prevalence of hypertension, including sleep disordered breathing (SDB) subjects. Furthermore, Ramos et al[14] demonstrated that participants (included mild SDB patients) with hypertension had a decreased sleep efficiency compared with participants without hypertension. Our results were consistent with the prior studies that OSA patients with reduced SE measured by PSG were associated with a higher risk of hypertension. Moreover, when examining the joint effect of OSA and SE on hypertension, compared to primary snoring, patients with OSA with <80% SE and those with 80% to 84.9% SE had significantly increased odds of hypertension. Our study provided evidences indicating that in addition to AHI and nocturnal hypoxemia, reduced SE may be an important factor of hypertension risk in patients with OSA. However, on the other hand, Thomas et al[22] found that SE was not associated with either nocturnal hypertension or nondipping SBP in

subjects, which including likelihood of OSA patients. The reason why the results were different from our study may be that SE was determined by subjective methods, and OSA was determined using the STOP-Bang questionnaire. Besides, in our study, there was no significant relationship between SE and hypertension in the primary snoring group, which was different from former studies. Zhao et al[13] found that reduced SE was significantly associated with the prevalence of hypertension in participants with AHI <5 events/h. Participants were different ethnic groups and almost middle-aged and older population may be the reason of inconsistent results.

The subgroup analysis showed that the association between hypertension and SE was observed in female, those <60 years old, obese, moderate and severe OSA patients. The results of two former studies were consistent with our results that younger OSA patients were associated with a higher risk of hypertension,[23,24] reflecting the reduced cardiovascular risk for older OSA individuals. As for the association between sex and SE, Kocevska et al demonstrated that women (≥41 years) reported a marginally lower SE than men from a meta-analysis about sleep characteristics in 1.1 million people.[25] Furthermore, hypertension was more prevalent in women with OSA,[26] and the significant relationship between SE and higher BP was driven by women and not men.[27] It is thus conceivable that, OSA women exhibited significant association between hypertension and SE in our study. Moreover, former studies[18,28] found that poor SE and BMI ≥30 kg/m^2 significantly increased the risk of hypertension. The relationship between OSA and BP is most closely associated with the degree of obesity.[29] Therefore, in our population, the association between hypertension and SE was extremely significant in obese OSA patients.

The mechanisms accounting for the association between reduced SE and hypertension remain unknown. It is possible that poor SE with hypertension may be through stimulation of sympathetic nervous system, as measured by increased secretion of catecholamines.[30] Moreover, reduced SE led to low sleep quality, which was demonstrated to damage endothelial function,[21,31] increase baseline brachial artery diameter,[27] aortic arterial stiffness[32] and carotid intima-media thickness,[33] resulting in hypertension and cardiovascular diseases.

There are several strengths in this study. This is the first study to investigate the interaction between reduced SE and hypertension using PSG records in Chinese OSA patients. Moreover, use of a large sample increases generalizability in our study. However, there are still some limitations in our study. First, our study is retrospective, cross-sectional study, and the association between SE and hypertension requires further study to prove. Second, we only measured sleep using one night of PSG, which may do not provide accurate information. Even though some studies found that actigraphy and PSG produced similar estimates of sleep duration and efficiency,[34,35] wrist actigraphy had an advantage because it allowed for multiple sleep monitoring. Thirdly, we obtained only clinic BP measurements. The definition of hypertension was based on the BP measurement and participants medical history, which may produce the bias of the result. Finally, the participants were Chinese, and our findings may not be applicable in other ethnic groups.

Conclusion

In conclusion, our results show that poor SE is correlated with the prevalence of hypertension in Chinese OSA patients, but not in those with primary snoring. Further studies with longitudinal designs are needed to determine the temporal association between SE and hypertension in patients with OSA.

Data Sharing Statement

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Informed Consent

The study procedure was approved by the ethics committee of XuanWu hospital of Capital Medical University (protocol No. Clinical research 2021-185). The institutional review board specifically approved the informed consent waiver because of the anonymous and purely observational nature of this study. Patient data confidentiality was covered by compliance with the Declaration of Helsinki.

Consent for Publication

All authors agreed to submit the manuscript for publication with all details, including images, tables and data.

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Disclosure

All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest, or non-financial interest in the subject matter or materials discussed in this manuscript.

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