Systemic Hypertension in Snorers with and without Sleep Apnea*

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To investigate the impact of sleep-disordered breathing events on daytime hypertension (HT) in patients with increased upper airway resistance during sleep, we studied 191 male snorers aged 49.9 ± 0.8 years. In 116 of them, an apnea-hypopnea index (AHI) above 10—defined as the presence of obstructive sleep apnea (OSA)—was found; the other 75 subjects had an AHI lower than 10 and were classified as habitual snorers (HSN). Prevalence of HT was not different between OSA (56 of 116 = 48 percent) and HSN (33 of 75 = 44 percent) and there was also no difference in systolic, diastolic, and mean blood pressures between the two groups. Hypertensive OSA patients had higher body mass index (BMI) than normotensive OSA subjects (31.4 ± 0.7 vs 29.4 ± 0.6; p < 0.05), but there was no difference in age, AHI, and nocturnal oxygenation parameters. The same was true for the HSN group, with hypertensive subjects being more obese than normotensive subjects (BMI: 30 ± 0.8 vs 27.3 ± 0.8; p < 0.05), but no difference in age and polysomnographic features. Discriminant analysis with HT as the classification variable and age, BMI, AHI, mean, and lowest nocturnal oxyhemoglobin saturation as independent variables, revealed an independent influence on HT only for BMI (F-prob = 0.001). Thus, our results stand against the hypothesis of a causal relationship between sleep-disordered breathing events and daytime hypertension. We conclude that the high prevalence of HT in male snorers is more directly linked to obesity than to sleep apnea, but an independent effect of snoring per se cannot be excluded.

\[ \text{AHI} = \text{apnea-hypopnea index}; \text{BMI} = \text{body mass index}; \text{HSN} = \text{habitual snorers}; \text{HT} = \text{systemic hypertension}; \text{OSA} = \text{obstructive sleep apnea}; \text{SaO}_2 = \text{oxyhemoglobin saturation}; \text{SaO}_2 \text{min} = \text{lowest oxyhemoglobin saturation during sleep}; \text{UAR} = \text{upper airway resistance} \]

Systemic hypertension is frequently found in patients with obstructive sleep apnea (OSA), with reported prevalences ranging from 50 to 90 percent.\(^\text{1,4}\) On the other hand, snoring is a widespread finding among hypertensives,\(^\text{5,6}\) and OSA has been demonstrated in 22 to 48 percent of them.\(^\text{6,9}\)

Whereas blood pressure (BP) normally goes down during sleep,\(^\text{10}\) in OSA this fall in BP may be absent or even replaced by a rise throughout the night with the highest values in the morning.\(^\text{1,11,12}\) Swings in BP during sleep have been shown to parallel dips in \(\text{SaO}_2\)\(^\text{1,13}\) and daytime BP in OSA is correlated with the level of oxygenation during sleep.\(^\text{14}\) From these observations, an association between OSA and HT that is closer than mere coincidence could be derived. However, daytime hypertension in OSA is also closely correlated with age and obesity.\(^\text{15,16}\)

Several studies have shown an increased risk for HT and cardiovascular diseases in snorers.\(^\text{5,16-20}\) Both snoring and OSA are manifestations of a sleep-induced increase in upper airway resistance (UAR). Assuming a causal relationship between increased UAR during sleep and elevated BP during the day, the question arises whether HT in snorers is the consequence of the sleep-induced impairment of upper airway patency itself or if sleep-disordered breathing events are a prerequisite for the development of HT. Hence, this study was designed to determine the impact of sleep-disordered breathing events on the occurrence of HT in snorers.

**MATERIALS AND METHODS**

We studied 191 men aged 49.9 ± 0.8 years. All of them had a history of loud snoring, which was the main reason for referral to the sleep laboratory in 84 of them. Apneas observed by their bed partners were reported by 115 of them, and 103 complained about excessive daytime sleepiness. A history of involuntarily falling asleep during the day was found in 116. All but 27 patients were either normal weight, or, their body mass index (BMI) was higher than 25. Mean BMI of our study population was 29.6 ± 0.4 and 73 of our patients were obese, with a BMI above 30.

All patients underwent full-night polysomnography with continuous recording of EEG, EOG, submental EMG, ECG, air flow at nose and mouth (thermistors), movements of rib cage and abdomen (Respiracare, Ambulatory monitoring, Ardsley, NY), and oxyhemoglobin saturation from the ear (Novametrix 505). Sleep staging was done according to standard criteria.\(^\text{21}\) Apneas were defined as a cessation of airflow at nose and mouth for longer than 10 s. Hypopneas were defined as a reduction in rib cage and abdominal movements to 50 percent or less compared with the preceding five breaths for longer than 10 s accompanied by a fall in \(\text{SaO}_2\) to 92 percent or lower if baseline was equal or above 94 percent or a fall in \(\text{SaO}_2\) of 3 percent or more if baseline was 93 percent or lower. The total number of apneas and hypopneas per hour of sleep represented the apnea-hypopnea index (AHI). Patients with an AHI below 10 were classified as habitual snorers (HSN) and those with an AHI above 10 as OSA.

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Hypertension was defined as either a history of elevated BP together with regular intake of antihypertensive medication or a resting supine BP above 160 mm Hg systolic and/or 95 mm Hg diastolic on two of three measurements on different days, which were the first visit to our outpatient clinic, the morning after the sleep study and the day scheduled for discussion of the sleep study's results. Only those patients not receiving antihypertensive drugs entered calculations and statistics based on absolute values of systolic, diastolic, or mean BP.

For statistical analysis of differences between groups, ANOVA was used. Correlations were calculated by Spearman's rank correlation. Discriminant analysis and linear stepwise regression analysis were done by a software package for microcomputers (BMDP). Results are given as mean ± SEM. Statistical significance was assumed at a p<0.05.

RESULTS

In 116 of the 191 male snorers, an AHI above 10 was found, which was defined as the presence of OSA. The remaining 75 men with an AHI below 10 were classified as HSN. Mean AHI was 36.7±2.1 in the OSA group and 3.6±0.3 in the HSN group. The two groups did not differ in age (50.3±1 vs 49.3±1.2; p = 0.52), but OSA patients were more obese than the HSN group (BMI: 30.4±0.5 vs 28.5±0.6; p = 0.011).

A total of 112 subjects had neither elevated BP on examination nor a history of HT or antihypertensive medication. In 89 subjects, HT according to our definition was found, with only 35 of them reporting regular intake of antihypertensive medication. The spectrum of antihypertensive drugs included calcium channel blockers in 28 patients, diuretics in 11, β-adrenergic antagonists in nine, and central antisympathetic drugs in seven. Whereas HT was sufficiently controlled by one drug in 19 patients, 12 needed two drugs and four patients needed three drugs.

Among the 89 hypertensives, 56 had an AHI above 10 and 33 had an AHI below 10. Thus, the prevalence of HT was not different between OSA and HSN (p = 0.55); it was 48 percent in the OSA group (56 of 116) and 44 percent in the HSN group (33 of 75). This did not change when comparing only the percentage of patients treated for hypertension in the two groups (24 OSA, 11 HSN; p = 0.29). There was also no difference between OSA and HSN looking at the absolute levels of BP in untreated patients of either group (systolic BP: 147±1.5 vs 146±1.8, p = 0.67; diastolic BP: 87±0.7 vs 86±0.8, p = 0.33; mean BP: 111±1 vs 110±1.2, p = 0.49).

Hypertensive and normotensive subjects did not differ in age (50.8±1.1 years vs 49.1±1 years; p = 0.28), but patients with HT were more obese than normotensives (BMI: 30.9±0.5 vs 28.6±0.5; p = 0.0014). Mean AHI was not significantly higher (p = 0.13) in patients with HT (26.6±2.7) than in those without (21.2±2.4).

Prevalences of HT together with the means for systolic, diastolic, and mean BPs in groups with different severity of sleep-disordered breathing are given in Figure 1. Although there was no difference in age and BMI among the four groups, the group with moderate OSA (AHI between 30 and 50), had higher absolute values for BPs than the other groups. However, prevalences of HT were not statistically different among the four AHI groups. Whereas AHI and age explained only 4.4 percent and 3.3 percent of the total variance in mean BP, the contribution of BMI was 15.9 percent, with a stronger influence on diastolic (25.2 percent of total variance) than on systolic BP (9.2 percent of total variance).

Looking at the different BMI groups (Fig 2), there was a marked increase in HT with obesity. However, within the three BMI groups, neither prevalences of HT nor the absolute values of BP were different between OSA and HSN. It seems worth mentioning that we found a surprisingly high percentage prevalence of HT in normal-weight snorers regardless of whether they had OSA or not, but these groups were rather small. Whereas we found a weak but significant correlation between AHI and BMI (Spearman's r = 0.26; p<0.001), age did not correlate with BMI.
(Spearman’s $r = 0.016$) nor with AHI (Spearman’s $r = 0.086$).

Comparison of OSA and HSN with and without HT (Table 1) showed no difference in age, AHI, and nocturnal oxygenation parameters between hypertensive and normotensive subjects in either group, but hypertensive subjects with OSA as well as hypertensive HSN were more obese than their normotensive controls. Although OSA patients as a whole had higher BMI than HSN, hypertensive OSA and hypertensive HSN exhibited comparable degrees of obesity, and there was also no difference in age.

Multiple stepwise linear regression analysis with mean BP as the dependent variable and age, BMI, AHI, mean, and lowest nocturnal SaO$_2$ as independent variables in those 156 subjects classified as normotensive or untreated hypertensives revealed an independent influence for only BMI ($F = 39.43$) and age ($F = 8.54$). The same was true for the OSA patients, whereas in HSN age had no independent influence. With diastolic BP as the dependent variable, the same sort of regression analysis showed an independent influence for solely BMI in either group.

To take into account also those patients receiving antihypertensive medication, discriminant analysis with hypertension as the classification variable was performed. As can be seen from Table 2, only BMI predicted HT, and age, AHI, and nocturnal oxygenation parameters did not contribute significantly.

**DISCUSSION**

This study shows that daytime hypertension in male snorers does not depend on the occurrence of apneas during sleep. As we found equal prevalences of HT in snorers with AHI >10 and snorers with AHI <10 (48 percent vs 44 percent), our results do not support the hypothesis of a direct link between sleep apnea and hypertension. If OSA causes HT, one would expect that patients with more severe OSA are more prone to develop HT or show higher BPs than those with mild disease or only simple snoring, but as in previous studies, neither prevalence of HT nor the absolute levels of BP paralleled polysomnographic features indicating severity of sleep-disordered breathing, such as the number of respiratory events per hour of sleep or the event-related dips in SaO$_2$.

Recently, a study comparing 21 hypertensive with

**Table 1—Comparison of Hypertensive (HT) and Normotensive (NT) Snorers with (OSA) and without (HSN) Sleep Apnea**

<table>
<thead>
<tr>
<th>Variable</th>
<th>OSA/HT (n=56)</th>
<th>OSA/NT (n=60)</th>
<th>HSN/HT (n=33)</th>
<th>HSN/NT (n=42)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>51.1±1.4</td>
<td>49.6±1.4</td>
<td>50.3±1.8</td>
<td>48.5±1.6</td>
</tr>
<tr>
<td>BMI</td>
<td>31.4±0.71</td>
<td>29.4±0.6</td>
<td>30±0.8†</td>
<td>27.3±0.8</td>
</tr>
<tr>
<td>AHI</td>
<td>39.8±2.4</td>
<td>33.9±2.3</td>
<td>4.1±3.1</td>
<td>3.2±2.8</td>
</tr>
<tr>
<td>SaO$_2$min</td>
<td>91.8±0.5</td>
<td>92.8±0.5</td>
<td>95.2±0.7</td>
<td>94.6±0.6</td>
</tr>
<tr>
<td>SaO$_2$min</td>
<td>3.6±1.7</td>
<td>75.6±1.5</td>
<td>86.2±2.2</td>
<td>89.3±1.9</td>
</tr>
</tbody>
</table>

*OSA = obstructive sleep apnea; HSN = habitual snorers; BMI = body mass index; AHI = apnea-hypopnea index; SaO$_2$m = mean oxyhemoglobin saturation during sleep; SaO$_2$min = lowest oxyhemoglobin saturation during sleep.†p<0.05.

**Table 2—Results of Discriminant Analysis with Hypertension as the Classification Variable in 191 Male Snorers**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Step 1</th>
<th>Step 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F-value</td>
<td>F-prob</td>
</tr>
<tr>
<td>BMI</td>
<td>10.5</td>
<td>0.001</td>
</tr>
<tr>
<td>AHI</td>
<td>2.3</td>
<td>0.127</td>
</tr>
<tr>
<td>Age, yr</td>
<td>1.2</td>
<td>0.276</td>
</tr>
<tr>
<td>SaO$_2$min</td>
<td>0.8</td>
<td>0.376</td>
</tr>
<tr>
<td>SaO$_2$m</td>
<td>0.6</td>
<td>0.424</td>
</tr>
</tbody>
</table>

*See Table 1 for explanation of abbreviations.

Figure 2. Prevalences of hypertension and mean blood pressures in normal weight, overweight, and obese snorers.
29 normotensive OSA patients demonstrated no difference in polysomnographic and anthropometric data between the two groups.\textsuperscript{21} We, too, found similar severity of sleep-disordered breathing in hypertensive and normotensive OSA, but in contrast to this study, our sleep apnea patients with hypertension were more obese than those without hypertension. Also, in the group of snorers without sleep apnea, hypertensives had higher BMI than normotensives. From this, obesity appears as the main factor linked to HT in snorers and—as shown by discriminant analysis—sleep-disordered breathing events have no independent influence. Thus, the model for the interrelationship between OSA and HT derived from our study would be that obesity is the common risk factor for both and there is no evidence for OSA to be the linking factor between obesity and HT. 

To date, the question of how sleep-disordered breathing events should cause elevated daytime BP has not yet been answered. The correlation between swings in BP and dips in SaO\textsubscript{2}\textsuperscript{11} during sleep does not explain HT in OSA, not even during the night, because hypoxemia is known to cause vasodilation. However, apnea besides hypoxemia causes hypercapnia and a fall in pH that leads to increased sympathetic activity associated with vasoconstriction.\textsuperscript{24,25} Increased sympathetic activity during sleep and also during wakefulness could explain daytime HT in OSA, but there are no studies addressing this question for increased UAR without apneas and thus without significant changes in gas exchange. Nevertheless, increased UAR may result in frequent arousals from sleep,\textsuperscript{26} and thereby increase sympathetic activity. The hypothesis of a link between disturbed sleep structure in snorers and HT would explain the high overall prevalence of HT in our study population compared with a general population with similar age and BMI.\textsuperscript{27,28} This model is further strengthened by the fact that there were lots of snorers among our study population complaining about daytime sleepiness, but having only few apneas and hypopneas during the sleep study. Accordingly, snoring \textit{per se} could be a risk factor for HT. However, except in patients with congenital facial abnormalities or impaired upper airway patency due to nasal or pharyngeal abnormalities, the development of snoring usually parallels weight gain, so that once again obesity and not increased UAR itself would be the primary cause of HT in these patients.

Although age had some independent influence on the level of mean BP in normotensives and untreated hypertensives, we failed to find an increase in prevalence of HT with age in our study population as a whole. This can be interpreted as an indirect sign of earlier appearance of HT in snorers, \textit{i.e.}, snoring could eventually accelerate the development of HT. However, due to the considerable amount of overweight in our patients, especially in the younger age groups, and the lack of an age- and weight-matched nonsnoring control group, we are unable to decide whether HT in the younger patients was an effect of snoring \textit{per se} or of obesity. Although we found a strong correlation between HT and obesity, there was also a relatively high prevalence of HT in normal-weight snorers. Thus, as supposed earlier,\textsuperscript{29,30} snoring could have an independent effect only in patients without significant overweight.

In summary, this study shows high prevalence of HT in male snorers that is not different whether or not there is sleep apnea. Although an independent influence of increased UAR during sleep cannot be ruled out from our data, obesity appears to be the main factor linked to HT in this population; the number of sleep-disordered breathing events as well as the degree of nocturnal hypoxemia do not predict hypertension in snorers.

\section*{References}

22 Hirshkowitz M, Karacan I, Gurakar A, Williams RL. Hypertension, erectile dysfunction, and occult sleep apnea. Sleep 1989; 12:223-32

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