Although a high prevalence of hypertension has been observed in snorers, whether there is a direct link between hypertension and snoring remains controversial. It has recently been demonstrated that an abnormal amount of breathing effort during snoring is responsible for sleep fragmentation even in the absence of sleep apnea syndrome criteria. We hypothesized that sleep fragmentation during snoring may be a direct risk factor for the development of hypertension. On the basis of polysomnographic data, 105 nonapneic patients between 40 and 65 years of age referred for snoring with social impairment were selected and categorized as snorers with (n=55) or without sleep fragmentation (n=50) based on whether the arousals index was 10 or greater or less than 10/h of sleep, respectively. Sleep distribution did not differ between the two groups, except for a longer duration of wake after sleep onset (58±43 min vs 42±38 min) and a shorter duration of sleep in the group with sleep fragmentation (72±34 min vs 97±34 min). Although there were no statistically significant differences between the snorers with and without sleep disruption in terms of age (51.3±7.7 vs 48.6±6.0 years), body mass index (26.9±4·0 vs 27.2±5·5 kg/m²), sex ratio, respiratory indexes during sleep, daytime sleepiness, and daytime tiredness, prevalence of systemic hypertension was significantly higher in the sleep-fragmented group (20/55 vs 7/50). This significant difference persisted (16/51 vs 8/49) when patients using antihypertensive drugs with possible effects on the CNS were excluded. Our data suggest that sleep fragmentation is common in patients who seek medical help for snoring with social impairment and may play a role in the development of hypertension.

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Key words: arousal; hypertension; sleep fragmentation; snoring

There is evidence that habitual snoring may be associated with hypertension\(^1\)\(^2\) and cardiovascular diseases in general.\(^3\)\(^4\) Although the mechanisms underlying these associations are unknown, obstructive sleep apnea, which may be highly prevalent in habitual snorers, is thought to be a major contributing factor.\(^5\) However, hypertension has been observed in nonapneic snorers. It has been shown that arterial BP in nonapneic snorers is not directly related to snoring but is associated with the degree of obesity.\(^6\) Recent reports have demonstrated that an abnormal amount of breathing effort during snoring, even in the absence of sleep apnea, can disrupt sleep architecture by causing very short arousals\(^7\) \(^8\) that are associated with a burst of sympathetic nervous system activity and transient increases in BP.\(^9\)\(^10\) We hypothesized that arousals from sleep may promote the development of hypertension. To examine the association of nonapneic snoring with abnormal sleep architecture and hypertension, we conducted a prospective study in middle-aged nonapneic heavy snorers.

**Materials and Methods**

All patients between 40 and 65 years of age who attended the otolaryngology clinic for snoring with social impairment between January 1994 and June 1995 were referred to our sleep laboratory. Between 6 PM and 8 PM, they were equipped with an ambulatory polysomnography device designed to perform electroencephalography (C\(_4\)-A\(_1\), C\(_3\)-A\(_2\)), electro-oculography, chin electromyography, electromyography of the tibialis anterior of both legs, oronasal airflow recordings, and rib cage movement recordings (Multi-Parameter Analysis recorder 2/Medilog 9200; Oxford Medical Instrument; Abingdon, England) and with an arterial pulse oximeter device (Nellcor B5; Nellcor Inc; Hayward, Calif). BP measurements were obtained using a mercury sphygmomanometer after a 1-h rest in a relaxed sitting position, during which time the leads were placed. Each patient completed a questionnaire, including (1) a French version of the Epworth sleepiness scale...
(ESS), and (2) a cardiac health history and an inventory of current medications. The patients were then asked to return home and to go to bed and get up in the next morning at their usual times. "Lights out" and "lights on" times were noted.

Sleep staging was performed according to standard criteria. EEG arousals were detected on the basis of an abrupt shift in EEG frequency, including alpha and/or frequencies greater than 16 Hz but not spindles, and were scored according to standard criteria. An abnormal breathing event during objectively measured sleep was defined according to commonly used clinical criteria as either complete cessation of airflow lasting 10 s or more (apnea) or a drop of 50% or greater fall in oronasal airflow for 10 s or more (hypopnea). The average number of episodes of apnea (apnea index [AI]) and apnea–hypopnea index (AHI) per hour of sleep was used as a summary measurement of sleep-disordered breathing.

Sleep apnea syndrome was ruled out on the basis of AHI and AI values of less than 10 and 5/h of sleep, respectively, and of a duration of episodes with an arterial oxygen saturation (SaO2) of less than 90% of less than 1 min. Nonapneic snorers were classified as sleep disrupted or nonsleep disrupted based on whether the number of arousals per hour sleep was 10 or more or less than 10.5

The daytime tiredness syndrome was defined as a "yes" response to an inquiry about presence of tiredness in the morning or throughout the day.

Hypertension was defined based on the following criteria: (1) previous diagnosis of hypertension and treatment with antihypertensive drugs, or (2) diastolic BP equal to or greater than 95 mm Hg and/or systolic BP equal to or greater than 160 mm Hg, confirmed by a repeated measurement of BP by a physician within the 3 months. We chose the diastolic and systolic thresholds to meet the criteria proposed by the World Health Organization to institute drug treatment in the absence of other cardiovascular risk factors after a follow-up period.

Results are expressed in the text and tables as means±SD. The differences between snorers with and without sleep disruption were compared using a Mann-Whitney test. Comparisons of prevalence rates between two groups were done using χ² analysis. The significance level was set at 5%.

**RESULTS**

Among 216 middle-aged snorers studied consecutively in our sleep laboratory, 105 were nonapneic snorers. Among these 105 patients, 55 had frequent (ie, ≥10/h) transient EEG arousals. Arousal index was 15.4±4.5/h of sleep in this group, vs 6.6±1.8 in the remaining 50 patients. No arousals due to periodic limb movements were recorded. General characteristics of the patients are presented in Table 1. Age, sex ratio, body mass index, daytime tiredness, and ESS scores were similar in both groups.

We found that hypertension was significantly more prevalent in the group with sleep fragmentation than in the group without sleep fragmentation (20/55, including 6/11 women vs 7/50, including 0/12 women) (Table 1). Four patients and one patient in the sleep-fragmented and nonsleep-fragmented groups, respectively, used β-adrenoceptor antagonists. Except β-adrenoceptor antagonists, no antihypertensive drugs with possible central and/or sleep effects were used. Thus, when patients using antihypertensive drugs with possible effects on sleep parameters were eliminated, hypertension remained significantly more prevalent in the group with sleep fragmentation (16/51, including 6/11 women) than in the group without sleep fragmentation (6/49).

Although use of antihypertensive drugs was more prevalent in the group with sleep fragmentation than in the group without sleep fragmentation (14/55 vs 5/50), diastolic arterial BP was significantly higher in the sleep-fragmented group (Table 1). However, no significant correlations between the arousal index and any arterial pressure were observed. Diastolic BP remained 95 mm Hg or greater and/or systolic BP remained 160 mm Hg or greater in two patients with sleep fragmentation despite use of antihypertensive drugs without β-adrenoceptor antagonist effects (Table 1).
of sleep fragmentation.\textsuperscript{17}

Definition of sleep fragmentation as an arousal index greater than 10/h of sleep\textsuperscript{4} is somewhat arbitrary. Although it has been shown that normal young subjects have no more than ten short EEG arousals per hour of sleep,\textsuperscript{18} to our knowledge, no data are available in older subjects such as those included in our study. Our decision to select ten arousals per hour as the cutoff was based mainly on a study in middle-aged snorers with sleep fragmentation, which showed that successful treatment of snoring was associated with a decrease in the arousal index below 10/h.\textsuperscript{7} In addition, in our study, slow-wave sleep values were normal in subjects with an arousal index below 10/h (Table 2) but not in those with an arousal index above 10/h. This finding is evidence that arousals occurring more often than 10 times per hour can have a significant impact on nocturnal sleep architecture.

Surprisingly, patients with sleep fragmentation and short slow-wave sleep duration did not complain of excessive daytime somnolence. In our study, the degree of somnolence was assessed only on the basis of the ESS. Scores on this simple scale were correlated with scores on the multiple sleep latency test\textsuperscript{19} in a large group of patients with a broad array of sleep disorders (r=-0.38, p<0.001, n=138).\textsuperscript{11} Similarly, we found only a weak inverse correlation between the ESS score and sleep latency in 27 patients with sleep apnea syndrome (r=-0.43, p=0.03) (unpublished results). It can be postulated that our patients, similar to some patients with sleep apnea,\textsuperscript{20} were poor judges of their own chronic level of sleepiness because their main complaint was snoring with social impairment. Use of a more objective test such as the multiple sleep latency test would perhaps have demonstrated a difference in daytime sleepiness between nonapneic snorers with or without sleep fragmentation.

Several studies demonstrated a significant association between snoring and a history of hypertension.\textsuperscript{1,4,21,22} Some of these studies found that the

duration was significantly shorter in the group with sleep fragmentation. Furthermore, there was a significant correlation between the arousal index and duration of wake after sleep onset (r=0.40, p=0.0001) and a negative correlation between the arousal index and duration of stage 4 sleep (r=-0.29, p<0.005).

**Discussion**

Our data from all-night home polysomnography recordings suggest a direct link between diurnal hypertension and sleep fragmentation in a group of habitual heavy snorers with social impairment who did not meet sleep apnea syndrome criteria.

Before discussing the implications of our findings, we will address several methodologic issues.

Because some antihypertensive medications, including some beta-blockers, may decrease sleep quality and induce sleep fragmentation, the question arose of whether arousal might be a consequence of antihypertensive medication use rather than the cause of the hypertension. To resolve this issue, we analyzed our data after excluding those patients who used antihypertensive drugs with possible CNS effects: significant differences in hypertension prevalence persisted between the group with sleep fragmentation and the group without sleep fragmentation.

Recent studies suggest that arousals during snoring are related to the degree of inspiratory effort.\textsuperscript{7,8,16} Thus, confirmation that arousals are linked to snoring would require monitoring of respiratory effort by esophageal pressure measurement during polysomnography. Esophageal pressure cannot be determined with portable recording devices. We obtained confirmation that arousals were related to respiratory effort in eight patients who underwent a second all-night sleep study, including esophageal pressure monitoring at our laboratory. In addition, leg electromyograms showed that none of our patients had arousals due to periodic limb movements, which, together with abnormal breathing effort, are the most common cause of sleep fragmentation.\textsuperscript{17}

<table>
<thead>
<tr>
<th>Sleep-Fragmented Nonapneic Snorers (n=55)</th>
<th>Nonsleep-Fragmented Nonapneic Snorers (n=50)</th>
<th>Statistics: Mann-Whitney</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time, min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>382±69</td>
<td>397±75</td>
<td>NS*</td>
</tr>
<tr>
<td>Wake after sleep onset, min</td>
<td></td>
<td>p&lt;0.02</td>
</tr>
<tr>
<td>58±43</td>
<td>42±38</td>
<td>NS</td>
</tr>
<tr>
<td>Sleep onset latency, min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25±21</td>
<td>25±19</td>
<td></td>
</tr>
<tr>
<td>Slow-wave sleep, min</td>
<td></td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>72±34</td>
<td>97±34</td>
<td>NS</td>
</tr>
<tr>
<td>Rapid eye movement sleep, min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>78±27</td>
<td>79±29</td>
<td>NS</td>
</tr>
<tr>
<td>AI, per hour of sleep</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.3±1.4</td>
<td>1.2±1.2</td>
<td>NS</td>
</tr>
<tr>
<td>AHI, per hour of sleep</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.5±3.0</td>
<td>3.6±2.7</td>
<td>NS</td>
</tr>
<tr>
<td>Mean nocturnal SaO2, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>95.7±1.2</td>
<td>96.0±1.3</td>
<td>NS</td>
</tr>
<tr>
<td>Mean low nocturnal SaO2, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>88.9±3.2</td>
<td>88.2±3.8</td>
<td>NS</td>
</tr>
</tbody>
</table>

*NS=not significant.
apparent association between snoring and hypertension was entirely due to confounding by obesity.21,22 Conversely, in other studies, the association between snoring and hypertension persisted after adjustment for body mass index.1,4 Because sleep recordings were not performed in these studies, however, the effect of sleep apnea on the relationship between snoring and hypertension could not be assessed. On the basis of all-night monitoring of snoring and respiratory parameters, Hoffstein et al.25 categorized snorers as apneic and nonapneic and found that snoring was not a direct risk factor for hypertension but was apparently linked to BP via its associations with obesity, obstructive sleep apnea, and nocturnal hypoxemia.

Guilleminault et al.7,8 demonstrated that sleep fragmentation related to abnormal inspiratory effort may occur in patients with nonapneic snoring and could be controlled by nasal continuous positive airway pressure. They called this syndrome the upper airway resistance syndrome. In addition, Guilleminault et al.23 recently reported on six subjects with snoring and upper airway resistance syndrome who had borderline hypertension that was controlled by 1 month of nasal continuous positive airway pressure treatment. These new findings indicated a need for assessing whether sleep fragmentation in snorers was a risk factor for hypertension. To our knowledge, our study is the first to look at the relationship between sleep fragmentation, evaluated by polysomnography, and diurnal arterial hypertension in heavy snorers. We found that the prevalence of systemic hypertension was significantly higher in the sleep-fragmented group, whereas there were no differences for other common risk factors for systemic hypertension, including age, obesity, sex ratio, AH1, and mean sleep oxygen saturation. Consequently, our findings clearly indicate an association between sleep fragmentation and hypertension in snorers without sleep apnea syndrome.

Because all our patients sought medical assistance at an otorhinolaryngology clinic for snoring with social impairment, they cannot be considered representative of the entire population of snorers. Rather, they represent a subpopulation of heavy snorers. The prevalence of sleep fragmentation is probably much lower in the general population of snorers than in our population. The failure of some studies21,22 to detect an independent association between snoring and hypertension may be ascribable to heavy dilution of sleep-fragmented snorers with snorers who did not have arousals or snored only occasionally.

The mechanism by which sleep fragmentation may promote development of diurnal hypertension has not yet been completely elucidated. A recent study showed that nocturnal BP patterns differ in nonapneic heavy snorers compared with nonsnorers.24 Among snorers, BP increased slightly during sleep, whereas the BP of nonsnorers decreased compared with wakefulness values. The increase in BP observed in snorers may be caused by larger intrathoracic pressure swings during snoring periods.25 Studies in patients with obstructive sleep apnea syndrome have convincingly demonstrated that transient elevation in systemic BP occurs with termination of obstructive apnea.26,27 This elevation may be due mainly to arousal28 rather than to resumption of ventilation28 or hypoxia.29 Similarly, normal sleeping subjects exposed to arousal stimuli exhibited transient increases in BP30 and sympathetic activity.10 A reasonable, although unproven, hypothesis is that nocturnal sympathetic activity surges linked to repeated arousals may contribute to the increase in diurnal sympathetic activity observed in patients with sleep apnea syndrome30 and, consequently, to the increase in incidence of arterial hypertension in this population.7 This hypothetical mechanism of hypertension occurrence in sleep apnea syndrome may also explain the increased incidence of hypertension in our population of nonsleep apnea snorers with sleep fragmentation.

In conclusion, our findings provide evidence that sleep fragmentation is frequent in patients who attend an otorhinolaryngology clinic for snoring with social impairment, and may play a part in the development of hypertension. Thus, sleep studies should be considered in the investigation of patients who complain of snoring even in the absence of sleep-related breathing disorders and subjective daytime sleepiness.

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