Nasal Continuous Positive Airway Pressure for Nonapneic Snoring?*

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The feasibility of nasal continuous positive airway pressure (CPAP) for heavy snoring associated with daytime sleepiness was studied in 118 consecutive patients with an apnea hypopnea index below 5. Fifty-nine of them reported daytime sleepiness in a questionnaire and were offered treatment with nasal CPAP. Whereas 48 patients refused it, the remaining 11 (19%) accepted nasal CPAP for home therapy. Acceptors and refusers did not differ in sleep structure, but acceptors had slightly more sleep-disordered breathing events per hour of sleep than refusers. The pressure needed to abolish snoring in these 11 patients was 7.3 ± 1.6 cm H2O. Six months after prescription, the built-in time counters of the patients' devices were read. By dividing the hours of operation by the days since initiation of treatment, we found a mean daily use time of only 2.8 ± 1.5 h. Nevertheless, eight patients (73%) reported that their sleepiness had improved with therapy. We conclude that only a minority of nonapneic snorers accept treatment with nasal CPAP on a long-term basis and that this subgroup is not predictable from polysomnography.

(Chest 1995; 107:58-61)

\[ \text{AHI} = \text{apnea hypopnea index; EDS = excessive daytime sleepiness; CPAP = continuous positive airway pressure; OSA = obstructive sleep apnea; UARS = upper airway resistance syndrome} \]

**Key words:** daytime sleepiness; nasal continuous positive airway pressure—compliance; snoring; upper airway resistance syndrome

**Patients and Methods**

We prospectively studied all patients referred to our sleep laboratory during the course of 6 months. Prior to polysomnography, all of them completed a self-administered questionnaire including 5 questions on daytime sleepiness: (1) Do you feel sleepy during daytime? (2) Do you involuntarily fall asleep when reading, (3) watching TV, (4) driving a car, (5) talking with others? For each question, a rating from 1 to 5 was requested, where 1 stood for never, 2 for seldomly, 3 for sometimes, 4 for frequently, and 5 for always. The sum of these ratings was defined as the EDS score. This EDS score correlates very closely (r=0.82; p<0.0001, Helmut Rauscher, MD, unpublished data) with the Epworth-Sleepiness-Scale in snorers with and without OSA, but has not been validated in patients with nonsnoring-related sleep disorders.

All patients underwent polysomnography including continuous monitoring of EEG, electro-oculography, submental electromyogram, ECG, airflow at nose and mouth (thermistors), movements of rib cage and abdomen (inductance plethysmography or strain gauges), and oxyhemoglobin saturation. Because every sleep study was videotaped, the presence or absence of snoring during the study night was documented. Sleep staging was done according to standard criteria. 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% or less compared with the preceding 5 breaths for longer than 10 s accompanied by a fall in arterial oxygen saturation to 92% or lower if baseline was equal or above 94% or a fall in arterial oxygen saturation of 3% or more if baseline was 93% or lower. The total number of apneas and hypopneas per hour of sleep represented the apnea hypopnea index (AHI). The number of arousals, lasting 3 to 15 s, per hour of sleep, i.e., the arousal index, was determined using the American Sleep Disorders Association criteria. Those patients with an EDS score of 10 or higher and an AHI below 5 were offered treatment with nasal CPAP. On CPAP-titration nights, therapy was started with a pressure of 4-cm H2O that was raised in steps of 1-cm H2O every 10 min until snoring disappeared in all sleep stages and body positions.

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Six months after initiation of therapy, the acceptors of nasal CPAP among those who fulfilled our definition of a sleepy snorer underwent a follow-up polysomnography while receiving nasal CPAP with the pressure set to the level used for home therapy, and the built-in time counter of the patient’s device (REMStar, Respironics) was read by the sleep lab staff. Once again, the patients completed a questionnaire that, aside from the previously mentioned questions on EDS, included two questions on compliance with therapy: How many nights a week do you use nasal CPAP? For how many hours on these nights do you use the machine?

Results
Among 265 consecutive patients in our sleep laboratory, 8 had a diagnosis not related to increased upper airway resistance, like narcolepsy or periodic limb movements and were excluded prior to the analysis. The remaining 257 patients (203 men, 54 women) had been referred for evaluation of suspected sleep apnea. An AHI lower than 5 was found in 118 patients (83 men, 35 women) and 59 of them (=50%) had an EDS score above 10 and were thus offered treatment with nasal CPAP. Whereas 30 of these nonapneic snorers did not even agree to undergo a CPAP titration night and 18 of them refused nasal CPAP after the first trial night, 11 patients accepted this kind of treatment for home therapy. Another 11 patients decided to undergo uvulopalatopharyngoplasty.

The 11 sleepy nonapneic snorers accepting nasal CPAP did not differ from those 48 refusing it in age (50.6±11.7 vs 49.8±10.9) and body mass index (28.4±3.5 vs 29.9±5.6). The sex distribution for acceptors and refusers was 8 male and 3 female subjects in the first group versus 28 male and 20 female subjects in the latter. There was no difference between the two groups in regard to the initial EDS score (13.4±2.6 vs 14.3±3.6), which was not substantially lower than that found in patients with typical OSA. Surprisingly, the rating for the question: “Do you feel sleepy during daytime?” was slightly higher in refusers (3.5±0.9) than in acceptors of CPAP (2.8 ±1.1; p<0.05), but acceptors rated loud snoring a bit higher than refusers (4.8±0.4 vs 3.9±1.4; p<0.05).

The polysomnographic data for the two groups are given in Table 1. As can be seen from these numbers, acceptors had slightly more apneas and hypopneas, but sleep quality appeared to be poorer in refusers. However, sleep continuity measured by the arousal index was the same in both groups.

At follow-up, ie, 6 months after initiation of therapy, the prescribed pressure (7.3±1.6 cm H2O) was still effective in abolishing sleep-disordered breathing events and snoring in all of our 11 patients. There was a significant change in the arousal index (Fig 1) from baseline until follow-up (5±3; p<0.001). Whereas the EDS score remained constant in 3 of our 11 patients, the other 8 (=73%) reported improved daytime vigilance (Fig 2). The mean EDS score at follow-up for the whole group was 9.2±1.9 (p<0.01 compared with baseline), and this change was not

Table 1—Polysomnographic Data of Acceptors and Refusers of Nasal Continuous Positive Airway Pressure Among Nonapneic Snorers With Daytime Sleepiness

<table>
<thead>
<tr>
<th></th>
<th>Acceptors (n=11)</th>
<th>Refusers (n=48)</th>
<th>Probability Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time, h</td>
<td>6.7±0.4</td>
<td>5.9±1</td>
<td>0.019</td>
</tr>
<tr>
<td>Wake after sleep onset, min</td>
<td>53±26</td>
<td>91±62</td>
<td>0.035</td>
</tr>
<tr>
<td>Sleep efficiency, %</td>
<td>87±5</td>
<td>78±13</td>
<td>0.02</td>
</tr>
<tr>
<td>Arousal index</td>
<td>20±10</td>
<td>20±11</td>
<td>NS</td>
</tr>
<tr>
<td>Rapid eye movement sleep, % total sleep time</td>
<td>16±6</td>
<td>16±5</td>
<td>NS</td>
</tr>
<tr>
<td>Slow wave sleep, % total sleep time</td>
<td>11±7</td>
<td>11±8</td>
<td>NS</td>
</tr>
<tr>
<td>Apnea index</td>
<td>2±1.8</td>
<td>0.8±1</td>
<td>0.038</td>
</tr>
<tr>
<td>Apnea hypopnea index</td>
<td>2.5±1.8</td>
<td>1.3±1.3</td>
<td>0.014</td>
</tr>
</tbody>
</table>
attributable to changes in concurrent medication, which consisted solely in antihypertensives in 4 of our patients, or significant weight loss.

Looking at compliance with nasal CPAP, we found that only 4 of our patients (=36%) used their machine for a mean of more than 4 h a night, the mean daily use time for the whole group being 2.8 ± 1.5 h. Self-reported nights of use ranged from 1 to 7, and there was also a wide variation for the reported hours of use per night (1 to 8 h).

To exclude that another sleep pathologic condition was responsible for the failure of CPAP to improve EDS in 5 of our 11 snorers, these subjects underwent a second polysomnography with CPAP on and monitoring of limb movements followed by a multiple sleep latency test to exclude sleep onset-rapid eye movement periods. Neither from history nor from our measurements was any patient found to suffer from periodic limb movements or narcolepsy. It seems worth mentioning that the mean daily use time of nasal CPAP was shorter than 1.5 h in 2 of the 3 nonresponders.

**DISCUSSION**

In 50% of those snorers referred for polysomnography because of suspected OSA, but showing less than 5 sleep-disordered breathing events per hour of sleep, we found substantial complaints about daytime sleepiness. However, impairment by daytime sleepiness was severe enough to accept nasal CPAP therapy in only 19% of them (11 out of 59), and this subgroup was predictable from neither polysomnography nor the reported severity of EDS.

Although our results suggest that daytime sleepiness is a rather frequent finding among heavy snorers, it is not justified to assume that the prevalence of EDS in nonapneic snorers is indeed as high as 50% for at least two reasons: First, our study population consisted of snorers referred for evaluation of suspected sleep apnea. Thus, these subjects had at least some symptoms of OSA, mainly apneas witnessed by the bedpartner or some kind of sleep disturbance, whereas really “asymptomatic” snorers were never seen in the sleep laboratory. Second, our definition of daytime sleepiness based on an EDS score derived from a questionnaire was obviously entirely arbitrary. However, even by performing multiple sleep latency tests in all of our patients, an arbitrary definition of EDS would have been necessary as it is unknown which sleep latency actually indicates impairment of daytime performance by EDS. Furthermore, objectively measured daytime sleepiness in these patients usually is not as severe as, eg, in OSA or narcolepsy, so that the subjective perception of vigilance appears to play a much more important role in nonapneic than in apneic snorers or narcoleptics.

Although acceptors of nasal CPAP had slightly more sleep-disordered breathing events than refusers during their initial sleep study, the severity of sleep fragmentation, ie, the arousal index, was the same in both groups. Nevertheless, acceptors slept longer and had better sleep efficiency, which may be interpreted as an indirect sign of a more pronounced sleep deprivation, probably indicating a longer lasting snoring-related sleep disturbance than in refusers. According to this hypothesis, those accepting nasal CPAP would have been in a later stage of the development from simple snoring to sleep-disordered breathing than those refusing it. Clinically, this could mean that complaints about poor sleep in snorers may be the first sign of ongoing sleep-disordered breathing. Because poor sleep may lead to the intake of sedatives or hypnotics, a drug-induced further increase in upper airway resistance is likely to worsen the situation.

Our study shows that neither polysomnographic nor clinical features can be used to determine those nonapneic snorers who will accept nasal CPAP and who will benefit from CPAP therapy in the long-term. On the other hand, EDS in the majority of nonapneic snorers can be successfully treated with nasal CPAP. Thus, it appears reasonable to perform a CPAP trial in all snorers complaining about EDS, irrespective of the results from polysomnography. In case of improvement of EDS, the patient should be considered for aggressive treatment, ie, maintenance of nasal CPAP or eventually upper airway surgery. Although acceptance of nasal CPAP in this group of patients is rather poor, the patient’s willingness to have some kind of treatment seems to be astonish-
ingly high in nonapneic snorers, evidenced by the fact that aside from the 11 patients accepting nasal CPAP, another 11 snorers decided to undergo surgery, *i.e.*, uvulopalatopharyngoplasty with or without correction of the nasal airway. Thus, a total of 22 of our 59 patients (37%) wanted to have vigorous treatment for their condition. Unfortunately, we lack a sufficient number of follow-up investigations in those who had surgical treatment, so that we are unable to compare the effects of nasal CPAP and surgery on EDS in this group of patients.

It is surprising that the short mean time of daily CPAP use was sufficient to improve EDS in 8 of our 11 subjects. One reason for this may be that these subjects suffered from relatively mild EDS that eventually did not recur after one or two nights without CPAP. This may have led them to an on-demand kind of treatment that resulted in such low numbers for the machine's hours of operation. Because it is not even known how much daily CPAP is enough to counteract EDS in patients with OSA, it is very difficult to objectively judge compliance with therapy in patients with the UARS, since their EDS is usually milder than that in OSA patients. Thus, the "poor" compliance we found in this study does not justify withholding CPAP in snorers without significant OSA. Rather, the more than 70% success rate should be an indication to consider nasal CPAP also for sleepy snorers with less than five sleep-disordered breathing events per hour.

**References**

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