Mouth Breathing Compromises Adherence to Nasal Continuous Positive Airway Pressure Therapy*

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Study objectives: Mouth leak compromises nasal continuous positive airway pressure (CPAP) therapy. We hypothesized that patients who breathe mainly through their mouths during sleep, compared to those who breathe mainly through their noses, would have more mouth leak during CPAP and therefore lower adherence to CPAP.

Design: A case-control study to compare adherence to CPAP at 1 year in mouth breathers (MBs) with nose breathers (NBs).

Setting: University teaching hospital with a sleep laboratory.

Patients: Fifty-one CPAP-naive patients (4 women), with a respiratory disturbance index (RDI) > 15/h. Of the 51 patients, 30 patients breathed through their mouths (mouth breathing > 70% of total sleep time [TST]), and 21 patients breathed through their noses (mouth breathing < 30% of TST). MBs between 30% and 70% of TST were excluded.

Interventions: Overnight polysomnography was performed at baseline, during CPAP titration, and at 3 months. Patients were followed up for 1 year after beginning CPAP.

Measurements and results: To measure mouth breathing, nasal and oral thermistors during polysomnography were separated by a 3 × 6-cm silicon transverse diaphragm. RDI decreased from (mean ± SD) 37.8 ± 21.5 to 1.8 ± 2.6/h at 3 months. Throughout the study, adherence to CPAP (mean daily CPAP use in hours) was better in NBs. Most NBs (71%) but only 30% of MBs used CPAP daily for > 4 h at 1-year follow-up. Mouth breathing decreased significantly from 84 ± 8.9% at baseline to 22 ± 14.4% at 3 months.

Conclusion: Patients with moderate-to-severe sleep-disordered breathing and a high percentage of mouth breathing during sleep were less adherent to CPAP therapy than patients exhibiting a low percentage of mouth breathing.

Key words: adherence; continuous positive airway pressure; mouth breathing; mouth leak; obstructive sleep apnea; polysomnography

Abbreviations: AUDIT = Alcohol Use Disorders Identification Test; BMI = body mass index; CPAP = continuous positive airway pressure; ESS = Epworth sleepiness scale; MB = mouth breather; NB = nose breather; NC = neck circumference; ODI4 = oxygen desaturation index 4%; RDI = respiratory disturbance index; REM = rapid eye movement; SDB = sleep-disordered breathing; SpO2 = pulse oxygen saturation; TST = total sleep time; WASO = wakefulness after sleep onset

Continuous positive airway pressure (CPAP) is an effective treatment for sleep-disordered breathing (SDB). Adherence to CPAP therapy in the community has shown rates ranging from 65 to 88%. Despite numerous improvements in the technology of CPAP devices, however, the major challenge to physicians is to increase acceptance of and adherence to this treatment.

Most studies have tried to predict adherence to CPAP based on the response during the early period of use, or on the severity of the disease. Meanwhile, identifying a risk group before starting CPAP would be economical and may justify the early use of autotitrating CPAP, chin strap, full face mask, oral mask, and heated humidification, in addition to basic educational and behavioral support.

Mouth opening increases upper-airway collapsibility during sleep and may contribute to the occurrence of SDB. Mouth leak with CPAP does occur in 10 to 15% of cases, and may compromise CPAP.
To predict this leak, we hypothesized that patients who breathe mainly through their mouths during sleep would have more mouth leak during CPAP and therefore lower adherence to CPAP. We therefore quantified mouth breathing during sleep in patients with moderate-to-severe SDB and followed their subsequent adherence to CPAP therapy.

**Materials and Methods**

**Patients**

From 231 consecutive CPAP-naive patients referred for snoring and a variable degree of daytime sleepiness, 119 patients were excluded because their respiratory disturbance index (RDI) was < 15/h. We also excluded 19 patients: 3 patients with a stroke < 1 year previously, 2 patients with severe psychiatric illness, 5 patients with a prior uvulopalatopharyngoplasty, 3 patients with persistent nasal symptoms, 1 patient who refused CPAP, and 5 patients to whom nasal surgical intervention was proposed. Patients with RDI > 15/h were checked by an otolaryngologist. A decision for nasal surgical intervention (septoplasty, chryotherapy, or radiofrequency thermoablation) was based on the presence of nasal symptoms, abnormal nasal structure, and abnormal rhinomanometry findings.

Quantification of mouth breathing during sleep was done during the baseline polysomnography (see below). We excluded those 42 patients whose percentage of mouth breathing fell between 30% and 70% of total sleep time (TST).

We studied 51 patients without nasal symptoms: 30 patients were considered mouth breathers (MBs) with mouth breathing > 70% of TST, and 21 patients were nose breathers (NBs) with mouth breathing < 30% of TST. All subjects gave their written informed consent. The Research and Ethics Committee of the Department of Internal Medicine at our hospital approved the protocol.

**Protocol**

Polysonmography was performed at baseline (diagnostic study), on the first night of CPAP titration, on the second night of CPAP, and at 3 months. The mean daily CPAP use in hours was calculated by a nurse who was blinded to the patient’s classification, using the following formula: ([No. of the built-in hour meter at 1 month, at 3 months, or at the last follow-up visit] − [No. of the hour meter at the beginning of CPAP])/time elapsed in days. The Epworth sleepiness scale (ESS) and the Alcohol Use Disorders Identification Test (AUDIT) values were obtained at baseline and during follow-up.

Recording of polysomnography and scoring of sleep and respiratory events were performed as previously described.

Nasal airflow was monitored with a thermistor (model No. 6240; Healthdyne Technologies; Marietta, GA) placed near the patient’s nostrils. Mouth airflow was monitored using a thermistor placed in front of the mouth. A 3 × 6-cm silicon transverse diaphragm was fixed at the nasal thermistor to prevent activation of the mouth thermistor during nose breathing (Fig 1). During CPAP, a pneumotachometer (Hans Rudolph; Kansas City, MO) was attached between the nasal mask and the CPAP generator (Fig 2). A videorecording of the patient’s face aided in viewing the opening of the mouth and the placement of thermistors.

**Mouth Breathing**

During polysomnographic calibration, patients were asked to breathe normally and exclusively through the nose and then...
through the mouth, while a specially trained nurse verified that each thermistor was activated exclusively. In polysomnographies with CPAP, the same maneuver for calibration of mouth breathing was done while nasal CPAP was on. The pneumotachometer measures the difference in pressure between its two pressure sensors and calculates the corresponding airflow according to an initial calibration. A drop in the difference in pressure means a drop in airflow. In our system, flow will be always positive when CPAP is on, but will vary with breathing. A mouth-breathing event was scored visually when at least one deviation from baseline was > 10% of the calibrated signal. This threshold of 10% is usually considered to fall within the normal range of thermistor recording variability. A mouth-breathing episode, scored over a period of 30 s, included one or more mouth-breathing events (Fig 3), ie, we considered the whole 30-s epoch as a mouth-breathing episode even if it contained one or more mouth breaths of > 10% of the calibrated signal. Only events during epochs scored as sleep were considered. No distinction was made between mouth breathing and mouth opening at the end of apnea caused by choking. Mouth breathing as a percentage of TST was calculated as follows: total mouth-breathing episodes × 0.5 × 100/TST (minutes). We meticulously checked the thermistors during the recording to avoid failure. As there is no known cutoff point between mouth and nose breathing, we considered patients with < 30% of TST of mouth breathing as NBs and > 70% of TST as MBs.

**CPAP Titration**

Patients underwent a session of familiarization with the CPAP device and mask. On the initial titration night, pressure was increased manually to correct respiratory events and flow limitation. The next day, the second CPAP night, a fixed pressure, which was determined on the first night, validated the pressure level. All patients used the same nasal interface (Ultra Mirage mask; ResMed; Sydney, Australia) and the same CPAP pump that contains a built-in hour meter (Sullivan Plus; ResMed). No full face, oral mask, chin strap, or humidity device was used.

**Data Analysis**

We considered as significant any difference of 1.5 h of CPAP daily use between MBs and NBs at 3 months. A Student t test and a χ² test served for numerical and categorical variables respectively, and a Spearman rank correlation for correlation analysis. We used a commercial statistical package (Statistica v.5; StatSoft; Tulsa, OK); p < 0.05 was considered significant.

**RESULTS**

**Baseline Demographics and Sleep Characteristics**

We studied 51 patients (4 women). MBs and NBs did not differ with respect to age, sex, body mass index (BMI), neck circumference (NC), ESS, AUDIT score, TST, TST while supine, rapid eye movement (REM) sleep, wakefulness after sleep onset (WASO), sleep efficiency, RDI while supine, or pulse oxygen saturation (SpO₂) awake. Meanwhile,
total arousal, respiratory arousal, RDI, and an oxygen desaturation index of 4% (ODI<sub>4</sub>) were higher in MBs, but the amount of delta sleep was lower. By design, the percentage of mouth breathing by MBs was higher than by NBs. Exact figures are presented in Table 1.

**CPAP Pressure and Adherence**

The prescribed CPAP pressure to MBs ranged from 6 to 15 cm H<sub>2</sub>O and to NBs from 6 to 14 cm H<sub>2</sub>O, Table 2. Four MBs (one woman) refused to use CPAP at home immediately after the titration, but are included in analyses. Their mean ± SD age was 45.7 ± 7.4 years; BMI, 35.9 ± 7.1; RDI, 50.2 ± 33.7/h; and ESS, 7.8 ± 5.1; and two of them had AUDIT scores >10. No NBs refused CPAP after the titration. Total follow-up period for CPAP use was 51.6 ± 42.5 weeks for all patients.

Throughout the study, adherence to CPAP was significantly better (p < 0.05) in NBs (Table 2). Of 30 MBs, 11 patients (37%) received CPAP for a mean of >4 h per night at 3 months, as did 9 MBs (30%) at the last follow-up visit, whereas 16 NBs (76%) received CPAP for >4 h per night at 3 months, as did 15 NBs (71%) at the last follow-up visit.

No significant difference existed in residual (receiving CPAP therapy) RDI or ODI<sub>4</sub> or in arousal index between MBs and NBs at the second CPAP titration night or the 3-month assessment (Table 2). We found no significant correlation between use of CPAP at 3 months and patient age, BMI, NC, ESS, AUDIT score, or with any sleep or respiratory parameter at baseline, neither between mouth breathing and RDI. Nevertheless, we found a significant correlation (R = 0.87, p < 0.001) for all patients between use of CPAP at 1 month and at 3 months or at the last follow-up visit.

**Effect of Nasal CPAP on Mouth Breathing**

In MBs receiving CPAP, mouth breathing decreased significantly (p < 0.001), from 84.5 ± 8.9% at baseline to 30.0 ± 18.9% and to 21.4 ± 17.5% of TST on the second titration night and at 3 months, respectively. In addition, in NBs, mouth breathing decreased significantly (p < 0.05), from 19.9 ± 7.8% at baseline to 7.7 ± 7.7% and to 11.1 ± 13.5% of TST on the second titration night and at 3 months, respectively. Mouth breathing remained, however, significantly higher (p < 0.05) in MBs than in NBs at the two follow-up time points (Fig 4).

**Effect of Nasal CPAP on Sleep**

In MBs and NBs, TST and sleep efficiency decreased significantly (p < 0.05) during the first titration night compared to baseline. Meanwhile, the amount of delta sleep increased significantly (p < 0.05) on the second titration night and at 3 months vs baseline. REM and sleep efficiency increased significantly (p < 0.05) on the second compared to the first titration night, whereas WASO decreased significantly (p < 0.05). No significant differences in sleep or respiratory parameters occurred between the second titration night and 3

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### Table 1—Baseline Demographics and Sleep Characteristics of MBs and NBs*

<table>
<thead>
<tr>
<th>Variables</th>
<th>MBs (n = 31) (Three Women)</th>
<th>NBs (n = 21) (One Woman)</th>
</tr>
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<tbody>
<tr>
<td>Age, yr</td>
<td>51.3 ± 1.9</td>
<td>51.4 ± 2.3</td>
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<tr>
<td>BMI</td>
<td>31.7 ± 1.1</td>
<td>30.8 ± 1.0</td>
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<tr>
<td>NC, cm</td>
<td>42.9 ± 0.6</td>
<td>42.2 ± 0.6</td>
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<tr>
<td>ESS (scale 0–24)</td>
<td>7.7 ± 1.0</td>
<td>7.9 ± 0.8</td>
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<tr>
<td>AUDIT (scale 0–40)</td>
<td>5.3 ± 0.8</td>
<td>6.9 ± 1.1</td>
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<tr>
<td>TST, min</td>
<td>363 ± 15.1</td>
<td>362 ± 15.8</td>
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<tr>
<td>TST supine, min</td>
<td>168 ± 25.2</td>
<td>135 ± 19.7</td>
</tr>
<tr>
<td>REM sleep, min</td>
<td>63.0 ± 6.7</td>
<td>65.0 ± 7.6</td>
</tr>
<tr>
<td>Delta sleep, min</td>
<td>26.3 ± 4.9</td>
<td>48.0 ± 4.9†</td>
</tr>
<tr>
<td>WASO, min</td>
<td>54.8 ± 8.4</td>
<td>67.2 ± 8.6</td>
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<tr>
<td>Sleep efficiency, %†</td>
<td>77.0 ± 2.4</td>
<td>75.7 ± 2.8</td>
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<tr>
<td>Total arousals, /h</td>
<td>58.4 ± 4.6</td>
<td>38.2 ± 3.1†</td>
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<tr>
<td>Respiratory arousals, /h</td>
<td>49.9 ± 4.7</td>
<td>32.2 ± 2.9†</td>
</tr>
<tr>
<td>RDI, /h</td>
<td>44.8 ± 4.5</td>
<td>28.7 ± 2.7‡</td>
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<tr>
<td>RDI supine, /h</td>
<td>52.4 ± 4.3</td>
<td>53.0 ± 4.3</td>
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<tr>
<td>ODI&lt;sub&gt;4&lt;/sub&gt;, /h</td>
<td>49.4 ± 4.8</td>
<td>26.5 ± 3.5§</td>
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<tr>
<td>SpO₂ awake, %</td>
<td>95.2 ± 0.3</td>
<td>95.3 ± 0.2</td>
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<tr>
<td>Mouth breathing, % of TST</td>
<td>84.8 ± 1.6</td>
<td>19.9 ± 1.7</td>
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*Data are presented as mean ± SD.
†TST × 100/time in bed.
‖p < 0.005.
§p < 0.01.
<table>
<thead>
<tr>
<th>Variables</th>
<th>MBs (n = 31) (Three Women)</th>
<th>NBs (n = 21) (One Woman)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Use, h/d</td>
<td></td>
<td></td>
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<tr>
<td>Month 0–1</td>
<td>3.00 ± 0.50</td>
<td>4.45 ± 0.56†</td>
</tr>
<tr>
<td>Month 0–3</td>
<td>2.99 ± 0.50</td>
<td>4.51 ± 0.51†</td>
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<td>Month 0 to last follow-up</td>
<td>2.87 ± 0.52</td>
<td>4.22 ± 0.52†</td>
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<td>Pressure, cm H&lt;sub&gt;2&lt;/sub&gt;O</td>
<td>9.08 ± 0.45</td>
<td>9.90 ± 0.45</td>
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<td>Residual, RDI/h</td>
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<td></td>
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<tr>
<td>Second titration night</td>
<td>2.2 ± 0.71</td>
<td>1.0 ± 0.31</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>2.2 ± 0.74</td>
<td>1.4 ± 0.37</td>
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<tr>
<td>Residual, ODI&lt;sub&gt;4&lt;/sub&gt;/h</td>
<td></td>
<td></td>
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<tr>
<td>Second titration night</td>
<td>4.5 ± 1.21</td>
<td>4.7 ± 1.71</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>2.9 ± 0.83</td>
<td>4.0 ± 1.43</td>
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<tr>
<td>Residual, arousals/h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second titration night</td>
<td>11.3 ± 0.94</td>
<td>11.9 ± 1.11</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>11.9 ± 1.04</td>
<td>10.7 ± 0.88</td>
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*Data are presented as mean ± SD. p < 0.05.
months (Table 3). At 3 months of CPAP therapy, we found a significant (p < 0.01) reduction in ESS in both MBs and NBs, from 7.9 ± 5.2 to 5.7 ± 4.2 and from 7.9 ± 3.5 to 4.9 ± 2.7, but no significant changes occurred between baseline, CPAP titration, and 3-month follow-up in BMI, NC, or AUDIT scores.

**Discussion**

The main finding of this study is that in patients with SDB, a high percentage of mouth breathing during sleep represents a risk for low adherence to CPAP. Furthermore, although mouth breathing decreases considerably when patients are put on nasal CPAP, MBs still have considerably more mouth breathing on CPAP than do NBs. We also found a significant correlation in all patients between use of CPAP at 1 month and its long-term use. These findings are consistent with others, that mouth leak may compromise CPAP therapy, and that the first period of CPAP use predicts long-term adherence.

Several limitations of this study may limit the generalizability of our results. We are a university sleep center receiving patients with SDB referred from local health-care centers. In addition, no patients with mild SDB were included, as the benefits of CPAP treatment for such patients are less clear. Our patients did not receive heated humidity or a full face mask, and 94% of them were male subjects. Our findings may not apply to women or apply to the use of humidity. Moreover, we did not use a chin strap to reduce mouth leak with CPAP, as our recent study showed the inefficacy of this method. In addition, thermistors used for monitoring nasal and oral airflow at baseline provide measurements more qualitative than quantitative. Finally we agree that the low ESS scores of our patients may have reduced their adherence to CPAP, as has been suggested.

When scoring mouth breathing, we did not distinguish mouth opening at the end of apnea caused by choking and oral breathing. As choking is more frequent in severe SDB, it is possible to explain the higher RDI in our MBs. Nevertheless, we could not consider mouth breathing as a marker of SDB severity, because no significant correlation appeared between RDI and mouth breathing. In a recent review, Rappai et al postulated that the switch to oronasal breathing that occurs with chronic nasal conditions is a final common pathway for SDB.

Our study showed that adherence to CPAP was significantly lower in MBs than in NBs. We postulate that MBs have more difficulties in accepting a new breathing pattern, one exclusively nasal, than do

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Figure 4. Mouth breathing at baseline, at CPAP titration, and at 3 months. **Upper panel:** Box and whisker plot (median, quartiles, range) representing mouth breathing at baseline, CPAP titration, and at 3 months. **Lower panel:** Individual evolution of mouth breathing. At the two follow-up time-points there was a significant (p < 0.05) decrease in mouth breathing and a significant (p < 0.05) difference between MBs and NBs.
NBs, resulting in more mouth leak with CPAP. Furthermore, our four patients who refused CPAP after titration were all MBs. In this study, because sleep and respiratory parameters were significantly better on CPAP for all patients, the lower adherence of MBs cannot be attributed to CPAP inefficiency, nor to the difference in patient age or gender, as has been suggested.2

The real cause of mouth breathing in patients with SDB is not fully known. We excluded patients with nasal problems, and thus may assume that mouth breathing, at the time of the examination, was unrelated to nasal obstruction. Nevertheless, one recent theory is that mouth breathing is related to SDB,25 and that medical treatment of rhinitis alleviates SDB.26

Our method for detecting mouth breathing requires a special setting not commercially available. An “easy-to-use” setting to separate mouth from nasal airflow needs to be developed. Nasal prongs, widely used and relatively well documented,27 mainly measure nasal airflow; however, no validated study has analyzed the shape of the nasal pressure curve in mouth breathing.

We recommend the detection of mouth breathing during sleep, not only for predicting adherence to CPAP therapy, but also for avoiding the use of CPAP titration devices that use nasal mask pressure-vibration detection as their only mode of pressure setting; such devices fail to recognize all the respiratory events in patients with significant mouth breathing.28 Whether mouth breathing predicts nasopharyngeal anatomic obstruction and, in turn, predisposes to more severe SDB needs further investigations.

We conclude that patients with moderate-to-severe SDB with a high percentage of mouth breathing during sleep would probably adhere less to nasal CPAP therapy than would patients with a low percentage of mouth breathing during sleep. Our finding is clinically important, and may be of considerable economic benefit, because it may predict the likely success of CPAP before its initiation.

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