The acute impact of continuous positive airway pressure on nasal resistance: a randomized controlled comparison

Stephanie Willing, Maybelle San Pedro, Helen S. Driver, Peter Munt, and Michael F. Fitzpatrick

Department of Medicine, Queen’s University, Kingston, Ontario, Canada

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Willing S, San Pedro M, Driver HS, Munt P, Fitzpatrick MF. The acute impact of continuous positive airway pressure on nasal resistance: a randomized controlled comparison. J Appl Physiol 102: 1214–1219, 2007. First published December 7, 2006; doi:10.1152/japplphysiol.00639.2006.—Subjective nasal obstruction is common among users of continuous positive airway pressure (CPAP). The aim of this study was to measure the acute effect of CPAP on nasal resistance and nasal symptoms in awake normal subjects. Twenty-four healthy CPAP-naive adults (8 men, 16 women; mean age 30 yr (SD 14)) underwent a randomized controlled crossover study comparing nasal CPAP (8 cmH2O) for 6 h on one occasion and the control condition (nasal mask without CPAP) on the other. Nasal resistance measurements (posterior active rhinometry) before and after the test exposure were similar on both test days. Nasal resistance: a randomized controlled comparison. J Appl Physiol 102: 1214–1219, 2007. First published December 7, 2006; doi:10.1152/japplphysiol.00639.2006.

MATERIALS AND METHODS

Subjects. Twenty-nine healthy CPAP-naive subjects (12 men, 17 women) were recruited to the study through newspaper and campus advertisements. Subjects were screened by telephone interview to exclude those taking medications that could alter nasal resistance (antihistamines, vasoconstrictors, vasodilators, topical or systemic steroids, and recreational drugs), smokers, and individuals with symptomatic nasal obstruction. Before recruitment to the study, all subjects underwent measurement of nasal resistance by posterior active rhinometry (14) to verify that the nasal resistance was within normal limits. Individuals with a history of allergic rhinitis were included if they were free of nasal symptoms, were outside their allergy season, and had normal baseline nasal resistance.

The subjects were aged 29 ± 3 yr (means ± SE) and had a body mass index of 25.5 ± 1 kg/m². Nasal resistance (see below) during the screening measurement for the group while seated erect was 2.8 cmH2O·1−1·s (SD 0.4) and while supine was 3.3 cmH2O·1−1·s (SD 0.4).

Study design. This was a prospective randomized, controlled, crossover study of the effect nasal CPAP on nasal resistance. CPAP was delivered at 8-cmH2O mask pressure, a pressure chosen to reflect usual daily CPAP exposure in a compliant patient. CPAP was applied without added humidification. The CPAP-patient interface was a nasal mask, carefully fitted to ensure that there was no external nasal compression or mask leak as previously described (14). The control arrangement was an identical nasal mask that was customized by removal of the sides of the mask to minimize dead space. CPAP was not applied in the control condition; specifically, the authors were concerned that inspired air temperature and humidity with subtherapeutic CPAP might be similar to that of therapeutic CPAP, and it would not be a true control. Because subjects with normal nasal resistance breathe almost exclusively through the nose during wakefulness and sleep (14), and the presence of a mouth leak has been clearly demonstrated to increase nasal resistance (31), the confounding effect of mouth breathing on nasal resistance was prohibited.

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) is the most commonly used treatment for patients with obstructive sleep apnea syndrome (OSAS), and its use has been associated with substantial improvements in disease-specific quality of life (15), somnolence (12), cognitive performance (13), and sleep quality (19) among patients with obstructive sleep apnea (OSA). Recent studies suggest that CPAP use may modify cardiovascular risk factors: blood pressure control in hypertensive patients (34), increased insulin sensitivity among diabetic patients (16), and improved secondary prevention against cardiovascular outcomes (26) in patients with OSA. CPAP compliance remains a major clinical challenge, and nasal symptoms are among the most common side effects encountered among users of CPAP treatment for obstructive sleep apnoea (OSA); such symptoms may appear within a few hours of CPAP exposure, causing the mask to be removed (6, 28).

However, nasal symptoms and nasal obstruction are already highly prevalent among individuals with OSA even in the absence of CPAP use (21, 38). This begs the question of what independent impact CPAP has on nasal resistance. To evaluate the effect of CPAP on the nasal airway, it becomes important to dissociate it from premorbid nasal obstruction and from changes in coexisting upper airway edema that may occur during sleep in patients with OSA (33). There has been little systematic study of the effect of CPAP on nasal physiology (30). The aim of the present study, therefore, was to evaluate the acute effect of CPAP on nasal resistance in normal subjects. Based on subjective reports of increased nasal symptomatology after initiation of nasal CPAP treatment, we hypothesized that the irritant effect of temperature and humidity differences between CPAP airflow and in vivo nasal conditions would lead to nasal mucosal inflammation and an increase in nasal resistance after application of CPAP.
during the study. An oral thermistor was placed across the lips to detect mouth breathing continuously throughout the study.

Posture has an important effect on nasal resistance (nasal resistance increases with recumbency) (32). To avoid this potential confounder, subjects were asked to adopt a standard posture (recumbency at 30° elevation) throughout the study on both days.

Subjects were asked to have breakfast before coming to the laboratory on each test day, and they were required to fast for the duration of the study on each test day. The washout period between the two study dates was a minimum of 48 h and a maximum of 2 wk.

**Nasal resistance measurement.** Nasal resistance was measured by posterior active rhinomanometry. An infant nasogastric feeding catheter (6-Fr diameter; MED-Rx Benlan, Oakville, ON, Canada) was lubricated and inserted through the right nostril until the distal tip of the catheter was 8 cm from the anterior naris. No local anesthetic or decongestant was given. The proximal end of the cannula was attached to a differential pressure transducer (Ultima Dual Pressure sensor model 0585; Braebon Medical, Kanata, ON, Canada) that was calibrated to ±4 V (= ±20 cmH2O). A CPAP mask was placed over the patient’s nose, taking care to ensure that there was no compression of the nasal airway by the mask (by monitoring the posterior nasal pressure before and after attachment of the nasal mask), and ensuring that there was no air leak from the mask. A heated pneumotach (3700 series, Hans Rudolph, Kansas City, MO) was placed at the outlet of the CPAP mask, and the patient was instructed to breathe quietly through the nose only, with the lips closed. An identical catheter to that used for measurement of posterior nasal pressure was employed to measure pressure at the anterior nares (this provided the reference pressure for calculation of the differential pressure across the nasal airway); this tube was passed through a port in the CPAP mask; the port was then made air tight using adhesive, and the proximal end of the catheter was attached to the differential pressure transducer. Each pneumotach was calibrated with a 3-liter syringe to an accuracy of ±0.5% before each study. Nasal resistance was measured as the change in pressure (cmH2O) across the nose for a standardized inspiratory flow rate of 0.3 l/s.

On each study date, the subject arrived at the sleep laboratory at 8 AM. Baseline pretreatment nasal resistance was measured in the erect and supine positions by posterior active rhinometry (14), and the posterior nasal cannula was left in place to permit further nasal resistance measurements throughout the day. After a 1-h baseline period, nasal CPAP or the control condition was applied for 6 h. During the CPAP condition, a pneumotach was placed in the outlet port of the nasal mask, and a connector with an exhaust port was placed between the pneumotach and CPAP tubing to facilitate carbon dioxide removal. This arrangement facilitated breath-by-breath measurement of the subject’s airflow while on CPAP, while catheters in the nasal mask and posterior nares permitted continuous measurement of the pressure differential between the CPAP mask and posterior nares. On the control study day, the setup was identical except for the customization of the mask to prevent carbon dioxide rebreathing. The absence of sides on the CPAP mask precluded continuous measurement of nasal airflow. For that reason, the mask was replaced by an intact CPAP mask with attached pneumotach for 5 min every hour during the control study day to permit hourly measurements of nasal resistance by posterior active rhinometry. Although subjects underwent simultaneous polysomnography (electroencephalogram, electrooculogram, electromyogram) to permit determination of the sleep-wake state, they were not prohibited from sleeping because it is already documented that the sleep-wake state does not alter nasal resistance (18, 25). End-tidal CO2 testing was conducted in a pilot study of the customized mask used on the non-CPAP day to ensure that the end-tidal CO2 was similar (within 2 Torr) to that while on CPAP.

After 6 h of CPAP exposure or the control condition, the mask was removed, and hourly measurements of nasal resistance were made over the succeeding 2-h period (to look for a delayed effect of CPAP exposure on nasal resistance) before the end of the experiment. Thus each subject underwent 9 h of recording on each of the two occasions: 1 h baseline, 6 h of exposure to CPAP or the control condition, and 2 h of postexposure monitoring.

**Subjective assessment.** On each test day before and after the (6 h) test exposure period, subjects were administered a short questionnaire (9) consisting of two questions indicating the degree of subjective nasal congestion. Table 1 displays the questionnaire used.

**Atopy and/or allergic rhinitis.** A score ≥7 on a validated questionnaire, the Score For Allergic Rhinitis (SFAR) (1), was used to determine the presence or absence of allergic rhinitis. Skin testing to a battery of common allergens was used to determine the presence or absence of atopy.

**Ethics.** This study was approved by the Research Ethics Board of Queen’s University (Kingston, Ontario, Canada).

**Data analysis.** The nasal resistance data were divided into three time periods on each of the two study dates: preexposure, exposure, and postexposure. The nasal resistance values during each of the three time periods were averaged for statistical analysis. The data were then analyzed to look for crossover, period, and order effects. A mixed-models approach, (which incorporated intersubject variability in the nasal resistance response to CPAP in the model) was used to compare nasal resistance values between the two study dates.

Table 1. CPAP and nasal obstruction study: subjective assessment of symptoms

<table>
<thead>
<tr>
<th>Question 1.</th>
<th>0 = absent symptoms (no sign/symptom evident)</th>
<th>1 = mild symptoms (sign/symptom clearly present, but minimal awareness; easily tolerated)</th>
<th>2 = moderate symptoms (definite awareness of sign/symptom that is bothersome but tolerable)</th>
<th>3 = severe symptoms (sign/symptom that is hard to tolerate, causes interference with activities of daily living and/or sleeping)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Please indicate which best describes how you feel:</td>
<td>Clear = fully open, no obstruction of passage</td>
<td>Slight block = barely noticeable blockage of air passage</td>
<td>Stuff = noticeable partial blockage, but not bothersome</td>
<td>Very stuffy = noticeable partial blockage and bothersome</td>
</tr>
<tr>
<td>Blocked = cannot move any air through nostril</td>
<td></td>
<td></td>
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</table>

CPAP, continuous positive airway pressure.

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Wilcoxon signed-ranks testing with Bonferroni correction for multiple comparisons, and Friedman’s test was used to compare subjective responses.

RESULTS

Twenty-four subjects (Table 2) completed the study. Testing on the remaining five subjects (3 men and 2 women) could not be completed because of scheduling problems (2 subjects), difficulty with mask fitting and mask leaks (1 subject), and high baseline nasal resistance on the test day (despite prior normal nasal resistance at screening; 2 subjects). The remaining 24 subjects completed both test days.

The median score on the SFAR was 1.5 (range 0–9). Four subjects had allergic rhinitis as defined by an SFAR score ≥7.

Figure 1 displays the nasal resistance (means ± SE) against time for all subjects on the CPAP exposure and control (No CPAP) days. Nasal resistance baseline measurements (before application of CPAP or no CPAP) were similar [CPAP baseline: 2.42 cmH₂O·l⁻¹·s (SD 0.93); No CPAP baseline: 2.24 cmH₂O·l⁻¹·s (SD 0.83), P = 0.37] on the 2 test days. Nasal resistance during the period of CPAP exposure [2.04 cmH₂O·l⁻¹·s (SD 0.72)] was significantly lower than during the matched time period on the control (No CPAP) day [2.67 cmH₂O·l⁻¹·s (SD 1.07)]; the mean difference in nasal resistance during the matching time periods on both days was 0.66 cmH₂O·l⁻¹·s (SD 1.1) (95% confidence interval 0.19–1.13 cmH₂O·l⁻¹·s; P = 0.0079). The postexposure nasal resistance values were similar [post-CPAP nasal resistance 2.14 cmH₂O·l⁻¹·s (SD 0.61); post-No CPAP nasal resistance 2.48 cmH₂O·l⁻¹·s (SD 0.90); P = 0.14] between the 2 test days. Mean nasal resistance over the 8-h period from initiation of exposure to 2-h postexposure was lower on the CPAP day than on the No CPAP day (P = 0.002).

Table 3 describes the within-day change in nasal resistance between the three phases of the study (preexposure, exposure to CPAP or control, and postexposure). There was a tendency for nasal resistance to fall on the CPAP day and to increase on the No CPAP day, but ANOVA (CPAP day, P = 0.52; No CPAP day, P = 0.4) demonstrates no statistically significant difference between time points on either day.

The pressure gradient between the CPAP mask and the posterior nares paralleled the nasal resistance change on CPAP, but it remained between 1.6 and 2 cmH₂O at all times (Fig. 2). There was no significant difference between the hourly measurements of the mask to posterior naris pressure gradient during the 6-h CPAP exposure (P = 0.25).

Data analysis revealed no significant order or period effects for the effects of CPAP or No CPAP on nasal resistance.

Peak nasal inspiratory flow rates were not different between the four test occasions (pre- and postexposure on each of the 2 test days) (Table 4).

Ambient experimental conditions. Ambient temperature was similar on the CPAP exposure (21.6 ± 0.3°C) and no CPAP (21.5 ± 0.5°C; P = 0.46) days, as was relative humidity (CPAP exposure day 40.2 ± 1.4%; no CPAP day 42.6 ± 1.9%; P = 0.35).

Subjective scores (Table 5). Although the subjective nasal symptom score tended to be slightly greater on the CPAP test day, this difference did not meet statistical significance (P = 0.07; Table 4). The change in subjective nasal congestion from pre- to posttesting was similar on the CPAP (median 0, range −1 to 2) and No CPAP (median 0, range −1 to 1) test days (P = 0.10).

DISCUSSION

This study demonstrates that CPAP exposure for 6 h is associated with a reduction in nasal resistance compared with the control condition, and with no significant change in subjective nasal symptoms. The study finding is contrary to our hypothesis that CPAP would be associated with physical irritation of the turbinate mucosa and a consequent progressive

| Table 2. Demographic and nasal resistance data on subjects completing the study |
|-----------------------------------|------------------|
| No. of subjects                   | 24               |
| Male/female ratio                 | 8/16             |
| Age, yr                           | 30 (14)          |
| Body mass Index, kg/m²            | 25 (4)           |
| Baseline Nasal resistance, cmH₂O·l⁻¹·s | 2.5 (4)       |
| CPAP day                          | 2.4 (0.9)        |
| No CPAP day                       | 2.2 (0.8)        |

Values are means (SD).

<table>
<thead>
<tr>
<th>Table 3. Changes in nasal resistance during the three phases of the study (preexposure, exposure, postexposure) on each test day</th>
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</thead>
<tbody>
<tr>
<td>Change in Nasal Resistance</td>
</tr>
<tr>
<td>-----------------------------</td>
</tr>
<tr>
<td>CPAP exposure day</td>
</tr>
<tr>
<td>Exposure vs. Pre-exposure</td>
</tr>
<tr>
<td>Postexposure vs. preexposure</td>
</tr>
<tr>
<td>Postexposure vs. exposure</td>
</tr>
<tr>
<td>No CPAP day</td>
</tr>
<tr>
<td>Exposure vs. preexposure</td>
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<tr>
<td>Postexposure vs. preexposure</td>
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<tr>
<td>Postexposure vs. exposure</td>
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</tbody>
</table>

Values are means ± SE. *The corrected value represents the Tukey-Kramer correction for multiple comparisons.
increase in nasal resistance over time. It is important to note that CPAP was administered in the present study without the potential ameliorating effects of heated humidification to the inspired CPAP airflow.

The observed differences in nasal resistance between CPAP and the control condition result from a tendency for nasal resistance values to increase during the control day and to fall during CPAP exposure. Why should nasal resistance increase during the control day? One possible explanation is the observed increase in nasal resistance on the control day is simply a random change. There are, to our knowledge, no reported measurements of nasal resistance among normal subjects over the 24-h period to ascertain circadian variation in nasal resistance. However, Paludetti and colleagues (27) measured nasal resistance in 56 normal subjects from 8 AM until 7 PM. They demonstrated, in addition to marked and reciprocal changes in unilateral nasal resistance over time (the nasal cycle), a tendency for the nasal resistance to increase from late morning until midafternoon, rather than at other times. The changes in nasal resistance off CPAP in the present study closely paralleled the time-wise changes in nasal resistance observed by Paludetti and colleagues. This “circadian change” in nasal resistance was abolished by administration of decongestant (27), suggesting that it arose from changes in mucosal vascularity and edema. In the present study, the daytime increase in nasal resistance was attenuated by the application of CPAP. Hence, it is possible that the tendency to increased nasal resistance during the control day may represent a circadian increase in nasal resistance rather than a random effect.

Why should nasal resistance be lower with CPAP? The present study is descriptive rather than mechanistic, and it cannot directly answer this question. One possible explanation would be an acute mechanical splinting effect of CPAP on the nasal soft tissues, as occurs in the oropharynx (36). In a study of subjects pretreated with the nasal decongestant oxymetazoline, Desfonds and colleagues (10) reported a reduction in nasal resistance to 30–45% of the baseline value immediately after application of nasal CPAP. Review of data from the present study, however, demonstrates a more modest reduction in nasal resistance with CPAP than that described by Desfonds and colleagues, and one that occurs gradually over a time course of 2–3 h. In fact, nasal resistance immediately after application of CPAP was not different from that immediately beforehand. These findings suggest that in the normal nasal airway, unlike the decongested nasal airway, the effect of CPAP is not simply a mechanical splinting action. One speculative explanation for the gradual reduction in nasal resistance observed during CPAP exposure in the present study is that the more positive intraluminal nasal pressure resulted in reduced turbinate mucosal vascularity and/or edema. The time course for interstitial fluid reduction with CPAP in other situations is typically over several hours rather than an immediate effect (4), but there are no published data on the effect of CPAP on turbinate mucosal size or vascularity.

The histological effect of CPAP on the nasal mucosa has been examined. The nasal mucosal epithelium demonstrated the appearance of microvilli, clumping of cilia, clustering of immunocompetent cells, and mucosal drying, after 3–6 mo of CPAP use (5, 8, 35). The effect of these changes on mucociliary transport time was inconsistent, being prolonged in one study (8) but normal in the other (5). There have been few attempts to measure the effect of CPAP on nasal resistance. Bossi and colleagues (5) reported no change in nasal resistance (measured by anterior rhinomanometry while off CPAP) in eight patients with OSA after 6 mo of treatment with nonhumidified CPAP. Although several authors have described the effects of different forms of humidification on mucociliary transport time (4), but there are no published data on the effect of CPAP on turbinate mucosal size or vascularity.

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### Table 4. Peak nasal inspiratory flow rates

<table>
<thead>
<tr>
<th></th>
<th>PNIF, l/min</th>
<th>P Value for Change in PNIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-CPAP exposure</td>
<td>94±9</td>
<td></td>
</tr>
<tr>
<td>Post-CPAP exposure</td>
<td>92±7</td>
<td>0.74</td>
</tr>
<tr>
<td>Pre-No CPAP exposure</td>
<td>95±7</td>
<td>0.42</td>
</tr>
<tr>
<td>Post-No CPAP exposure</td>
<td>91±7</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ±SE, PNIF, peak nasal inspiratory flow rate.

### Table 5. Subjective symptom scores: response to question 1 in Table 1

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>25th Percentile</th>
<th>75th Percentile</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before CPAP</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>After CPAP</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Before No CPAP</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>After No CPAP</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

Values represent median, interquartile range, maximum, and minimum symptom scores recorded.
Previous studies have not measured simultaneous nasal resistance and subjective nasal congestion in response to CPAP. In the present study, there was no significant change in nasal symptom scores between the 2 test days, despite a significant objective difference in nasal resistance for the period of CPAP exposure. However, the relationship between objective nasal resistance and subjective nasal congestion is often discordant (11, 20), and, in the present study, there was no significant difference in nasal resistance between the two test occasions after removal of CPAP. Of note, the four subjects with inactive allergic rhinitis behaved no differently in their response to CPAP to the other subjects in this study. There is one single case report of a symptomatic individual with active allergic rhinitis in whom the addition of heated humidification resulted in improved CPAP compliance, a significant reduction in nasal symptoms and an improvement in peak nasal inspiratory flow (PNIF) rate (37); however, nasal resistance was not measured.

PNIF rate did not change significantly between test days, despite the observed change in nasal resistance between the two test exposures. It is important to appreciate that PNIF was measured before and after CPAP exposure, and not during CPAP exposure, on the two test occasions. Hence the absence of any change in PNIF with CPAP is consistent with the similarity in nasal resistance values at the same time points off CPAP on each test day. PNIF was included in the protocol in the hope that it might provide a simple practical surrogate for posterior active rhinometry to detect CPAP-induced changes in nasal resistance. The value of PNIF is limited by effort dependency, the requirement for a leak-free seal between the nasal mask and face, and potential collapse of the lateral nasal walls during the sniff maneuver with associated flow limitation, which may not reflect the nasal resistance during tidal nasal breathing. In the current study the variability in PNIF measurements was greater than that for posterior rhinometric measurement of nasal resistance.

**Clinical significance of the findings in this study.** Although this study was conducted in normal subjects and not in patients with OSA, the study may have some relevance to CPAP treatment in patients with OSA. We have demonstrated that CPAP use per se is not associated with any acute increase in nasal resistance in normal subjects. This finding should prompt the clinician faced with the common acute symptom of nasal obstruction in patients commencing CPAP to consider other factors that could be contributing to the nasal obstruction. Mouth breathing is an important factor that may increase nasal resistance in patients wearing CPAP (31) and may result in reduced CPAP adherence (3). Hence, the complaint of nasal obstruction in individuals commencing CPAP treatment should prompt a suspicion of mouth breathing (which can often be alleviated by use of a chinstrap or other means)(2). Other factors that could be responsible for nasal obstruction in patients wearing CPAP include rhinitis medicamentosa (rebound nasal congestion following cessation of decongestant treatment) (22) and a change to a more recumbent posture (32) while wearing CPAP. The latter is important because patients with OSA are more likely to have nasal obstruction at baseline, and the presence of preexisting nasal obstruction results in a larger than normal postural change in nasal resistance (10). It is the authors’ opinion, based on the findings of this study, that if the complaint of nasal obstruction persists in subjects treated with CPAP, after ruling out the simple aforementioned causes, some objective evidence of nasal obstruction should be sought.

**Limitations.** This study has certain limitations. First, the study was designed to look specifically at the acute effect of CPAP on nasal resistance and nasal symptoms. As such, this study does not address the long-term effect of CPAP on nasal resistance or nasal symptoms. Second, the study was conducted primarily during wakefulness rather than during overnight sleep, and was conducted on normal subjects rather than sleep apneic patients. Hence one could argue that these may not apply to the clinical situation: the sleeping patient with OSA. The study design, however, was aimed at isolating the effect of CPAP on the nasal airway. Patients with sleep apnea tend to have higher nasal resistance and thus a tendency to mouth breathe when asleep, and the degree of mouth breathing may be highly variable from one individual to the other, and very difficult or impossible to control (24). It is already quite clear that mouth breathing increases nasal resistance (17, 31) such that any observed change in nasal resistance with CPAP exposure would be invalidated by alterations in mouth breathing. There have been only two published studies of the effect of the sleep-wake state on nasal resistance, and both studies concluded that the sleep-wake state does not alter nasal resistance (18, 25). It was for these reasons that the study design specifically targeted normal subjects and was conducted during wakefulness to eliminate the confounding effect of mouth breathing (2). Finally, it was not possible to blind subjects to the CPAP vs. control test days in this study because of the obvious mask pressure while on CPAP, and the knowledge of the test condition could potentially impact nasal symptom scores.

In conclusion, the effect of an acute exposure to nasal CPAP for 6 h is to reduce nasal resistance compared with the control (No CPAP) exposure. The reduction in nasal resistance is a gradual phenomenon over 2–3 h. Acute nasal CPAP exposure in normal subjects was not associated with any change in subjective nasal symptoms. The authors urge caution when attributing symptomatic nasal congestion occurring soon after CPAP exposure, to CPAP per se, in the absence of objective measurements of nasal resistance.

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Present addresses: M. San Pedro, c/o Sleep Laboratory, St. Michael’s Hospital, Toronto, Ontario, Canada; S. Willing, c/o Kingston Regional Cancer Center, Kingston, Ontario, Canada.

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