A Physiologic Comparison of Nasal and Oral Positive Airway Pressure*

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Study objectives: The effectiveness of nasal continuous positive airway pressure (CPAP) in treating obstructive sleep apnea (OSA) is based on raising the intramural pressure above a critical collapsing pressure of the oropharyngeal airway. It is currently unclear whether CPAP delivered orally is also capable of raising pressure in the oropharynx above the critical collapse pressure.

Design: We tested a novel oral CPAP device to determine whether the pressure-flow relationships are similar to nasal CPAP and whether the device alters these relationships. Patients were selected based on having moderately severe apnea and were randomized to nasal CPAP, nasal CPAP with oral device, or oral CPAP.

Setting: Johns Hopkins University, The Johns Hopkins Asthma and Allergy Center, Baltimore, MD.

Patients: Five men and two women with OSA were studied.

Interventions: Individual pressure-flow curves were constructed during the application of nasal or oral CPAP.

Results: We found the following: (1) a similar effective pressure eliminated inspiratory flow limitation for the nasal or oral CPAP; (2) as pressure in the nose or mouth was lowered below the effective pressure, a linear pressure-flow curve was obtained and a critical closing pressure was described; (3) similar mean (± SD) critical pressures of −0.3 ± 5.3, 1.7 ± 4.0, and 0.5 ± 2.8 cm H₂O, respectively, occurred for nasal CPAP, nasal CPAP with the oral device in place, and oral CPAP conditions (p > 0.1); and (4) the comparable mean values for upstream resistance were 27.8 ± 19, 19.1 ± 8.3, and 26.5 ± 26.7 cm H₂O/L/s, respectively, for the above three conditions (p > 0.1).

Conclusions: We concluded that comparable upper airway pressure-flow relationships were obtained during oral and nasal breathing. Moreover, effective treatment pressure is obtained when constant pressure is applied through either the nasal or oral route.

Key words: obstructive sleep apnea; polysomnography; positive-pressure respiration

Abbreviations: AHI = apnea-hypopnea index; CPAP = continuous positive airway pressure; NREM = non-rapid eye movement; OSA = obstructive sleep apnea; Pcrit = critical pressure; Pn = nasal pressure; REM = rapid eye movement; Rus = upstream resistance

Since its original introduction in 1981, nasal positive airway pressure has become the mainstay for the treatment of patients with moderate-to-severe obstructive sleep apnea (OSA).³ Although numerous studies have attested to its efficacy,² there has been increasing evidence that patients may not be able to adhere to the device for a variety of reasons, including bulky headgear and claustrophobia.²,³ To accommodate patients who cannot use nasal continuous positive airway pressure (CPAP), alternative types of full facemasks have been proposed and are currently in use.⁴,⁵ It is generally thought that the mechanism of CPAP action is due to the effects of raising the intraluminal upper airway pressure to a pressure above the positive critical transmural pressure of the pharynx or hypopharynx.⁶

In the current study, a novel oral device was employed to determine whether upper airway obstruction could be relieved with orally administered CPAP. We first determined whether the pressure-flow relationships of oral CPAP were similar to those...
of nasal CPAP, and, second, whether the oral device itself altered these relationships. Our findings demonstrates that the oral CPAP device (Oracle; Fisher and Paykel Healthcare Ltd; Auckland, New Zealand) produced remarkably similar pressure-flow relationships to those of nasal CPAP and that the device itself did not impose obvious alterations in overall upper airway function.

Materials and Methods

Subject Selection

Patients from the Johns Hopkins Sleep Disorders Center who had OSA were selected on the basis of results from standard polysomnography. Patients with an apnea-hypopnea index (AHI) of > 10 episodes per hour in non-rapid eye movement (NREM) and rapid eye movement (REM) sleep who had already been treated with nasal CPAP were selected for the study. Patients with underlying cardiac pulmonary disease were excluded, and the protocol was approved by the Johns Hopkins Institutional Review Board.

Experimental Setup

Patients underwent routine polysomnographic monitoring and staging of sleep with bilateral electrooculograms, electroencephalograms (C3-A2 and C3-O1 leads) and submental electromyograms. Pressure-flow curves were derived with both the nasal mask and the oral device. Pressure was continuously monitored throughout the experiment. Airflow was monitored with a pneumotachometer (Hans Rudolph; Kansas City, MO) and a differential pressure transducer (± 2 cm H₂O; Validyne; Northridge, CA).

The equipment setup and design for constructing pressure-flow curves have been described previously. In brief, the equipment was designed to maintain constant levels of nasal or oral pressure that could be abruptly changed from one level to another over a range of pressures from -10 to 20 cm H₂O. The pressure sources were connected to a valve that could be turned manually to toggle between positive and negative pressure sources. The outflow from this valve then was connected in series to the pneumotachometer and nasal mask as described above.

As can be seen in Figure 1, top, the oral device consists of two very flexible plastic shields between which the patient places his upper and lower lips. No other straps or head gear is required. A solid tongue glide of the device was designed to extend into the oral cavity to prevent the tongue from being displaced rostrally.

In Figure 1, bottom, an MRI scan of a patient with 10 cm H₂O pressure applied during wakefulness is shown. As can be seen, the oral retainer depresses the anterior portion of the tongue toward the floor of the mouth while the pressure elevates the velopharynx forming a seal posteriorly (arrow).

Protocol

Patients were monitored in the supine position with a pillow placed under the head. The precise protocol has been described previously. In brief, patients were allowed to enter sleep while the nasal pressure (Pn) was maintained at a “holding pressure” that ranged between 4.0 and 18.0 cm H₂O. The holding pressure was derived by systematically raising Pn until flow limitation was eliminated, as previously described. The holding pressure also represents the effective pressure that is used to treat our patients with OSA. During stable periods of NREM sleep, Pn was abruptly reduced for five breaths before being increased back to the holding Pn. By repeatedly reducing the Pn, the pressure-flow curves were constructed, and the level at which airflow ceased was defined as the critical pressure (Pcrit). This protocol was repeated under the following three randomized conditions: (1) nasal CPAP; (2) nasal CPAP with the oral device occluded to prevent leaks out of the mouth; and (3) nose clipped and oral CPAP.

Data Analysis

Five consecutive breaths were analyzed with each reduction in Pn. The relationship between maximal inspiratory flow and Pn was examined, and the least squares linear regression equation for the relationship was computed (Minitab Inc; State College, PA). For each experimental condition, a separate regression equation was generated and solved to determine the oral and nasal Pcrit, and the oral or nasal upstream resistance (Rus) from the site of pharyngeal collapse was calculated as the reciprocal of the slope of the regression equation. For each outcome variable (ie, Pcrit and Rus), an analysis of variance was utilized (Minitab Inc). A value of p < 0.05 was considered to be significant, and the values are reported as the mean ± SD.
RESULTS

Anthropometric and Polysomnographic Parameters

Seven patients with OSA were selected for study (Table 1). There were five men and two women. Overall, the patients were obese with a mean body mass index of 39.0 ± 13.4 kg/m². Their baseline sleep studies demonstrated clear-cut OSA with a NREM AHI of 67.5 ± 35.2 episodes per hour of sleep and a REM frequency of 60.5 ± 24.4 episodes per hour of sleep. Mild-to-moderate oxyhemoglobin desaturation was noted.

Nasal vs Oral CPAP

The Pn was maintained at an identical holding pressure of 11.4 ± 4.7 cm H₂O during both nasal and oral CPAP (Table 1), as well as during nasal CPAP with the oral device in place (not shown). Figure 2 shows typical pressure-flow plots from one subject for each of the three conditions. As previously shown, a linear pressure-flow relationship was demonstrated for the nasal CPAP condition with a Pcrit of 0.2 cm H₂O given by the level of Pn at zero flow (Fig 2, top). With an oral device in place, a similar pressure-flow relationship and Pcrit were demonstrated during the application of nasal CPAP (Fig 2, middle; Pcrit, 0.7 cm H₂O) and oral CPAP (Fig 2, bottom; Pcrit, 0.9 cm H₂O). It is also apparent that the selective application of oral CPAP results in similar pressure-flow relationships during sleep.

For the seven patients studied, the mean Pcrit values were 0.3 ± 5.3, 1.7 ± 4.0, and 0.5 ± 2.8 cm H₂O, respectively, for the nasal CPAP, nasal CPAP with oral device in place, and oral CPAP conditions. The Rus values were 27.8 ± 19, 19.1 ± 8.3, and 26.5 ± 26.7, respectively, for the three conditions. No statistically significant differences in Perit or Rus were observed.

DISCUSSION

The major finding of the current study is the demonstration of comparable pressure-flow relationships with positive airway pressure applied either through the oral or nasal route. Regardless of the breathing route, a similar Pcrit was demonstrated and essentially parallel slopes of the relationship were observed. Also, we were able to demonstrate that the device itself did not significantly alter upper airway collapsibility or the slope of the pressure-flow relationship. Thus, the application of comparable positive pressure either through the nose or mouth is equally effective in overcoming the upper airway collapse that is characteristic of patients with OSA.

The findings of the current study extend our previous understanding of the mechanics of the upper airway during the application of positive pressure. It has been shown previously that when pressure is applied through the nasal passages, a collapsing Pcrit and a pressure-flow relationship

Table 1—Baseline Patient Characteristics and Polysomnographic Data

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<th>HP</th>
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<td>84.6</td>
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<tr>
<td>% obstructive</td>
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<td>10.0</td>
<td>8.0</td>
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*TST = total sleep time; Avg = average; SaO₂ = arterial oxyhemoglobin saturation; Pholding = holding pressure; F = female; M = male.
can be described for the upper airway. In the current study, when either oral or nasal CPAP was applied, flow limitation was evident until the pressure was raised approximately 11 cm H₂O above the Pcrit in all subjects. In general, the treatment pressure for CPAP can be considered to be effective when flow limitation is eliminated. For illustrative purposes, we have shown the relationship of the effective pressure relative to the Pcrit with pressure applied with a nasal or oral device (Fig 3). Although the Pcrit is illustrated at the base of the tongue, in fact, the collapsing Pcrit reflects a composite pressure that is exerted by the

![Figure 2](image_url)

**Figure 2.** Individual pressure-flow plots from one patient during nasal CPAP (top), nasal CPAP with oral device in place (middle), and oral CPAP conditions (bottom). Po = oral pressure, Vmax = maximal inspiratory flow.

![Figure 3](image_url)

**Figure 3.** Anatomic relationships in a patient with CPAP applied nasally (top) and orally (bottom). The mean Pcrit value recorded in the current study is noted at the level of genioglossus muscle, and the mean holding pressure (ie, the effective intraluminal pressure) of 11.4 cm H₂O is noted. The atmospheric pressure is recorded as zero.
surrounding tissues of the upper airway at the site of collapse. Since the soft palate is compliant, it is, therefore, not surprising that the Pcrit remains similar under both experimental conditions, assuming that the site of collapse remains similar. Finally, although both the Pcrit and Rus were similar regardless of the breathing route, it is possible that a type 2 error occurred due to the small numbers of patients studied. Based on our previous data, we estimated that a clinically significant difference in Pcrit of 2 to 3 cm H₂O could have been detected in our patient sample with a power of approximately 80%. Therefore, the effect of positional maneuvers on subtle differences in Pcrit or Rus cannot be excluded.

There are some limitations to the current study. First, the number of patients studied was small. However, the results of the pressure-flow curves were remarkably similar under the various conditions studied. Moreover, while more obese patients might demonstrate higher Pcrit values, the therapeutic effective pressure would rise similarly. Second, it should be noted that nose clips were used for experimental purposes during oral CPAP administration to prevent the leakage of air through the nose. Nevertheless, at the end of the experiment, the nose clips were removed and the patients continued to sleep without evidence of disordered breathing. During this period of observation, no subsequent fall in mask pressure was detected, indicating no significant leakage through the nasal passages. Finally, it cannot be determined whether oral and nasal CPAP are comparable clinically since physiologic testing was our primary objective and long-term compliance was not assessed.

There are several immediate clinical implications. In general, the clinically effective CPAP pressure can be assumed to be the pressure that eliminates inspiratory flow limitation. As can be seen in Table 1, the effective pressure or the holding pressure was the same in both the nasal CPAP and oral CPAP groups and, as noted above, was approximately 10 cm H₂O above the Pcrit value. In fact, several current self-titrating CPAP devices are based on the principle of adjusting pressure to eliminate flow limitation. There are some immediate clinical implications. In general, the clinically effective CPAP pressure can be assumed to be the pressure that eliminates inspiratory flow limitation. As can be seen in Table 1, the effective pressure or the holding pressure was the same in both the nasal CPAP and oral CPAP groups and, as noted above, was approximately 10 cm H₂O above the Pcrit value. In fact, several current self-titrating CPAP devices are based on the principle of adjusting pressure to eliminate flow limitation.12–17 Currently, there are no standardized methods for titrating the CPAP device other than to report the reduction in the number of respiratory events, which may vary considerably between laboratories that utilize different event criteria and technology. Whether flow limitation needs to be eliminated to clinically treat sleep apnea is unclear. Nevertheless, there is little disagreement that if normal inspiratory flow is established, obstructive apneas and hypopneas cannot occur unless there is significant alteration in the underlying pressure-flow relationships during the night. Thus, patients with low Rus and/or low Pcrit values will need relatively little CPAP, while individuals with higher values for these parameters will need higher CPAP pressures. Based on our initial findings of almost parallel pressure-flow curves (indicating similar Rus values) and comparable Pcrit levels, it may be possible to prescribe a treatment pressure using either route at the initial titration. Should the patient prefer to use the alternative device after a period of usage, our results suggest that a retitration might not be necessary. Nevertheless, additional clinical studies will be necessary to confirm the clinical application, the need for retitration, and the complications associated with the oral CPAP device.

References

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