our results, taken together with those of others, strongly support the hypothesis that, for patients treated with inhaled steroids, clinical worsening of asthma could be controlled, at least in part, by increasing the dose of inhaled corticosteroids.

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Heat It or Wet It?
Nasal Symptoms Secondary to the Use of Continuous Positive Airway Pressure in Sleep Apnea

To the Editor:

We read with great interest the articles in CHEST by Martins de Araújo and colleagues (January 2000) and the editorial by Brown (March 2000) concerning the effects of humidification on nasal symptoms during nasal continuous positive airway pressure (CPAP) therapy. Recently gathered data suggest a role for heated, but not for cold, humidification in preventing nasal symptoms and improving compliance to CPAP treatment. From these clinical studies and the early research by Richards et al. it is not clear, however, which mechanism is responsible for the improvement in nasal symptoms: heat or humidity.

We recently studied a patient in whom severe nasal symptoms were resolved with natural warming of the nasal CPAP (nCPAP) circuit.

A 56-year-old woman, who had been treated for secondary hypothyroidism, was evaluated for suspected obstructive sleep apnea syndrome (OSAS). There was no history of rhinitis or sinusitis. She had marked daytime somnolence (Epworth score, 22), and her physical examination was unremarkable. She presented normal lung function but had resting hypoxemia (PaO₂, 66.4 mm Hg). The results of routine blood analyses were normal, and thyroid function was within normal range throughout our observations. A split-night polysomnography revealed an apnea/hypopnea index of 50 and established a CPAP pressure of 8 cm H₂O. Home CPAP then was started with good initial compliance, no side effects, and a significant clinical improvement (Epworth score, 8; PaO₂, 96 mm Hg).

Figure 1. Serial nPIF (black line) and nasal symptoms score (bars) in a patient with OSAS before and after circuit natural warming (placing the circuit of the nasal CPAP under bedclothes).
After 6 months of treatment, she began complaining of severe nasal symptoms: mucous rhinorrhea, sneezing, frontal headaches, and nasal obstruction. These symptoms were related to the usage of a nasal mask and improved with the voluntary interruption of CPAP. This noncompliance (average number of hours of CPAP use, 2.2 h) made OSAS symptoms recur (Epworth score, 20). No signs of infection were detected, the results of skin prick tests to common allergens and latex were negative, and an upper airway CT scan was normal.

Nasal peak inspiratory flow (nPIF) (In Check; Clement Clarke; Harlow, UK) was evaluated seven times daily before and after trying to restart nCPAP. The minimum daily value fell from 180 to 70 L/s, and nasal symptoms reappeared. Due to worsening OSAS symptoms, she agreed to another nCPAP challenge with monitoring of the nasal symptoms score (NSS) and nPIF, nasal lavage, and active anterior rhinomanometry, before and after 7 days of use. Compliance with treatment (ie, the number of hours of effective treatment) was downloaded from the CPAP machine using compliance software. The results of those tests showed an increase of the symptoms score from 0 to 15.3, a fall in nPIF, and a decrease in nasal flow on nCPAP therapy (total expiratory airflow decrease, 1,059 to 769 mL/s). No inflammatory cells were observed in nasal lavage fluid on both occasions.

After unsuccessful therapeutic approaches (ie, oral antihistamines, local and oral vasoconstrictors, nasal (pronitromide, and topical steroids), air temperature was thought to be a possible cause for the symptoms, and natural warming of the nCPAP circuit was tried by placing it under the bedclothes. This simple measure achieved complete resolution of nasal complaints (mean NSS, 1.3), with stabilization of nPIF records around 110 L/s and a gain in nCPAP effective compliance (average length of use, 6.3 h) with improved OSAS symptoms (Epworth score, 3) [Fig 1].

Our case shows that a simple, inexpensive approach resolved nasal complaints during CPAP therapy.

Although the mechanisms involved in the improvement of nasal symptoms secondary to CPAP in this patient are not fully elucidated, the beneficial effect of air warming suggests a role for the temperature of the inspired air. Before considering heated humidification (an expensive solution) for upper airway complaints during continuous positive airway pressure, a simple measure like natural warming of the circuit should be tried first.

So, for nasal symptoms secondary to CPAP wet it or/and heat it?

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To the Editor:

I have carefully read the clinical case reported by Winck et al, following the publication of our study1 and the editorial by Brown.2

This case report presents a lot of clinical details. The delayed onset of nasal symptoms is somewhat surprising, but the resolution of these symptoms after treatment was stopped, and their return after challenge favors a causal relationship with continuous positive airway pressure (CPAP).

However, this case report lacks important information. In particular, it does not tell us about the degree of relative humidity or room temperature at the time of the study, nor the temperature gain or variations of relative humidity of inspired air obtained with this “natural warming” technique. It would be possible, theoretically, to propose the opposite hypothesis to that proposed by the author, with a harmful effect of the increased temperature of inspired air. When air is heated, it increases its capacity to hold moisture and becomes unsaturated. Unsaturated air will absorb water from a wet surface by evaporation, until it reaches an equilibrium. This increased temperature of inspired air could increase drying of the nasal mucosa. However, it is true that a flow of cold air (at a temperature much lower than the usual temperature of the bedroom) through the nose has increased nasal resistance and led to the release of inflammatory mediators.3 In the study by Richards et al,4 increasing the temperature of inspired air (totally humidified) to >30°C limited elevation of airways resistance induced by mouth leaks with CPAP. However, this beneficial effect was small compared with the difference observed between cold and heated humidification. In our own study, there was no significant temperature difference at the end of the hole between cold and heated humidification, while the duration of mouth leaks was significantly decreased by heated humidification. Therefore, according to the conclusions of Richards et al,4 the nasal symptoms observed during treatment with CPAP appear to be due more to drying of the mucosa than to cooling of the mucosa.

Overall, as emphasized by the authors, the mechanism for improvement of nasal symptoms reported in this case is not fully elucidated. The results of this case report, already published as mentioned by the author, must therefore be confirmed by a prospective study supported by measurements of temperature and humidity.

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