Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Please include a cover letter with a complete list of authors (including full first and last names and highest degree), corresponding author’s address, phone number, fax number, and email address (if applicable). An electronic version of the communication should be included on a 3.5-inch diskette. Specific permission to publish should be cited in the cover letter or appended as a postscript. CHEST reserves the right to edit letters for length and clarity.

Short-term Budesonide Dosage

To the Editor:

We read with interest the study in CHEST by Foresi et al (February 2000),1 in which a randomized, double-blind, multicenter, parallel group-designed study was used to investigate the effect of a short-term increase in daily dosage of budesonide on the exacerbations of asthma. In this study, an exacerbation was defined by a fall in peak expiratory flow <70% from baseline value, calculated during the last 2-week pretreatment period, on at least 2 consecutive days. Patients were randomized into three groups. Patients in group 2 and group 3 received the same daily dosage of budesonide, 100 μg bid. The patients randomly assigned to group 2 received an additional course of budesonide, 200 μg qid, in the event of an exacerbation, while patients in group 3 received a placebo. The daily dosage of budesonide was the same for group 2 and group 3, the only difference being the method of treatment for an exacerbation. This difference could become clear only after the first exacerbation.

In the preprotocol analysis, the difference in patients experiencing at least one exacerbation was significant (χ²; p < 0.025). Unfortunately, this meant that the patients were not well randomized, because the a priori change for experiencing at least one exacerbation was the same when patients had the same baseline characteristics and when they received the same daily doses of budesonide.

The effect of the increased daily dose of inhaled budesonide was measurable only after the first exacerbation, because the effect of the increased dose may then have become apparent. However, the difference between the two groups of patients experiencing two or more exacerbations was not large and therefore seemed insignificant.

Because the patients were not well randomized, we think it was impossible to make a comparison between the patients of group 2 and those of group 3 in the number of days with exacerbations.

Based on results of this study, we do not think it was justified to conclude that a short-term, increased daily dosage of budesonide had a beneficial clinical effect on the onset of an asthmatic exacerbation.

Hanneke J. van der Woude, MD
René Aalbers, MD, PhD
Groningen, The Netherlands

Correspondence to: H.J. van der Woude, MD, Martini Ziekenhuis, afdeling longziekten, Locatie van Swieten, Van Swietenlaan 4, Groningen, The Netherlands; e-mail: h.woude@rnzh.nl

REFERENCE
1 Foresi A, Morelli MC, Catena E, et al. Low-dose budesonide with the addition of an increased dose during exacerbations is effective in long-term asthma control. Chest 2000; 117: 440–446
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.

Antonio Foresi, MD
Servizio di Fisiopatologia Respiratoria
Sesto San Giovanni, Italy

Correspondence to: Antonio Foresi, MD, Fisiopatologia Respiratoria, Mod Allerg Immun Polmonare, Viale Matteotti 83, Sesto San Giovanni 20099, Italy; e-mail: foresi@betacom.it

REFERENCES
2 Rodrigo G, Rodrigo C. Inhaled flunisolide for acute severe asthma. Am J Respir Crit Care Med 1998; 157:698–703

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.

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 ($PaO_2$, 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 H$_2$O. Home CPAP then was started with good initial compliance, no side effects, and a significant clinical improvement (Epworth score, 8; $PaO_2$, 86 mm Hg).

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21956/)

**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).