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.

Heliox Effect or Rapid-Onset Acute Severe Asthma

To the Editor:

We read with great interest the article by Kass and Terregino that appeared in the August 1999 issue of CHEST.1 In this article, the authors conducted a randomized controlled study to evaluate the effect of heliox on airflow obstruction and dyspnea in adult patients with acute severe asthma. The authors concluded that "heliox rapidly improves airflow obstruction and dyspnea in patients with acute severe asthma and may be useful as a therapeutic bridge until the corticosteroid effect occurs." Through a close inspection of the entry data, we found that the heliox group presented an important (statistically nonsignificant) difference in the mean duration of attack prior to emergency department presentation (32.6 vs 51.1 h). Accordingly, the heliox group is composed of patients with a more rapid-onset asthma than the control group. Additionally, it is well known that rapid-onset asthmatic patients exhibit a more severe asthma at presentation and a quick recovery compared with slow-onset asthma patients.2-5 The study data are congruent with this idea. Thus, heliox group patients appear more severe at presentation (peak expiratory flow, 120.9 vs 140.0 L/min; dyspnea score, 6.18 vs 4.04; respiratory rate, 30.1 vs 24.3 breaths/min; heart rate, 115.4 vs 104.6 beats/min), and had a fast recovery. Because the authors did not remove the bias introduced by that difference at the initial level, their conclusions appear doubtful. Consequently, the patient’s improvement could be due to that factor.

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REFERENCES


To the Editor:

The study by Kass and Terregino (CHEST; August 1999)1 adds to the growing body of evidence that heliox is effective in improving airflow obstruction and dyspnea in patients treated in the emergency department (ED) for acute, severe asthma. However, in the first paragraph of their discussion the authors state that “similar objective improvements have been demonstrated in children” and reference two studies, one of which was performed by my colleagues and myself.2 Using a double-blind cross-over design, we found that heliox had absolutely no effect on either FEV1 or dyspnea in 11 children who were admitted to the hospital with acute asthma. We concluded that heliox was not beneficial in our study population. While we did find that the children with less severe airway obstruction had some improvement in peak expiratory flow rate (PEFR), though not in FEV1, with heliox, those with the most severe airway obstruction had essentially the same values for PEFR and FEV1 during air and heliox breathing. PEFR is the spirometric value that is most likely to be associated with turbulent flow and, thus, is the most susceptible to improvement with heliox. I wonder whether the patients in the study by Kass and Terregino had any significant change in FEV1? As Kass and Terregino did not calibrate the peak flowmeters with heliox, we really do not know whether their two patient groups started from the same baseline. Moreover, how can we be sure that a change in PEFR with heliox using an uncalibrated peak flowmeter is equivalent to the same change in PEFR while breathing air? Nevertheless, there is substantial evidence that heliox can benefit patients presenting to the ED with acute, severe asthma. However, there are few data that confirm the benefit from heliox in the inpatient setting. Our study in children hospitalized with acute asthma found no benefit from heliox and does not support the findings by Kass and Terregino.

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Inhaled Indomethacin in Bronchorrhea in Bronchioalveolar Carcinoma

Role of Cyclooxygenase

To the Editor:

In patients with bronchioalveolar carcinoma, airway obstruction by a copious amount of sputum provides a risk for respiratory failure and even death.1 Homma et al (CHEST, May 1999)2 have shown that inhaled indomethacin reduces refractory bronchorrhea in bronchioalveolar carcinoma, suggesting a role of cyclooxygenase in airway hypersecretion in this disease.

We thus studied the effect of inhaled indomethacin on sputum production and the expression of cyclooxygenase-2 (COX-2) messenger RNA (mRNA) in seven patients with bronchioalveolar carcinoma having bronchorrhea. A transbronchial lung biopsy was taken from the most affected area, and the gene expression was assessed by Northern blotting. The COX-2 primers were 5′-GTCTGTAGTGATGCGCACATCGC-3′ (sense) and 5′-GATGCCAGTGTAGAGGCTTAAA-3′ (antisense), giving rise to a 276-base pair polymerase chain reaction product.3 Then, each patient received inhaled indomethacin, 75 mg/d, for 4 weeks, while the sputum expectorated was collected and weighed daily, and the values were reduced to weekly averages. A comparison between two groups, one showing positive for COX-2 mRNA expression and another negative, was made by analysis of variance and Scheffé’s test.

The expression of COX-2 mRNA was detected in four patients. During the baseline period, a mean sputum production was greater in the COX-2 messenger RNA-positive group (570 g/d) than in the COX-2 mRNA-negative group (165 g/d, p = 0.033, Fig 1). After the 4-week treatment, daily production of sputum decreased in the COX-2 mRNA-positive group by a mean ± SD of 49 ± 17% (p = 0.025), but it remained unchanged in the COX-2 mRNA-negative group. With respect to the change from baseline value, there was a significant difference between the two groups (p = 0.029). Therefore, upregulation of COX-2 in carcinoma cells and the resultant synthesis of cyclooxygenase products of arachidonic acid may be involved in the pathogenesis of...