Magnitude of Bronchoprotection of Albuterol vs Methacholine
Relationship to Baseline Airway Caliber

To the Editor:

Airway hyperresponsiveness (AHR) to methacholine likely relates to smooth muscle dysfunction in asthma patients and to a geometric function of reduced airway caliber in COPD patients. This geometric phenomenon should contribute to AHR in asthmatic patients with nonreversible obstruction.

Therapy with albuterol markedly protects against methacholine-induced smooth muscle contraction. We hypothesize that the geometric AHR component due to airway narrowing in asthmatic patients should be less inhibited by the administration of albuterol. In this brief report, we examine data from previous studies in 28 asthmatic patients.

All patients were nonsmokers with no other lung disease. All had refrained from using inhaled β2-agonists for > 2 weeks (indicating that β2-agonist tolerance would not affect the magnitude of bronchoprotection), and no patients were using controller medications. Study subjects were chosen only once, sequentially, as follows: 12 patients from one study; 7 patients from a second study; 6 patients from a third study; and 3 patients from a fourth study. The provocative concentration of methacholine causing a 20% fall in FEV1 (PC20) was measured before and 10 min after inhaling 2 puffs (200 µg) of albuterol. The bronchoprotective effect of albuterol was expressed as the dose-shift, which is the number of doubling concentrations that the PC20 had improved after albuterol administration. We examined the baseline FEV1 in the 10% strata, with 3 patients between 70 and 79% predicted, 8 patients between 80 and 89% predicted, 10 patients between 90 and 99% predicted, and 7 patients at ≥ 100% predicted.

There was a wide range of bronchoprotection (0.7 to 5.4 doubling doses; mean [± SD] number of doubling doses, 3.2 ± 1.0). There was no overall significant relationship between baseline FEV1 and bronchoprotection; however, in the FEV1 stratified data, a trend emerged (Fig 1). The mean bronchoprotection increased from 2.3 doubling concentrations in subjects with an FEV1 in the 70% predicted range to 3.6 doubling concentrations for those with an FEV1 of > 100% predicted. The regression of these four data points is significant (r = 0.96; p = 0.027).

These data are not definitive. However, they do support the hypothesis that the AHR component due to airway obstruction is less inhibited by therapy with inhaled β2-agonists. Confirmation in one or more larger studies comparing this in asthma vs COPD patients would be particularly interesting.

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Figure 1. The vertical axis represents the mean bronchoprotection for each of the four groups in doubling-dose shifts, and the horizontal axis represents the four FEV1 strata (70 to 79%, 80 to 89%, 90 to 99%, and ≥ 100% predicted).

Resource Allocation Issues and Clinical Practice Guidelines

To the Editor:

In their recent article in CHEST on “Resource Allocation Issues in Recommendations From Clinical Practice Guideline Panels” (January 2006),1 Guyatt and colleagues deliberated on the productive allocation of health-care resources to alternative management strategies. The authors stated, that “the annual cost of clopidogrel for 100 patients might equal the salary of one nurse in the United States, but the salaries of three nurses in Poland.” For the sake of everyone who would like to cite this example, we feel obliged to amend it. The authors cited inaccurate data provided bona fide by one of us (RJ). The appropriate data from the Polish General Statistical Bureau on the average salaries of nurses and the average cost to patients of clopidogrel indicate that the annual cost of clopidogrel for 100 patients would equal the salaries of at least 10 nurses in Poland.

We apologize for these misleading statistics that we provided to authors and readers alike.

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Staphylococcus aureus in Community-Acquired Pneumonia

To the Editor:

We are concerned about the frequencies of bacterial pathogens in patients with culture-positive community-acquired pneumonia (CAP) that were reported by Kollef et al (December 2005).1 Among 2,221 such patients, Staphylococcus aureus was identified in 25.5% of patients, Streptococcus pneumoniae was identified in 16.6% of patients, and Haemophilus influenzae was identified in 16.6% of patients.

From November 1999 to October 2003, 668 patients who were hospitalized in our department had a discharge code of pneumonia, chest radiograph infiltrates, and no hospitalization during the preceding month. Among 191 patients (median age, 68 years) with culture-positive CAP (ie, significant bacterial pathogens identified by blood culture [n = 60] and/or sputum culture), Staphylococcus aureus was identified in 57% (n = 109; 46 bacteremic cases), H influenzae was identified in 27% (n = 51; 3 bacteremic cases), and S aureus was identified in 7.3% (n = 14; 5 bacteremic cases). This frequency of S aureus is consistent with the level of 2.9% in seven previous studies of hospitalized CAP patients that were summarized by Marrie.2 In our opinion, the frequencies of bacterial pathogens reported by Kollef et al1 are too odd to support the guideline recommendations for CAP.

In the pneumonia population in the study by Kollef et al,1 S aureus was the only pathogen that was associated with increased mortality. Among our CAP patients detailed above, death within 30 days occurred in 29% of the 14 patients with S aureus identified (n = 4; one bacteremic case), compared with 5.1% of the 177 other patients with culture-positive CAP (n = 9; nine bacteremic cases; p = 0.009 [Fisher exact test]). Severe necrotizing pneumonia can be caused by S aureus carrying the Panton-Valentine leukocidin gene.3 However, S aureus isolates from three of our patients who died were tested and were negative for the Panton-Valentine leukocidin gene.4 Since S aureus is an uncommon cause of CAP, it does not need to be covered by the empirical CAP treatment. However, the severity associated with S aureus pneumonia reinforces the importance of performing routine blood and respiratory cultures in pneumonia patients.

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Diet and Exercise-Induced Bronchoconstriction

To the Editor:

We read with great interest the review article in CHEST by Parsons and Mastronarde (December 2005)5 on exercise-induced bronchoconstriction (EIB) in athletes. We believe that the discussion on nonpharmacologic therapy omitted to mention a...