mortality from asthma has by and large, remained unacceptably high.

Taylor and Sears (CHEST 1994; 106:552-59) review the evidence that purports to incriminate β-agonists after marshaling an impressive array of reports—"a selective examination of literature"—as your leader puts it. The current popular theory is that regular use of β-agonists is the cause of increased asthma morbidity and mortality. We are told that (1) administering an "as needed" dose of salbutamol or other β-agonist in chronic asthma is good therapy; (2) larger doses of the same must be given in acute asthma; (3) salbutamol premedication dampens allergen induced bronchoconstriction; and (4) yet small doses of salbutamol when regularly taken by a chronic asthmatic is harmful to the extent of causing mortality. All this is difficult to accept not just because of convoluted reasoning but also because it is based on nothing more substantial than laboratory evidence obtained from mild asthmatics in remission. The laboratory changes are "small and their significance cannot readily be translated to the clinical situation."3

We can read much into the altered patterns of airway reactivity. Bronchial hyperreactivity is a useful feature that separates the normal subjects from the asthmatics, but this test is of dubious value when it is applied to an asthmatic population. This is so, because the relation between airway caliber and bronchial responsiveness remains uncertain and controversial.4

Taylor and Sears bolster their hypothesis by citing some of the voluminous epidemiologic data. Their emphasis is on reports coming out of a small geographic area over a relatively brief period—the second epidemic. No doubt the epidemic was terminated by withdrawal of fenoterol, but it cannot be held up as an explanation for a global phenomenon of 30-years duration.

A disease that was once considered a benign one, has assumed menacing dimensions in terms of morbidity and mortality. Until we find the real cause no therapeutic agent or modality should be above suspicion.

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To the Editor:

Dr. Govindaraj rightly argues that no therapeutic agent or modality should be above suspicion until the real cause of increased morbidity and mortality from asthma is determined. We have assembled evidence that strongly suggests that high doses of β-agonists used long term (in contrast to short-term use in acute severe asthma where high doses are both necessary and safe) can have deleterious effect on chronic asthma. We are surprised that Dr. Govindaraj rejects our findings as being "based on nothing more substantial than laboratory evidence obtained from mild asthmatics in remission." Well conducted, randomized, controlled clinical trials provide a more secure basis for determination of beneficial and harmful effects of drugs that do not accord with the experimental data. No comparable experimental data exist to suggest corticosteroids may increase morbidity or mortality.

The charge of "selective examination of literature" is an echo of Ziment’s recent editorial and is unfounded. Critical analysis of the available literature on long-term effects of inhaled β-agonists shows that many studies which purport to show beneficial effects are inadequate to answer the question.5 What the literature does show is that while a modest dose of bronchodilator is very effective when needed, larger doses used frequently can have deleterious effect. Within a population, this effect can explain increased trends in morbidity and mortality. Again we emphasize that quality of evidence obtained or laboratory is not linked with cardiac effects of β-agonists, but rather is linked with episodes of life-threatening asthma,6 the severity of which is aggravated by frequent use of inhaled β-agonist.3

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Treating Acute Asthma

To the Editor:

We read with interest the article by Rodrigo and Rodrigo on the treatment of acute asthma in the Emergency Department (ED) (CHEST 1994; 106:1071-76). This is a well-designed study with adequate sample size that confirms the results of previous studies done in the ED.1,2 However, the authors’ conclusion that intravenous aminophylline does not add therapeutic benefit to the ED treatment of acute asthma should not be extrapolated to the care of patients hospitalized for acute asthma. We recently published a prospective study of adults hospitalized for acute asthma exacerbation.3 In our study, intravenous aminophylline did provide therapeutic benefit to asthmatics during the post-ED treatment. Improvement in FEV1 over 48 h was significantly better in the aminophylline group than in the placebo group. Also, the amount of “as needed” nebulized albuterol was significantly less in the aminophylline-treated patients. Although we agree that aminophylline is not beneficial in the ED treatment of acute asthma, we think it has a useful role in the treatment of adults hospitalized for an acute exacerbation of asthma, as long as the dosage is guided by serum concentration measurement.

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