bronchial mucosa were negative for cancer. The patient improved and was discharged after 2 weeks. After 6 months the atelectasis persisted, and a second bronchoscopy confirmed bronchial collapse.

It has been recognized that in patients with chronic bronchitis or emphysema, forced expiration causes disproportionate collapse of the large proximal bronchi, particularly the lower lobe bronchi. This occurs as a result of the abnormal increase in bronchial compliance due to pathologic bronchial changes in these patients. We can postulate that the following chain of events constitutes the pathogenesis of chronic bronchial collapse in our patient: (1) The primus movens probably was lower lobe atelectasis due to an acute bronchial obstruction (i.e., a mucus plug). (2) Since reduction in lung volume markedly increases bronchial obstruction, atelectasis caused persistent bronchial collapse during inspiration as well. (3) The consequent chronic bronchial obstruction, preventing recovery of the collapsed lung, produced chronic lobar atelectasis.

In conclusion, we believe that increased collapsibility of the lower lobe bronchi may represent a pathogenetic mechanism for chronic lower lobe atelectasis in patients with obstructive lung disease.

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β-Agonists and Bronchial Asthma

To the Editor:

In an article that appeared in the June 1992 issue of Chest, Dr. Ziment* questions whether asthma deaths are due to overtreatment or undertreatment, and refers to studies that I and colleagues have undertaken over the last 10 years with respect to asthma morbidity and mortality. Dr. Ziment states that it is "curious" that I now believe that there are risks attached to the use of β-agonist therapy, whereas I previously stated that there was no evidence for direct toxicity of high-dose β-agonist therapy. Dr. Ziment has not distinguished between management of acute severe asthma (for which there is no evidence that high-dose β-agonist treatment is responsible for mortality) and the long-term treatment of asthma (for which there is strong evidence that frequent use of inhaled β-agonist may contribute to deteriorating asthma). This distinction is crucial to understanding the issues. What I and others stated, when presenting the results of our studies of asthma mortality, is that we did not find evidence of cardiac-related death attributable to excess use of β-agonist therapy during the fatal attack. Since that time, Molfino and colleagues have provided further evidence that death from asthma is primarily due to asphyxia, not cardiac causes. We are all agreed that in acute severe asthma, β-agonist therapy is essential and that high doses can be administered safely along with corticosteroids and oxygen.

Two years ago we published a study showing clearly that the chronic regular use of a potent β-agonist was associated with worsening asthma control and increased morbidity. It is on the basis of that study, and other supporting evidence, that many of us now believe that the use of β-agonist as chronic maintenance therapy for asthma, whether on a regularly scheduled regimen or just used frequently for persistent symptoms, is deleterious to asthma control. The effect of frequent use of β-agonist is to increase severity, and thereby increase the risk of asthma mortality. The link between fatal asthma and the use of β-agonist is not the effect of the last 20 puffs, but rather chronic severe asthma related to the last 20,000 puffs, which have directly or indirectly increased the severity of asthma, which in turn has placed the patient at risk of a fatal attack.

Dr. Ziment states that overuse of sympathomimetic aerosols is not a factor in the increasing death rate from asthma in the United States. As evidence for this position, he states that no deaths have resulted from unrestrained use of aerosolized epinephrine, and that high doses of β-agonists have been used in the emergency room with no evidence that such treatment is hazardous. The first statement misses the point that the free availability of β-agonists and their frequent use as maintenance therapy for asthma may gradually (almost imperceptibly) increase the severity of asthma such that a larger proportion of the population have asthma of sufficient severity to be at risk of a fatal attack. The second observation again refers to the management of acute severe asthma in an emergency room setting, in which high-dose β-agonist therapy is appropriate and safe.

The problem is not simply overtreatment or undertreatment, but inappropriate treatment of asthma. It is inappropriate to use regular or frequent β-agonist therapy to control asthma on a long-term basis, as this appears to gradually increase the severity of asthma, although the mechanism is not yet determined. There is no evidence that use of high-dose β-agonist in the acute severe attack is related to mortality, even though some cardiac changes can be documented in the laboratory.

Again, I make the plea that authors and readers clearly differentiate between the beneficial effects of β-agonist in the treatment of the acute severe attack, and the adverse effects of frequent use of β-agonist on the control of asthma in the long term.

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To the Editor:

A few thousand patients die of asthma each year in the United States, and Dr. Sears fears that an explanation may be found in his discovery that some patients with relatively mild asthma appear to suffer deterioration when they use 3-adrenergic aerosols routinely. Nevertheless, it is evident that many millions of patients use routine 3-adrenergic aerosol therapy, and there is good reason to believe that as a result they enjoy a comfortable life. I feel that there is a logical inconsistency in the extension of Sears's claims: he implies that patients with chronic, variable asthma are harmed by using standard daily doses of 3-adrenergic therapy, but their resultant lapse into acute asthma can be safely managed by giving far greater doses of these same drugs. It is possible that the apparent harm of routine therapy is only of significance in a subgroup of patients such as those who use partially use fenoterol.

The established dogma of using routine 3-agonist aerosol therapy is supported by the bulk of the literature and by a vast experience. Before aerosol steroids were introduced in the 1960s, children with asthma relied principally upon sympathomimetic aerosols. With few exceptions, it appears that these children not only did well, but they "grew out" of their asthma. In the era of aerosol steroids, there is still enough documented experience to suggest that long-term 3-adrenergic therapy given alone is beneficial in children and younger adults. The vision of deaths that Dr. Sears relates to the continuing daily use of 3-agonists appears to be a mirage; most deaths occur in poorly compliant, psychologically disturbed, or economically disadvantaged patients with brittle asthma who fail to use their medications appropriately.

Fortunately, there is an important point that both sides can agree upon: without doubt, all drugs can be dangerous if used in excess. Thus, if a patient uses "excessive" doses of a 3-adrenergic aerosol, there is a risk of an adverse outcome. The critical issue lies in the definition of "excessive"; all parties might agree that this means "more than is symptomatically necessary." Two problems face us, however: (1) Do all symptoms (eg, wheezing on forced exhalation as well as wheezing on effort) deserve treatment? (2) How should we interpret the term "p.r.n.?" If I hear an asthmatic wheeze, but he does not report dyspnea on effort (perhaps because he makes no effort), I tend to advise treatment with a 3-adrenergic aerosol several times a day. Dr. Sears presumably would tend not to treat such a patient, unless the individual started exercising and became aware of dyspnea. Thus, Dr. Sears advocates 3-adrenergic aerosols only for airway obstruction that becomes symptomatic under special circumstances, whereas I favor treatment for all patients who present any evidence of reversible asthmatic bronchoconstriction.

The division between the Sears camp and traditionalists such as myself may depend on inappropriately loose terminology, and our differences may be partially resolved if we could correlate the need to treat clinical disease with the presence, as Sears1 suggests, of objective quantified measures of disordered physiology. If we could establish a rational set of criteria for defining "p.r.n.," we might find that our different approaches are really very similar. Thus, it is probable that those asthmatic patients for whom I choose to not prescribe routine 3-agonists are similar to those of Dr. Sears, whereas those for whom he does end up having to treat with routine daily "p.r.n. 3-agonist therapy are similar to those whom I treat with two- to four-times-a-day therapy. There is an intermediate group for whom the establishment of an appropriate therapeutic regimen is a challenge for each of us. However, it is now important for the two apparently opposed views on asthma therapy to define a mutually acceptable approach to 3-agonist dosing that covers the majority of patients with asthma—leaving room for disagreement on the remaining few.

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Prominent Elevation of the ST Segment by Convulsion

To the Editor:

Elevation of the ST segment on electrocardiography reflects the electrophysiologic state of injured myocardium, as in myocardial infarction, variant angina, and pericarditis. In acute myocardial infarction, the ST segment is elevated with a curve that is upwardly convex in the leads facing the infarction. An increase in serum creatine phosphokinase MB isoenzyme (CPK-MB) confirms the diagnosis. I report the case of a patient who showed a prominent elevation of the ST segment on ECG and an increase in serum CPK-MB, but had no evidence of myocardial infarction at autopsy.

A 66-year-old man with a history of symptomatic epilepsy due to brain infarction and chronic renal failure caused by bilateral renal stones was admitted to our hospital for acute pylonephritis and exacerbation of renal function. The pylonephritis did not respond to therapy and produced a high-grade fever, which was considered to be a trigger of general convulsion. The convulsion appeared repeatedly in spite of anticonvulsive drug infusion. Elevation of the ST segment was noticed on a monitored ECG 4 days after admission. A 12-lead ECG was then recorded immediately after an episode of general convulsion. The ECG showed a remarkably elevated ST segment, which was prominent in lead V1 (Fig I). The shape of the ST segment and the lack of a Q wave were not identical with the appearance in acute myocardial infarction. Echocardiography during a convulsion did not show dyskinesis of the left ventricular wall.

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