would be clinically significant. This dilemma in patient care is not easily resolved.

Clearly, the greater the number of diagnostic tests performed, the greater the likelihood of establishing the correct diagnosis. Conversely, when employing invasive diagnostic procedures more often, the risk of complications also increases proportionately. Ultimately, the exact risk-benefit ratio is not known for this diagnostic strategy. A study much larger and more comprehensive than either ours or that of Cazzadori et al is necessary to definitively answer this question.

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Primary Pulmonary Disease due to
Mycobacterium avium-intracellulare

To the Editor:

I read with interest Dr. Reich's report of a case of primary pulmonary disease due to Mycobacterium avium-intracellulare, which appeared in the May 1992 issue of Chest. However, in that article Dr. Reich states that "only three cases, all involving children, have been reported."

In August 1984 we reported a case similar to that of Dr. Reich in an 82-year-old male infant who presented with cough, stridor, and wheeze of 3 weeks' duration. Bronchoscopy showed a fleshy mass on the anterior wall of the left main bronchus. Acid-fast bacilli were seen on microscopy, but cultures were not done. Later, fasting gastric washings grew M avium-intracellulare scrofulaceum (MAIS) on culture. The Mantoux reaction was 8 mm to 10 IU of human tuberculin purified protein derivative (PPD) and 18 mm to 10 IU of avian PPD. An attempt to trace the source produced MAIS on several cultures of composted soil near the patient's house, which was in a rural area. However, the strain was not identical with that cultured from the patient. He made a rapid recovery on a regimen of isoniazid and rifampicin, even though the organism was resistant to these drugs, as well as to streptomycin and ethambutol.

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REFERENCES
1 Reich JM. Primary pulmonary disease due to Mycobacterium avium-intracellulare. Chest 1992; 101:1447-48

To the Editor:

I am grateful to Dr. Proust for bringing his case report to my attention. In reviewing his citations and my own, I find that the statement that "only three cases [of primary pulmonary disease due to M avium complex], all involving children, have been reported" is erroneous; the correct number is six. 1-4 Of considerable interest is that his patient, like ours, had avian exposure, as did one of two cases reported by Lincoln and Gilbert. 1 The individual reported by Kelsey et al resided "in a rural area with many domestic farm animals in the vicinity." No information concerning avian exposure was provided in the two cases reported by Powell and Walker. 1 Engbaek 5 reported three fatal cases of progressive pulmonary disease due to M avium complex in two siblings and their mother, and indicated that chickens were allowed to wander freely in the kitchen and bedroom.

In summary, there is strong circumstantial evidence that domesticated birds play a direct or indirect role in the causation of primary pulmonary disease due to M avium complex.

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Chronic Bronchial Collapse and Lower Lobe Atelectasis

Computed Tomographic-Bronchoscopic Correlation

To the Editor:

We would like to report the following interesting case. An 82-year-old man was admitted for cardiorespiratory failure. Thoracic computed tomography showed left lower lobe atelectasis. The left lower lobe bronchus was obstructed by endobronchial tissue of waterlike density. A diagnosis of endobronchial mucus plug was hypothesized. Bronchoscopy was immediately performed and showed diffuse tracheobronchomalacia as well as collapse of the left lower lobe bronchus during all the phases of the respiratory cycle (Fig 1). Specimens obtained by multiple-forceps biopsy of the

FIGURE 1. Bronchoscopy shows the collapsed left lower lobe bronchus (asterisk) and the patent upper lobe bronchus (arrow).
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 *primum movens* probably was lower lobe atelectasis due to an acute bronchial obstruction (ie, 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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REFERENCES
1 Wright RR. Bronchial atrophy and collapse in chronic obstructive pulmonary emphysema. Am J Pathol 1969; 37:63-77

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, Molinolo 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 unrefrained 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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REFERENCES
1 Ziment I. Infrequent cardiac deaths occur in bronchial asthma. Chest 1992; 101:1703-05