HIV infection. However, there is insufficient evidence to recommend the use of screening chest radiography in asymptomatic patients.

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REFERENCES

To the Editor:

We appreciate the insightful comments by Drs. Rosen and Schneider. We agree that direct comparisons to the Pulmonary Complications of HIV Infection study cohort cannot be made due to the numerous differences in methodology noted by ourselves and by Schneider et al.1 A large reason for the discrepancy between the two trials is the specialized nature of our cohort; most of our study subjects were inmates, the majority in psychiatric or prison wards. However, we would like to clarify several points. First, although initially identified by hospital ledgers, the entire medical record was reviewed for each patient; patients with incomplete data in their medical record were excluded. Each patient had been seen by medical staff of the Bellevue Chest Service, and both a history and physical were performed. Each patient had been seen by medical staff of the Bellevue Chest Service, and both a history and physical were performed. We are confident, therefore, that these patients truly had an absence of specific pulmonary symptoms.

Second, we agree with Drs. Rosen and Schneider that this study does not support the use of widespread screening chest radiograph (CXR) in HIV-1–infected individuals. We believe that such screening was proven cost ineffective both by the Pulmonary Complications of HIV Infection Cohort and by Olson et al.2 However, a directed screening of extremely high-risk individuals, as presented in our study, may still be warranted. Rosen and Schneider correctly point out that the population of our study is much different from that observed in other screening trials. While it is true that CXR should be obtained as a case finding study for those subjects with significant extrapulmonary disease and constitutional symptoms, and these subjects comprised a large percentage of our subjects, only 24% had constitutional symptoms. Furthermore, 40% of our subjects were admitted only for evaluation of their abnormal CXR findings. Most who had received a screening CXR were prisoners and were admitted only for evaluation of their abnormal CXR findings. Since patients with psychiatric illnesses and prisoners are usually underrepresented in clinical trials, it is likely that this explains some of the differences between our study and theirs.

Finally, an important point of our study is not whether directed screening CXR should be performed in select groups of HIV-1–infected individuals, but rather that an aggressive diagnostic approach should be used to evaluate the abnormality, and such an approach will have a high diagnostic yield. Furthermore, the high incidence of pulmonary tuberculosis has significant public health implications. Last, we wished to provide the clinician the results of the diagnostic modalities we employed to make the diagnosis. The Pulmonary Complications of HIV Infection trial did mention the radiographic abnormalities present in their cohort; this was misstated in our article.

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Cylooxygenase-2 Inhibitors in Aspirin-Induced Asthma

To the Editor:

Martin-Garcia et al (June 2002)1 have indicated the safety of a cyclooxygenase (COX)-2 inhibitor, rofecoxib, in patients with aspirin-induced asthma (AIA). The roles of COX-2 in allergen-induced lung inflammation remain poorly understood. When COX-1-deficient and COX-2-deficient mice are immunized with ovalbumin (OVA), lung inflammation indices (ie, number of cells in BAL fluid, proteins, IgE, and lung histology) are significantly greater in both mice than in wild-type mice.2 In contrast, the short-term inhibition of both COX-1 and COX-2 during allergen sensitization in BALB/c OVA-sensitized mice resulted in significantly greater airway hyperresponsiveness and higher levels of interleukin-13 in lung supernatants than was the case in untreated mice that had been OVA-sensitized.3 Although these murine models are not believed to be models of AIA, these two in vivo studies indicate that both the lack of COX genes during development and short-term COX inhibition may exacerbate allergic airway inflammation.

An increase in the release of cysteinyl leukotrienes (Cys-LTs) has been suggested to play a pivotal role in the pathogenesis of AIA. The decrease in prostaglandin E2 release due to aspirin-induced COX inhibition increases the level of Cys-LTs. A report4 has demonstrated that Cys-LT protein is overexpressed in bronchial biopsy specimens from patients with AIA. It also has been documented that the increase in Cys-LT levels following aspirin stimulation is unique to patients with AIA, compared with aspirin-tolerant asthma patients and healthy control subjects.5 Therefore, large amounts of COX-2 inhibitor may cause the deterioration of asthmatic symptoms resulting from higher levels of Cys-LT concentrations in patients with AIA. Further careful
experimental and clinical studies are needed to establish definitive guidelines for the safe use of COX-2 inhibitors in AIA.

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References


Talc Pleurodesis vs Iodopovidone

To the Editor:

The report by Olivares-Torres et al (August 2002)1 is an important first step in seeking a safe, inexpensive alternative method to talc for treating patients with pleural effusions by pleurodesis using iodopovidone. The numbers and results are quite similar to those in our 1991 report2 on thoracoscopic talc poudrage (TTP) in which we found a 100% success rate at 1 year when the occurrence of trapped lung was eliminated from the data (which Olivares-Torres et al did a priori). There were no instances of ARDS in our 39 patients or in 360 cases of TTP reported by my mentors.3

Since the calculated incidence of ARDS after talc is on the order of 1 in 700 to 1 in 140 patients,4 it would require a study involving thousands of patients treated with iodopovidone pleurodesis to prove that it is safer than talc pleurodesis. Dicyclicine and bleomycin pleurodesis have been reported in relatively few patients compared to talc pleurodesis, yet both have been associated with catastrophic respiratory failure.5 Ideally, a prospective randomized study will be performed.

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The 9% incidence of ARDS referenced by Olivares-Torres et al actually occurred in patients who had undergone surgical procedures, including pleural abrasion and talc application, suggesting that pleural abrasion may be a risk factor for ARDS. But their 17 cases of simple talc poudrage were associated with no instances of ARDS.6

It is worrisome that severe hypotension and pain due to iodopovidone occurred in three of the five patients with mesothelioma. We did not encounter this phenomenon among the 26 mesothelioma patients who had received TTP.7

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