pathologic diagnosis of asbestosis had normal films, but in five of these six the "structural abnormality was so mild as to suggest minimally altered function despite the nameable lesion" and their pulmonary function test results appear to have been normal.4

In the paper by Kipen et al., although 25 of 138 (18 percent) cases of asbestosis were "not radiographically detectable", there was pleural thickening and/or plaques in 15 so that only ten of 138 (7 percent) were normal x-ray films.5 It is claimed that pulmonary fibrosis was moderate or severe in nine of these ten. This is not in agreement with the studies cited above which demonstrated only mild histologic changes in patients with radiographically normal lungs. It is not our experience that functionally moderate or severe asbestosis is ever associated with radiographically normal lungs. No clinical or functional data are available for comparisons as this was an autopsy series of lung cancer patients. Pathologic methods in this series are open to question as the asbestos fiber burden was not quantified and minimal criteria for the diagnosis of asbestosis do not seem to have been applied (peribronchial fibrosis with asbestos bodies);6,10 thus, other causes for pulmonary fibrosis (ie, usual interstitial pneumonia, radiotherapy, chemotherapy, and adult respiratory distress syndrome) cannot be adequately excluded. Moreover, "the radiographs analyzed were taken at various times, anywhere from immediately preceding death to a few years before" and the films with the earliest evidence of carcinoma were selectively chosen, so that intragenic fibrosis from cancer treatment developing in the interval between the selected films and death is a real confounding variable not adequately excluded.

The one patient reported by Heard had no clinical, functional or radiologic signs of asbestosis but "mild fibrosis" at autopsy.11

The data presented by Rockoff et al.12 in which eight of 57 patients with "clinically diagnosed asbestosis" had "normal or near normal lungs roentgenographically" can be called into question for two reasons. First, it is difficult to clinically diagnose asbestosis in the face of normal roentgenograms.13 Second, against recommendations,14 the films in this series were read by only one interpreter.

Drs. Rockoff and Schwartz' statistical analysis should be entitled "probability of no radiographic pulmonary parenchymal disease with histologic asbestosis"; surely they concede that patients with pleural plaques and/or thickening do not have normal roentgenograms. The data used for the analysis are questionable for the reasons outlined above.

In their discussion, Drs. Rockoff and Schwartz state that the presence of asbestos-induced lung disease can best be diagnosed by a complete review of clinical, roentgenographic, laboratory and—when available—pathologic data. Has anyone ever denied this truism? It is then claimed that the early pulmonary lesion of asbestosis consists of discrete foci of peribronchial fibrosis which could be responsible for symptoms even if invisible roentgenographically. First, these lesions must be associated with accumulations of asbestos bodies to be ascribed to asbestosis.15 Secondly, bronchiolar wall thickening may be a response to cigarette smoking, smoking is "a significant cause of small airways abnormalities" (both pathologic and functional).13 Third, the clinical significance (if any) of minimal asbestosis detected pathologically in the face of normal pulmonary function tests, normal physical examination and normal radiographs is unknown. The frequency of this constellation of findings is certainly low. It is possible that high-resolution CT will detect mild interstitial lung disease in a small percentage of patients with no roentgenographic evidence of asbestosis. There is little doubt that CT can improve assessment of those portions of pulmonary parenchyma obscured by overlying pleural disease on radiographs.4

Drs. Rockoff and Schwartz conclude that "the ILO classification of normal should no longer be interpreted as the absence of lung disease in the asbestos-exposed individual" and that "the ILO classified film, in isolation, is of limited usefulness in predicting the presence of early asbestosis-induced disease in the individual subject, and should be better reserved for epidemiologic studies". We believe that either an ILO-classified normal film or a film not ILO classified but still normal is strong evidence against the presence of asbestosis. We agree with the ACPP/ATS committee that in the clinical setting, when the diagnosis of asbestosis has to be made in the absence of lung tissue, chest roentgenographic (and high-resolution CT) findings are of cardinal importance, and considerable caution is warranted in making a diagnosis of asbestosis in the absence of radiologic evidence of interstitial lung disease.15

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To the Editor:

We would like to make a short comment on the special report entitled, "Roentgenographic underestimation of early asbestosis by International Labor Organization Classification" written by Doctors Rockoff and Schwartz (Chest 1988; 93:1085-91).

We fully agree with the authors' warning on the frequency of
false negative x-ray results for asbestosis.

We would like to stress what we consider an equally important problem which is the frequency of false positive x-ray results (ie, a reading of irregular opacities of category I/0 or more when autopsy shows no asbestosis).

In 1976, a cross-sectional radiologic survey of 2,245 asbestosis miners was carried out using the ILO classification of 1971. Three readers experienced in pneumoconiosis work reported on the films.

Subsequently, by the end of 1983 we had obtained the lungs of 172 of the men for pathologic examination.

Percentages for false positive and false negative assessments for the three readers are given in the table.

It seems that the "higher" reader reads, the more false positives will be reported; the lower he reads, the more false negatives.

This re-enforces the opinion that the authors appear to share that the classification is an epidemiologic tool and should not be used for clinical or medico-legal work.

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The authors respond:

We are pleased that our article on the radiographic underestimation of early asbestosis generated such great interest and we welcome the opportunity to respond to the comments received.

Dr. Ducatman's letter supports our view that the ILO readings are neither intended for nor appropriately applied to the clinical diagnosis of asbestosis in individuals. In addition he relates his own provocative data concerning interobserver variability among "B" readers when interpreting profusions of greater than I/0 in medical surveillance of asbestosis-related disease. A greater than 200-fold prevalence range was demonstrated among the cases read by 23 observers. Moreover, expert readers (those who teach the courses of ILO classification methodology) tend to perceive fewer abnormalities than their colleagues.

As a consequence of the wide range in inter-observer variability, Dr. Ducatman is not only concerned with the application of the ILO methodology to clinical diagnosis, but he is also troubled about the reliability of results from application of the ILO classification to epidemiologic studies.

The American Thoracic Society has stated its position that a radiograph showing profusion of I/1 or greater is "... of recognized value" in the clinical diagnosis of asbestosis.1 However, Dr. Ducatman's data showing marked inter-observer variability, even among "B" reader instructors, would tend to reinforce our position that the use of any arbitrary threshold derived from the ILO system for the diagnosis of asbestosis is unreliable when applied to individual subjects.

We would also like to point out a recent article by Rossiter and colleagues2 in which a series of 100 radiographs were reviewed by 12 experienced readers from ten countries using the ILO classification rules. The discrepancy in the range of readings of more than one subcategory (on a ten-category scale of 0/0 to 3/3) varied from the median by 13 to 32 percent, with only four readers differing by less than 20 percent. Their findings would tend to support the international nature of inter-observer variability using the ILO system.

In an era of increased public awareness and federal legislation concerning the control of exposure to environmental dusts, we expect to be seeing fewer cases of severe asbestosis in the future and relatively more cases of "early" (and radiologically less obvious) asbestosis. We point out in our paper the generally accepted view that it is in the earliest forms of asbestosis that greater radiographic uncertainty occurs.

Based upon the observations made in our paper and Dr. Ducatman's data, we would make two specific recommendations: 1) inappropriateness of application of current ILO methodology to individuals be stressed to ILO "B" readers, and 2) ILO methodology be re-examined and restructured with a view to making it more sensitive to early asbestosis, so that it is a more meaningful epidemiologic tool in the future.

Dr. Cohen's communication basically makes one point; in his opinion, pulmonary asbestosis exists only if there is pulmonary impairment. This, however, is contrary to currently accepted scientific opinion. Authoritative sources,1,3 including the ILO itself, uniformly define asbestosis as the presence of interstitial fibrosis caused by the fibrous silicate asbestos, without reference to functional alterations. Also, The American Thoracic Society states: "The term asbestosis should be reserved for the interstitial fibrosis of the pulmonary parenchyma in which asbestosis bodies or fibers may be demonstrated."

We agree that histologically demonstrable asbestosis may or may not be clinically important when discovered. However, since one cannot predict which cases will be stable and which will progress, the absence of symptoms or functional deficits may mean that the asbestosis is more difficult to diagnose, but it does not mean that the presence of the disease is unimportant. Use of the ATS (or any arbitrary) criteria does not diminish the reality that radiographically normal lungs can be present despite the presence of histologically diagnosable asbestosis.

In advancing his viewpoint, Dr. Cohen holds that because the clinical criteria established by a committee of the American Thoracic Society (ATS)3 were not satisfied, he concludes that Epler and co-workers,4 Kipen and co-workers5 and my co-workers and I6 using histologic methods were not studying asbestosis. Given Dr. Cohen's stated reliance on the ATS position, we find this conclusion confusing since the ATS official statement on the diagnosis of asbestosis states: "... direct examination of lung tissue is the most reliable method of diagnosis..." With regard to recommending the application of the clinical criteria, it states: "In the absence of pathologic examination of lung tissue, the diagnosis of asbestosis is a judgment based on a careful consideration of all relevant clinical findings."

A further contradiction of Dr. Cohen's opinion can be found in a recent research report by Dr. Rosenstock and co-workers11 in which the relationship between pulmonary function, chest roentgenographic abnormalities and smoking status in a group of asbastes-exposed individuals was studied. They demonstrated that at least 5 percent of the persons in their research population with I/0 profusion abnormalities (which would be considered normal by ATS criteria) had restrictive impairment by spirometric criteria (10 percent if black workers are excluded), and showed that pleural changes alone were related to decrements in pulmonary function within the ILO categories with little or no parenchymal fibrosis (ie, ILO < 1/0).

They thus show that asbestos-exposed workers may have pulmonary impairment whether or not they have radiographically apparent interstitial pulmonary fibrosis, and they caution that although the