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 (i.e., a reading of irregular opacities of category 1/0 or more when autopsy shows no asbestosis).

In 1976, a cross-sectional radiologic survey of 2,245 asbestos 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" a 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 1/0 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 1/1 or greater is "... of recognized value" in the clinical diagnosis of asbestosis. 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 colleagues 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, 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 asbestos 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)
were not satisfied, he concludes that: Epler and co-workers, Kipen and co-workers and my co-workers and I 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-workers in which the relationship between pulmonary function, chest roentgenographic abnormalities and smoking status in a group of asbestos-exposed individuals was studied. They demonstrated that at least 5 percent of the persons in their research population with 1/0 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 (i.e., 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
ILO may be a useful epidemiologic tool it should be applied only with caution in the clinical evaluation of individuals. Dr. Cohen does not disagree with our observation that a significant minority of asbestos-exposed individuals may have histologic asbestosis despite a normal chest radiograph. We regret his failure to understand that asbestos may exist without symptoms or functional changes or, conversely, that asbestos-induced functional changes may exist in the presence of radiographically normal or near normal lungs. We would remind him that the ATS criteria are arbitrary, developed by consensus and without supporting research on compound probabilities, and that the radiographic ILO criteria used by the ATS are arbitrary and without histologic basis. ATS criteria may therefore not be the optimum approach to the clinical diagnosis of asbestosis.

We are pleased that Drs. Friedman and Fiel agree that asbestosis may be present despite a normal chest radiograph, and the ILO classification is purely descriptive. We would agree with them that our article could have been alternatively entitled: "Probability of no radiographic pulmonary parenchymal disease with histologic asbestosis." However, the remainder of their letter requires some discussion to put it into proper perspective, remembering that our principle thesis is that the ILO methodology is inappropriately applied to individuals, especially in early asbestosis.

Drs. Friedman and Fiel aptly point out that overreading of chest radiographs (using ILO classification) may be sizable and quote articles finding 11 to 33.9 percent incidence of overreading of small irregular opacities in individuals without asbestosis. We would point out that if these data are coupled with our calculated 10 to 20 percent underestimation of radiographs with histologic asbestosis, then there is an inappropriate ILO attribution of degree of disease, (ie, underreading and overreading) of significant magnitude. Thus, these collective data would support our conclusion that application of the ILO method to individuals is inappropriate. Also, relative to the above discussion by Dr. Ducatman regarding interobserver error, overreading data provide added concern about the application of the ILO system to epidemiologic studies as well.

ATS criteria for the diagnosis of asbestosis are advocated by Drs. Friedman and Fiel. However, based on the above limitations, we would join Dr. Hans Welle— a member of the ATS Committee which developed the criteria—in having reservations about the arbitrary criterion of 1/1 profusion, determined from the ILO classification, being recommended for the diagnosis of asbestosis.

Drs. Friedman and Fiel suggest that functional alterations are necessary for the existence of asbestosis, a criterion not necessary for the existence of other diseases in medicine including cancer and coronary artery disease and not germane to our discussion. Also, we address this issue above in some detail in our response to Dr. Cohen's letter.

They hold that the finding of pleural plaques makes a film "abnormal," which is true, but this radiographic finding has no systematic relationship to histologically demonstrated pulmonary asbestosis, which is what we are discussing. However, we would point out parenthetically that asbestos-induced pleural changes alone were recently shown to be capable of causing functional changes, in spite of radiographically normal lungs.11

Drs. Friedman and Fiel are concerned with our prior paper on fissural thickening in asbestos exposure.10 We agree that it is difficult to diagnose asbestosis in the face of normal lungs on a chest radiograph. The point of our previous paper was to show that fissural thickening may aid in the diagnosis in such cases. They also state that we used only one radiographic reader in evaluating the radiographs of the eight of 57 patients with clinical asbestosis but normal lungs radiographically. This is not true. As described in the Methods section and in Table 6 of that article, an interpretation of these eight cases by an outside B reader was available at the time of our own evaluation to enter them into our study. We would also observe that in the work by Gaensler and Carrington,19 which Drs. Friedman and Fiel quote and on which they heavily rely, there appears to have been only one x-ray reader (ie, no inter-reader variability data are given) and these authors state explicitly that they used only one histology reader.

Drs. Friedman and Fiel bring up the use of computed tomography (CT) and state "there is little doubt that CT can improve assessment . . ." of asbestosis.14 The use of CT is, of course, extraneous to our subject but we would note in passing that their work was a self-fulfilling prophecy since the authors made CT the "gold standard" at the outset and did not objectively evaluate the competing (or hopefully complimentary) modalities.

In their conclusion, Drs. Friedman and Fiel state that a diagnosis of asbestosis should be made with caution in the radiographic absence of interstitial lung disease. We agree. However, our point is that a negative chest radiograph does not, in itself, rule out asbestosis and, in fact, such may be the case 10 to 20 percent of the time.

We are pleased that Drs. Friedman and Fiel mention no flaws in our mathematic analysis quantifying the degree to which underestimation using the ILO classification may occur. We would point out that the ILO-related issues raised by them only serve to emphasize the need not to apply the ILO—or any other arbitrary system of classification—to radiographs of individuals until the system has been validated as being reliable at the histologic level.

Dr. Sluis-Cremer's and Ms. Hnizdo's letter, in agreeing with our opinion concerning the inappropriate use of the ILO classification for clinical (or medico-legal) purposes, reinforce this concept by supplying histologic documentation for both the false positive and false negative interpretations which occur with utilization of the ILO. They also suggest an interesting and logical observation; that readers who tend to interpret films as positive have fewer false negatives and vice versa. We take Dr. Sluis-Cremer's views seriously since his 1964 study10 is the only sizable radiographic-histologic correlation in asbestosis, the results of which showed that a large proportion of cases of histologically demonstrated asbestosis of slight degree and nearly a third of cases of moderate asbestosis were not detected radiographically.

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Communications

No More Alphabet Soup!

To the Editor:

Although I like alphabet soup, I hate initials in journal articles. They are confusing if they are many, difficult if they are nonstandard, despairing in they are outside my specialty, and burnable if accompanied by poor English. They slow reading and discourage its completion. I will gladly pay for the extra space for "control" instead of "c" , "gentamycin" instead of "g" and "diameter" instead of "diam." Don't encourage this mischief—keep the letters in the soup.

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Reexpansion Pulmonary Edema Localized to a Lobe

To the Editor:

Reexpansion pulmonary edema following drainage of pneumothorax is well known and is reported to affect the whole lung. We report the first case of reexpansion pulmonary edema localized to a lobe.

Three days before admission, a 48-year-old man presented a right-sided pleuretic chest pain. Examination and chest radiography showed a large right-sided pneumothorax with complete collapse of the right upper lobe and partial collapse of the other lobes.

An intercostal drain was inserted in the mid-axillary line and 20 cm H₂O negative pressure was applied to the pleural space. Chest roentgenogram two hours following the procedure showed a complete reexpansion of the right lung. Twelve hours later, chest radiography demonstrated alveolar infiltrates strictly limited to the right upper lobe. The patient was asymptomatic and examination disclosed crackles throughout the upper lobe. The lower lobe was clear. No specific therapy was needed. Marked radiographic improvement was seen on the second day and complete resolution was noted on the third day. On day 2, the chest tube was removed. The patient was discharged on day 6 after fiberoptic bronchoscopy results were normal.

Reexpansion pulmonary edema (RPE) occurring after drainage of pneumothorax or pleuresy is a well recognized complication. RPE involves usually the whole affected lung. To our knowledge, an RPE localized to one lobe has never been reported in the literature. A variety of factors associated with the development of RPE have been described: duration of collapse over 72 hours, complete collapse, high negative intra-pleural pressure and rapid reexpansion. 1 In our observation, RPE was localized in the right upper lobe that had been completely collapsed before pleural aspiration, and respected the other lobes that were incompletely collapsed.

Though the different lobes had the same duration of collapse and the same negative pressure was applied to them, RPE was limited to the upper lobe. Our case shows the important role of complete collapse as a risk factor for RPE. Fiberoptic bronchoscopy did not show any endobronchial obstruction that might explain the disparity of collapse of the right lung.

This observation is the first documented case of RPE localized to a lobe. RPE occurred in the lobe that was completely collapsed and respected the lobes that were only partially collapsed.

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Pressure-supported Breathing

To the Editor:

I read with great interest the article entitled "A Hazard of Pressure Support Ventilation" (Chest 1988; 93:333-35). In particular, the point that new ventilatory modes can yield unforeseen trouble-shooting problems is very much appreciated.

However, there is one point which I believe should be addressed by the authors. The article states that, in the presence of a large leak, the pressure-supported breaths on a 7200a ventilator will not cycle from inspiration to expiration. This is certainly true, but the article leads one to believe that the pressure support level will be held on the airway indefinitely in this circumstance; this is not true.

When the apnea interval set on the ventilator has elapsed without the initiation of a new inspiration, the pressure will be released and the ventilator will alarm as it goes into apnea ventilation mode. In our institution, the apnea interval is always set at 20 sec. The authors do not report what their apnea interval was, but I feel it was very likely set at 60 sec, and that this is the only reason that the phenomenon they reported lasted long enough to be observed.

Bennett, in order to get their cycling mechanism in line with