are in accord with two large studies of patients with various interstitial fibrosis, including asbestosis. Actually, these two large studies were the above-reviewed studies of Epler and Gaensler and Carrington, essentially the same cohort group.

Rockoff and Schwartz reported fissural thickening on chest x-ray films in 54.5 percent of 320 asbestos-exposed workers. A second group of 59 workers (ten or more years of asbestos exposure) were evaluated clinically—eight (19.5 percent) were felt to have clinical findings indicative or diagnostic of pulmonary asbestosis. In those patients who underwent pulmonary function testing, two had mild restriction, one severe obstruction (pathology showed severe pulmonary fibrosis) and four had mild small airways obstruction. Pathology findings revealed interstitial fibrosis in four patients.

There is general agreement with Rockoff and Schwartz that sole reliance upon the ILO-classified chest x-ray film for determination of the presence of early pulmonary asbestosis in individual cases is inappropriate. The presence of asbestosis induced lung disease can best be diagnosed by complete review of clinical, roentgenographic, laboratory and, when available, pathologic data.

A diagnosis of clinical asbestosis should be made in accord with the official statement published by the American Thoracic Society Ad Hoc Committee of Scientific Assembly on Environmental and Occupational Health in conjunction with the American College of Chest Physicians in 1986. Of these criteria, findings on chest roentgenogram were said to be the most important.

Since neither Rockoff's study nor his references satisfy these criteria for diagnosing asbestosis, Rockoff and Schwartz's statistical analysis of these studies cannot logically conclude that asbestosis exists in 10 to 20 percent of asbestos-exposed individuals with normal chest x-ray results.

I have performed over 1,000 comprehensive disability evaluations on asbestos-exposed workers in the San Francisco Bay area over the past ten years. In reviewing the medical records, I occasionally encountered a diagnosis of asbestosis in workers with normal chest x-ray film and borderline normal pulmonary physiologic testing (eg lowered vital capacity and/or lowered diffusion capacity). Usually, Epler's study was cited as a supporting reference.

I believe that such diagnoses are erroneous, and that the borderline pulmonary function tests are explainable by such factors as obesity, lack of maximal patient effort, other disease entities such as COPD, failure to correct for racial differences, or interlaboratory variations in techniques and/or predicted values. Hopefully, clinicians will adhere to the ATS guidelines when making a diagnosis of asbestosis and no longer diagnose asbestosis in an asbestos-exposed worker with a normal chest x-ray film.

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REFERENCES


To the Editor:

We read with interest the article by Rockoff and Schwartz on roentgenographic underestimation of early asbestosis by ILO criteria. Although it has a valid point to make—that "asbestosis" may be present in the face of a normal chest x-ray film—we feel that this point is not placed in proper clinical perspective. It is our opinion that the authors' arguments do not do full justice to all sides of the issues, and that they have set up the ILO system as a straw man.

In their introduction, the authors state without supporting references that "we have become aware that the chest roentgenographic examination tends to underestimate the presence of early interstitial disease" and do not refer to the more prevalent opposite side of the coin as presented in articles in their own bibliography—overreading of roentgenograms compared to histologic appearance. Over 33 percent of chest x-ray films from normal, occupationally-unexposed control subjects may be interpreted as demonstrating irregular opacities of low profusion (1/0 to 1/2). Marked pleural disease may be misclassified as parenchymal disease on radiologic interpretation.

Overreading of chest roentgenograms is addressed in other articles not cited by Rockoff and Schwartz. One study found an 11 percent incidence of what was interpreted as grade 1/0 through 2/1 profusion of small opacities in patients with no documentable dust exposure or any other specific medical etiology. Smoking was considered a possible contributing factor. A recent review article presents a strong case that smoking causes diffuse interstitial pulmonary fibrosis which is radiologically visible with low profusion (0/1 to 1/0) in low prevalence, creating ambiguity in distinguishing roentgenologic signs of early asbestosis from changes related to smoking. Another recent article found a false-positive rate for asbestosis of 17 percent on chest roentgenogram interpretation by B-readers.

Drs. Rockoff and Schwartz write that the ILO classification is based on consensus, gives only semiquantitative data and is purely descriptive. This is hardly news to anyone who has taken the American College of Radiology course on the pneumoconioses—the ILO system does not purport to be anything else! They then state, in a perjorative fashion, that "the ILO classification is applied by an x-ray reader without knowledge of the worker's dust exposure history, clinical symptoms, physical signs, or laboratory data" and that the B-reader examination does not require expertise in these areas of pulmonary medicine. This should not be viewed as a criticism. In order to function properly as an independent variable in the clinical assessment of an individual exposed to asbestos, the chest x-ray film (or chest CT) should be interpreted without knowledge of these clinical data!

In the paragraph outlining the "failure of roentgenographic-histologic correlation in early asbestosis", the authors cite a paper with one patient with asbestosis and no evidence of interstitial lung disease on chest x-ray film and three papers with large numbers of patients and substantial percentages of normal lungs radiologically in the face of histologically-proven asbestosis. The latter two papers are from the same institution with most of the same authors, so those are probably the same patients. Gaensler reported that in his eight patients with asbestosis and normal chest x-ray results, "the lesions (histologic) were so slight, so few, or so small that the pathologist's estimate of functional loss was graded 0". Pulmonary function test results from these eight patients cannot be gleaned from this paper. Epler reported that six of 58 patients with a
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.  

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.  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 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).  

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 iatrogenic 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.  

The data presented by Rockoff et al. 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.  Second, against recommendations, 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.  Secondly, bronchiolar wall thickening may be a response to cigarette smoking; smoking is a "significant cause of small airways abnormalities" (both pathologic and functional).  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.  

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 ACCP/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.  

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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; 93:1095-91).  

We fully agree with the authors' warning on the frequency of