certainly deserves our very careful study.

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Exercise Impairment in Asbestos-induced Pleural Fibrosis

Is it Really Just Interstitial Fibrosis?

Cardiopulmonary exercise testing is a useful tool for assessing the functional integrity of the cardiovascular and respiratory systems during maximal exercise.1,2 Numerous reports document its use in clearly defining the adverse effects of asbestos exposure on pulmonary physiology.5-5 Shih and co-workers in this issue of Chest (see page 1370) attempt to determine if research subjects with only radiographic evidence of asbestos-induced pleural fibrosis have abnormal pulmonary physiology during maximal exercise.

Subjects with significant asbestos exposure are at risk for a number of pulmonary/pleural complications including interstitial lung disease, pleural plaques, pleural thickening, mesothelioma, and bronchogenic carcinoma. Roentgenographic evidence of asbestosis is documented classically by use of the International Labor Organization (ILO) classification system of pneumoconioses.6 Although it is well recognized that asbestos-induced interstitial lung disease is associated with physiologic impairment, recent studies have shown that asbestos-induced pleural changes are also associated with restrictive lung function.7,8 However, the mechanisms underlying this association are poorly understood. It is entirely possible that asbestos-induced pleural changes are associated with occult parenchymal disease, which is not recognized by standard roentgenographic methods.

Shih and coworkers present convincing evidence that patients who have no roentgenographic evidence of asbestosis, ie, asbestos-induced interstitial lung disease, may have significant impairment of gas exchange at maximal exercise. Interestingly, this observation (evidenced by an elevated alveolar-arterial oxygen pressure difference gradient and an elevated dead space ventilation/tidal volume at maximal exercise) occurred in patients with roentgenographic evidence of diffuse pleural thickening, but it did not occur in subjects with either a normal pleura or pleural plaques alone. These findings were not likely the result of restriction in chest wall mechanics, as subjects with pleural thickening failed to exhibit high respiratory rates or low tidal volumes. The authors suggest that these subjects with diffuse pleural thickening may, in fact, have subclinical or occult asbestos-induced parenchymal lung disease. The data suggest that subjects with asbestos-induced pleural disease may have associ-
ated interstitial fibrosis not detected by conventional chest x-ray films. Additional imaging techniques such as high resolution computerized tomography may be useful in documenting interstitial changes not present on routine chest x-ray films.

The assessment of occupational lung disorders has been standardized by the use of the ILO classification for roentgenographic abnormalities as well as the use of routine pulmonary function testing. Cardiopulmonary exercise testing may also be of benefit in the assessment of patients at risk for the development of pneumoconioses. Other technologies such as high resolution computerized technology may further improve our ability to assess this patient population. The downside, however, is the added cost associated with sophisticated methods used to document occupationally related disorders. In the era of healthcare reform, careful consideration needs to be given to the use of additional methods to assess suspected impairments of pulmonary physiology. If future studies support the findings of Shih and coworkers, it is possible that additional roentgenographic classifications, eg, diffuse pleural thickening, may uncover subjects at risk for parenchymal lung disease. Further documentation of suspected impairment in these selected individuals may be justified by the use of additional imaging or physiologic testing.

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