Cigarette smoking and asbestos exposure are two of the most widely known and extensively studied causes of chronic lung injury. It is not uncommon for these two exposures to occur in the same individual. Pulmonologists are frequently asked to define the extent of lung injury in individuals with combined exposure and to attribute or pro-rate the injury to specific causes. In this issue of Chest (see page 299), Barnhart and coworkers offer an important observation about the complexity of using pulmonary function tests to assess lung injury in individuals with combined exposures.

Cigarette smoke and asbestos produce different patterns of lung injury, and both patterns of lung injury can occur within the lungs of a single individual. An understanding of the effects of combined exposure begins with an understanding of the patterns of injury of the separate exposures.

Cigarette smoking results in three separate, but often interconnected disease processes: (1) chronic mucus hypersecretion, resulting in chronic cough and phlegm production; (2) airway thickening and narrowing with expiratory airflow obstruction; and (3) emphysema, which is an abnormal dilation of the distal airspaces along with destruction of alveolar septa. The pattern of change on pulmonary function testing is one of increased static lung volumes (TLC, FRC and RV) and decreased rates of expiratory airflow (FEV₁, FEF25-75 and FEV/FVC). Among the earliest changes noted are in measures of small airway function and these measures correlate well with respiratory bronchiolitis with goblet cell metaplasia, inflammation of the bronchiolar wall, smooth muscle hypertrophy, small amounts of peribronchiolar fibrosis, and pigmentation of the bronchiole. Individuals with abnormal test results of small airway function from cigarette smoking often also have airflow obstruction using measures of expiratory airflow (FEV₁, FEF25-75, FEV/FVC). Chest roentgenographic changes due to smoking are slight in comparison to the functional changes. Only a small fraction of smokers have roentgenograms interpreted as showing interstitial fibrosis, and the amount of fibrosis present is modest.

The pattern of change with asbestos exposure in nonsmokers is one of reduced static lung volumes, particularly TLC, and relative preservation of the rates of expiratory airflow. The FEV₁ declines, but when adjusted for the reduction in lung volume (FEV₁/FVC), it is preserved or increased. Nonsmoking asbestos workers may have an increased upstream resistance due to changes in the small airways, but the FEV₁/FVC is normal. This preservation of flow rates is due to an increased elastic recoil of the lung which provides a greater driving pressure for airflow to overcome the increased resistance in the small airways. The chest roentgenographic changes secondary to asbestos exposure are well described and include interstitial fibrosis that may be both diffuse and severe.

The injury that results from the combination of cigarette smoking and asbestos exposure is similar to that expected from a combination of injuries due to isolated exposure. Becklake and coworkers have shown that smoking asbestos-miners have less of a decline in static lung volumes (including TLC) than nonsmoking asbestos workers, but the miners who smoke have a greater decline in FEV₁, FEF25-75 and FEV/FVC.

This pattern suggests that the lungs of smoking asbestos workers are experiencing simultaneous injuries from both cigarette smoke and asbestos fibers. The pulmonary function pattern is one of an obstructive process due to the inflammatory and emphysematous changes due to smoking superimposed on a restrictive process due to the interstitial fibrosis produced by asbestos. For some measures of lung function (FEV₁) both of these processes of injury influence the measure in the same direction (decrease); but for TLC the emphysematous changes due to smoking increase TLC while the fibrotic changes due to asbestos decrease TLC. As a result, TLC may be artificially preserved at nearly normal values even in the presence of extensive disease.

Because these two processes affect TLC in opposite directions, TLC becomes a poor measure of the extent of disease and an insensitive measure of the presence of disease when lung injury is due to the combination of a restrictive disease process and an obstructive disease process.

As Barnhart and coworkers observe, workers with both interstitial fibrosis and COPD (whose TLC was normal) had a lower FEV₁, and DCO and a larger A-a gradient than the group with only interstitial fibrosis (whose TLC was significantly decreased). In this setting, the pattern of physiologic change on pulmonary
function testing should be evaluated in assessing the extent of impairment, rather than the statistical normality or abnormality of the individual volumes or flow rates.

One caveat needs to be considered in extrapolating the observations made by Barnhart and coworkers to other asbestos exposed populations. As they discuss, cigarette smokers with no known asbestos exposure have interstitial fibrosis on chest roentgenograph in a low percentage of cases. Cigarette smoking does not result in the extensive pulmonary fibrosis that characterizes severe asbestosis, but can result in a small fraction of smokers having roentgenograms that are interpreted as abnormal at the level of the subjects in this study (1/0-1/2). Because the subjects were selected from a larger population based on the presence of an abnormality on the chest roentgenogram, it is not clear that the interstitial fibrosis found in the group with interstitial fibrosis and COPD is entirely the result of asbestos exposure. Even a low prevalence of abnormal roentgenogram due to smoking could produce the small number of subjects evaluated in this study. The subjects studied represent a mixture of individuals with interstitial changes on roentgenogram due to asbestos exposure, cigarette smoking and possibly other exposures. A great deal of caution must then be used in extrapolating the observations made in this study to other populations defined on the basis of their asbestos exposure alone.

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REFERENCES


New Recommendations for Standardizing Spirometry

The November 1987 issue of the *American Review of Respiratory Disease* includes an updated American Thoracic Society (ATS) Statement on the Standardization of Spirometry. Physicians and other health care professionals who purchase or use spirometers should review this new document carefully.

Spirometric tests are becoming increasingly important to diagnose disease, determine the severity of disease, decide whether treatment is necessary or effective, evaluate surgical risk and assess when an individual is too impaired to work or if the work-place has caused impairment. Inaccurate spirometric data can be costly to the patient and to society.

A valid spirometric test requires that several factors be considered. The ATS has made recommendations in the following areas:

1. Equipment selection and testing. Selecting adequate equipment is pivotal to acquiring accurate spirometric results. The ATS Statement makes recommendations about selecting and testing spirometers. We are concerned that only 27 of 53 (51 percent) contemporary devices met the new recommendations in a recent test. While the computers that have become an integral part of almost every spirometer can improve the accuracy and efficiency of testing, software errors are still a major problem. The Medical Devices Act of 1976 has not eliminated the manufacture and sale of “inaccurate” spirometers. The Food and Drug Administration (FDA) has classified spirometers as class II devices which should meet performance standards, but has not yet developed a performance standard. As a result, under section 510k of the Act, each new spirometer manufacturer must prove only that the new device is substantially equivalent to a previously manufactured spirometer. This, coupled with clinicians' beliefs that they do not need the accuracy of a clinical pulmonary function laboratory, has allowed some low quality spirometers into the market.

The responsibility for accurate spirometer data starts with the decision about which spirometer to purchase. Before buying a spirometer, the manufacturer should be asked to provide documented...