

Medical Examination for Asbestos-Related Disease

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There are millions of workers whose exposure to asbestos dust prior to the implementation of asbestos regulation and improved control measures places them at risk of asbestos-related disease today. In addition, workers are still being exposed to significant amounts of asbestos, when asbestos materials in place are disturbed during renovation, repair, or demolition. Given the continued presence of asbestos-containing materials in industrial, commercial, and residential settings throughout the U.S., a sizeable population remains at risk of asbestos-related disease.

This article reviews the health effects associated with exposure to asbestos and delineates the steps necessary for the comprehensive screening and clinical assessment for asbestos-related disease, in order to assist physicians in identifying and preventing illness associated with exposure to asbestos among their patients. Am. J. Ind. Med. 37:6–22, 2000. © 2000 Wiley-Liss, Inc.

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INTRODUCTION

The National Institutes of Health in 1978 estimated that 8–11 million individuals had been occupationally exposed to asbestos in the U.S. since the early 1940's [NIH, 1978]. There are no more recent governmental estimates of the population at risk from work-related exposures. Approximately 4.5 million workers were employed in U.S. naval and civilian shipyards during World War II, a great many of whom were heavily exposed to asbestos [Nicholson et al., 1982]. From 1890 to 1970, some 25 million tons of asbestos were used in the United States, approximately two-thirds of which were used in the construction industry. More than 40,000 tons of fireproofing material, containing 10–20% asbestos by weight, were sprayed annually in high rise buildings in the period from 1960 to 1969 [Selikoff, 1980]. Much of this material remains in buildings,

factories, and homes. During the renovation, repair and demolition of these structures, exposure to asbestos dust becomes a hazard, especially for those in the construction trades.

In many countries, especially in the developing world, the use of asbestos has continued and, in some cases, has increased while the use of asbestos in the United States has declined since the late 1970s [Nicholson, 1997]. Despite the decrease in the U.S., approximately 3.2 million workers who work in the construction of new buildings, building renovation, maintenance, and custodial work have sufficient exposure to asbestos to be covered by the current OSHA standard for the construction industry [U.S. Department of Labor, 1995]. A partial list of occupations that place workers at risk for asbestos-related illness is presented in Table I.

Asbestos inhalation is recognized as a cause of pulmonary fibrosis (asbestosis), pleural scarring (localized and diffuse), rounded atelectasis, and benign pleural effusion [Selikoff, 1980]. Exposure to asbestos dust also causes lung cancer and diffuse malignant mesothelioma of the pleura and peritoneum. Exposure at high concentrations has also been associated with cancer of the gastrointestinal tract, kidney, pancreas, and larynx [Selikoff and Seidman,

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TABLE I. Occupations with Significant Exposure to Asbestos (Partial List)

Acoustic product installers
Asbestos cement makers, users
Asbestos grout makers, users
Asbestos millboard makers, users
Asbestos millers
Asbestos miners
Asbestos paper makers, users
Asbestos plaster makers, users
Asbestos products manufacturers
Asphalt mixers
Auto mechanics
Boiler makers
Brake lining repairers
Brake refabricators
Bricklayers
Carpenters
Chemical workers
Clay workers
Construction workers
Demolition workers
Drywall tapers
Electricians
Electrical wire makers
Firefighters
Gasket makers, users
Glass workers
Iron ore miners and millers
Insulators
Laborers
Machinery producers
Maintenance and custodial workers
Oil and gas extraction workers
Petroleum refinery workers
Primary metal industry workers
Pipecoverers
Pipefitters
Plumbers
Powerhouse workers
Railroad repair workers
Rubber makers
Reinforced plastic makers, users
Roofers
Sheet metal workers
Shipyards workers
Stationary firemen
Steamfitters
Stone workers
Talc miners
Textile workers
Tile makers, users
Transportation equipment repairers
Transportation workers
Turbine manufacturing workers

1991]. Each of the malignant and non-malignant effects of asbestos exposure may occur independently of the others [Rosenstock and Cullen, 1994].

Other than for mesothelioma, discussion of the diagnostic approaches to asbestos-related neoplasms will not be presented here, since the steps in the diagnostic evaluation for malignancies caused by asbestos are the same as for those cancer types that result from other causes. Although some discussion of the asbestos related cancers is included, the main focus of this article is the medical screening and clinical assessment of the non-neoplastic asbestos-related diseases.

Screening individuals to identify asbestos-related disease as early as possible is important in order to:

- Provide appropriate medical monitoring for individuals with asbestos-related disease.
- Identify opportunities for work place intervention to prevent further exposure where the potential for ongoing exposure exists.
- Provide referral for non-medical (compensation and disability) services for individuals with asbestos-related disease.
- Identify additional groups of workers at risk for asbestos-related disease.
- To introduce modifications of current exposure conditions, life style factors and smoking cessation, when appropriate.

MINERALOGY AND USE

The term asbestos refers to a group of six, naturally occurring, fibrous mineral silicates of magnesium and iron. Asbestos-containing rock is mined, crushed, and milled to obtain the fibrous material, which is subsequently processed further into finer fibers. Asbestos fibers are categorized into two groups: amphiboles (straight fibers) and serpentines (curly, bundled fibers) [Speil and Leineweber, 1969]. The amphiboles used commercially include amosite, anthophyllite, and crocidolite. Other amphiboles (tremolite and actinolite) are frequent contaminants of other silicates, including some vermiculites and talcs. Chrysotile is the only type of serpentine asbestos in commercial use and represents 95% of all asbestos which has been incorporated into commercial products in the United States [Selikoff and Lee, 1978].

The natural resistance of asbestos to heat and acid, its tensile strength, and its remarkable thermal, electrical, and sound insulating properties have led to its use in over 3,000 applications, including floor tiles, boiler and pipe insulation, roofing materials, brake linings, and cement pipes [Rom, 1992]. The commercial use of asbestos has resulted in the very wide distribution of asbestos in the environment.

All types of asbestos fiber are associated with the development of asbestos-related scarring [Bignon and Jaurand, 1983] and malignancies [Dement et al., 1983; Seidman et al., 1979; Wignall and Fox, 1982; Meurman et al., 1974; McDonald et al., 1997]. A debate has been evolving on the role of chrysotile in the causation of mesothelioma [Mossman et al., 1990; Nicholson, 1991; Harington, 1991; Huncharek, 1994; Churg 1993; Boffetta 1998]. In the U.S. to date, exposure to asbestos is regulated without distinction among the fiber types. There is compelling evidence that chrysotile is capable of causing both lung cancer [Dement et al., 1983] and mesothelioma [Smith and Wright, 1996]. Longer fibers ($>5\ \mu\text{m}$) of all types of asbestos appear to be more fibrogenic [Davis and Jones, 1988] and carcinogenic [Davis et al., 1986].

HISTORY OF REGULATION OF EXPOSURE

The first case of asbestosis was reported in England in 1907 [Murray, 1907]. By 1918, American and Canadian insurance companies refused to insure asbestos workers due to their increased number of illnesses [USDHHS, NIOSH, 1976]. In 1946, the American Conference of Governmental Industrial Hygienists (ACGIH) recommended guidelines for a maximum acceptable concentration of exposure to asbestos dust for workers. In 1960, the U.S. government required that contractors on government projects costing over \$10,000 adhere to the ACGIH limits on asbestos exposure.

In 1971, guidelines restricting exposure to asbestos in the U.S. became law under the new OSHA Act. By contrast, in the United Kingdom, asbestos exposure had already been regulated for forty years, after reports of asbestosis in English factory workers began to appear more widely in the medical literature [Selikoff, 1980]. At present, the permissible exposure limit (PEL) is 0.1 fibers/cc for an 8 h time-weighted average. This is a substantial reduction from the 1972 PEL of 2 fibers/cc.

Respiratory protection to reduce exposure to asbestos dust was rarely provided for U.S. workers before the 1970s. Prior to the passage of the OSHA Act, many workers were unaware that they were working with asbestos and had little or no knowledge of the possible health consequences. Unfortunately, exposure to asbestos continues today in many occupational settings. The recognition of the hazard and the understanding of the need to avoid exposure are far from universal among workers at risk.

ROUTE OF EXPOSURE

Asbestos-related disease, with the exception of asbestos warts, results from the inhalation of asbestos fibers into the upper airways and the lung. While it has been hypothesized that the swallowing of fiber-laden respiratory mucus leads to

the observed increased risk of gastrointestinal cancers in heavily exposed populations, animal experimental studies of long-term, high-level ingestion of asbestos fibers have failed to demonstrate a reproducible carcinogenic effect [Condie, 1983]. More recently, however, human epidemiological evidence indicating that ingestion of asbestos fibers can cause human disease has been reviewed, generally involving communities exposed to asbestos-contaminated drinking water. An associations between ingested asbestos fibers and cancer of the stomach and pancreas has been found with some degree of consistency [Kanarek, 1989].

Aerosols of asbestos fibers of varying diameters and lengths can be generated by the disturbance of asbestos-containing materials by mechanical forces in the process of mining, milling, and product manufacture, in the end use of the product, and by the disturbance of asbestos-containing materials in place. Once airborne, fine asbestos fibers remain in the air for many hours, even under still conditions. Air movement easily re-aerosolizes asbestos fibers which may have settled on surfaces.

PATHOGENESIS

On inhalation, the larger asbestos fibers are deposited in the nose and upper airway. Fibers with diameters in the range of 0.5–5 μm can penetrate deep into the recesses of the lung and deposit at the bifurcations of alveolar ducts [Brody et al., 1981]. Asbestos fibers that reach the airways undergo partial clearance by the mucociliary escalator or are transported into the interstitium. The presence of the fibers provokes the accumulation of alveolar macrophages in the alveolar ducts and the peribronchial regions adjacent to the terminal respiratory bronchioles, which become thickened by interstitial macrophages and fibroblasts. The alveolar epithelial cells are damaged by the fibers and by mediators released by alveolar macrophage after phagocytosis of free fibers. Over time, there is an increase in the volume of interstitial macrophages, polymorphonuclear leukocytes, fibroblasts and non-cellular matrix. This fibrotic process progresses, leading to a stiffened, smaller lung with diminished capacity for gas exchange [Rom, 1992]. Progression can occur even after external exposure has ceased, due to the retention of fibers in the lung and the consequent persistent stimulus to the inflammatory response [Sluis-Cremer and Hnizdo, 1989; Finkelstein, 1986]. Ongoing inhalation exposure will result in an increasing fiber burden in the lung, thereby increasing the risk for asbestos-related illness.

Asbestos fibers which are phagocytized by macrophages may be coated with an iron-containing mucopoly-saccharide matrix, forming an asbestos body or ferruginous body [Suzuki and Churg, 1969]. Only a small proportion (perhaps 1%) of fibers become coated [Suzuki and Churg, 1969], and there is evidence that amphiboles form asbestos

bodies more readily than chrysotile. The presence of asbestos bodies in lung tissue, broncho-alveolar lavage fluid, or in sputum, has been used as a marker of exposure, although there appears to be considerable individual variability in the propensity to form these structures. The finding of increased asbestos body concentrations in sputum ($\geq 1/\text{specimen}$) [Teschler et al., 1996], broncho-alveolar lavage fluid ($>1/\text{mL}$ of fluid) [De Vuyst et al., 1987], or lung tissue ($>1000/\text{g}$ wet tissue) [Sebastien et al., 1988] indicates a history of exposure to asbestos in excess of “background” and can support the diagnosis of asbestos-related disease [Rom, 1998].

Some asbestos fibers that penetrate into the interstitium of the lung migrate to the pleura, most likely by lymphatic channels. Some are distributed to other tissues in the body via the lymphatic circulation [Auerbach, 1980].

HEALTH EFFECTS

Asbestos-Related Non-Neoplastic Diseases

Pulmonary asbestosis

Pulmonary asbestosis is the diffuse, interstitial fibrosis in the lung parenchyma caused by the deposition of asbestos fibers (of all types) in the lung. The fibrosis results in a restrictive lung disease that generally becomes manifest clinically 15–20 years after the onset of exposure. Even short-term exposure (e.g., less than one month), if sufficiently intense, can result in asbestosis [Seidman et al., 1979].

The most prominent symptom of asbestosis is the insidious onset of dyspnea on exertion, with progression over time. Cough, either dry or producing a small amount of clear sputum, may be present. Chest pain, either pleuritic or aching in character, occurs in a small proportion of patients with asbestosis. On physical examination, end-inspiratory basilar rales that persist after cough may be heard. Clubbing of the fingers may occur in advanced fibrosis. The chest x-ray shows small, irregular opacities in the mid- and lower lung zones after sufficient fibrosis has accumulated, although the characteristic pathologic findings of interstitial fibrosis may be evident on microscopic examination of tissue well before the radiographic abnormalities become detectable [Selikoff, 1980].

Pulmonary function abnormalities include a restrictive impairment, with a decreased forced vital capacity (FVC), total lung capacity (TLC), and diffusing capacity ($D_L\text{CO}$). Small airway narrowing has been reported to accompany the interstitial fibrosis [Wagner, 1963], although this has been found as well among non-smoking workers exposed to asbestos whose chest x-rays were normal [Glencross et al., 1997; Wang et al., 1998]. Impaired gas exchange due to the

accumulation of interstitial scarring can lead to arterial oxygen desaturation, evident at first only during exercise.

There is often poor correlation among radiographic appearance, degree of dyspnea, and pulmonary function studies in individual cases [Selikoff, 1980]. Some patients with marked parenchymal abnormalities on x-ray may have no symptoms and normal pulmonary function. The converse may also be true, with symptoms seemingly out of proportion to the degree of radiographic abnormality. Population studies, however, demonstrate associations among these parameters [Bader et al., 1965; Miller et al., 1994].

In severe cases of asbestosis, respiratory impairment can lead to death [Peto et al., 1985; Selikoff et al., 1979]. When fibrosis becomes extensive, increased resistance to blood flow through the pulmonary bed may ensue, from obliteration of the vascular bed and pulmonary capillary constriction caused by alveolar hypoxia, with resulting pulmonary hypertension and compensatory hypertrophy of the right ventricle. Cor pulmonale (right heart failure) has been reported to be associated with severe cases of asbestosis [Lemen et al., 1980]. A list of the most common conditions in the differential diagnosis of asbestosis is presented in Table II. Most important of the conditions listed are idiopathic pulmonary fibrosis and congestive heart failure.

Pleural thickening

Pleural thickening or asbestos-related pleural fibrosis, either localized in discrete plaques or occurring as a diffuse fibrotic process involving the costophrenic angle, is the most common consequence of exposure to asbestos in the occupational setting [Becklake, 1994]. Evidence of pleural scarring usually appears 20 or more years after the onset of exposure to asbestos dust, and a latency of 30–40 years is not uncommon [Rom, 1992].

Histologically, the plaques appear as acellular deposits of collagen. Circumscribed pleural scarring more com-

TABLE II. Differential Diagnosis of Pulmonary Asbestosis—Most Common Other Causes of Interstitial Fibrosis

Idiopathic pulmonary fibrosis
Congestive heart failure (radiographic appearance)
Hypersensitivity pneumonitis
Scleroderma
Sarcoidosis
Rheumatoid lung
Other collagen vascular diseases
Lipoid pneumonia
Desquamative interstitial pneumonia
Other pneumoconioses

TABLE III. Differential Diagnosis of Asbestos-Related Pleural Thickening [Adapted from Rosenstock and Cullen, 1994]

Discrete	Diffuse
Chronic mineral oil aspiration	Chronic beryllium disease
Chest trauma	Collagen vascular diseases
Infectious processes (old TB, pneumonia)	Drug reactions
Lymphoma	Infection
Metastatic cancer	Loculated effusions
Mica and talc reaction	Mica and talc reaction
Myeloma	Sarcoidosis
Scleroderma	Silicosis
	Uremia

monly involves the parietal pleura and often can be found on the surfaces of the diaphragm. The pericardium and the mediastinal pleural surfaces may also be involved. Although non-calcified thickening is more prevalent, calcification of areas of pleural scarring, whether localized or diffuse, is frequently evident on the chest radiograph and is more prevalent with increasing time since the onset of exposure. The differential diagnosis of pleural thickening is presented in Table III.

When parietal and/or visceral pleural thickening deforms the underlying lung, rounded atelectasis or a pseudotumor may develop. These lesions are characteristically less than 2 cm in diameter, are adjacent to an area of pleural fibrosis, and often have a “comet’s tail” which extends from the hilum to the mass [Hillerdal, 1989]. Evaluation by comparison with old chest x-rays, or with CT scan of the chest will usually reveal these characteristic features and avoid unnecessary biopsies [Lynch et al., 1988]. Nevertheless, given the increased risk of lung cancer among asbestos-exposed workers, the diagnosis of rounded atelectasis should be made with appropriate caution [Rosenstock and Cullen, 1994]. In cases where findings are equivocal, the lesions should be biopsied.

In the past, pleural thickening was thought to represent only a marker of prior exposure to asbestos; but pleural thickening, even when circumscribed, has more recently been shown to impair lung function, measured by spirometry [Lilis et al., 1991; Schwartz et al., 1994] or by exercise testing [Picado et al., 1987].

Diffuse pleural scarring is associated with a reduced FVC and D_LCO , even in the absence of radiographically apparent interstitial fibrosis [McLoud et al., 1985]. Entrapment or encasement of the lung, leading to pulmonary impairment and death, has been described in severe cases [Miller et al., 1983; Kee et al., 1996].

Both asbestosis and asbestos-related pleural fibrosis can be detected with greater sensitivity by CT scanning, especially by high resolution CT (HRCT) scan [Gamsu,

1991]. This technique may be useful in resolving cases that are equivocal on the plain chest radiographs [Harkin et al., 1996]. The radiation exposure, time, and cost involved in performing a delimited HRCT scan at the base of the lungs have decreased, making the CT scan more accessible as a tool for early detection.

Benign asbestotic pleural effusions

These effusions may occur within the first 10 years of the onset of exposure and may, therefore, be the first manifestation of asbestos-related illness. The diagnosis is one made by exclusion of other causes: cultures of pleural fluid are negative, and pathological examination reveals no malignant cells. Spontaneous resorption generally occurs within several weeks, but thoracentesis, including percutaneous pleural biopsy, is frequently performed for diagnostic purposes and for relief of pleuritic chest pain and/or dyspnea. Many patients, however, are asymptomatic [Gaensler and Kaplan, 1971]. There is evidence that diffuse pleural thickening may be the sequel of benign asbestotic effusions following their reabsorption [McLoud et al., 1985]. This form of pleural scarring may appear earlier than circumscribed pleural thickening.

Asbestos “warts”

These are small, cornified lesions on the hands caused by the penetration of the skin by asbestos fiber. These appear within 10 days of skin exposure and have been described as feeling like the foreign body reaction to a splinter [Alden and Howell, 1944]. There is no evidence that these local reactions are associated with an increased risk of skin cancer.

Asbestos-Related Cancers

Lung cancer

Lung cancer is the most common asbestos-induced neoplasm [Selikoff, 1980] and is the principal cause of death from asbestos in developed countries [Kilburn, 1998]. Reports of lung cancer in asbestos-exposed workers first appeared in 1935 [Lynch and Smith, 1935; Gloyne, 1935]. A latency of 20 or more years from onset of exposure to diagnosis has been shown for asbestos-related lung cancer. Over one third of the cancer deaths among heavily exposed asbestos insulators were due to lung cancer [Selikoff and Seidman, 1991].

Lung cancers associated with asbestos exposure are similar in cell type and histological features to other primary cancers of the lung. A predominance of adenocarcinomas has been reported in a number of studies, but all cell types of bronchogenic cancers are seen in excess [Rom, 1998]. Lung

cancers occur with increased frequency in all locations of the lung following exposure to asbestos, but have been reported to occur with the greatest frequency peripherally in the lower lung zones [Kannerstein and Churg, 1972]. Recent studies of lung cancer distributions by cell type and lobe of origin found no difference in anatomical site or histological characteristics between the cancers associated with asbestos exposure and those related to cigarette smoking [Brodtkin et al., 1997].

The role of interstitial fibrosis in the pathogenesis of asbestos-associated lung cancer is the subject of considerable debate [Warnock and Isenberg, 1986; Hughes and Weill, 1991; Hillerdal and Henderson, 1997]. Workers exposed to asbestos have been shown to have an increased risk of lung cancer, even when chest x-rays have shown no parenchymal fibrosis [de Klerk et al., 1996; Wilkinson et al., 1995; Finkelstein, 1997]. Studies have demonstrated that histologically evident pulmonary interstitial fibrosis may be present in cases of lung cancer where the chest radiograph is normal [Kipen et al., 1987], leaving the question of the role of “occult” interstitial fibrosis unresolved. As a practical matter, it is not necessary to demonstrate asbestosis on the chest x-ray or in biopsied tissue in order to attribute a causal role to asbestos in cases of lung cancer [Consensus Report, 1997].

Diffuse malignant mesothelioma

This is a tumor arising in the mesothelial cells of the pleura and peritoneum, described first by Klemperer and Rabin [1931]. Three histological patterns are recognized: epithelial, sarcomatous, and mixed or biphasic [Suzuki, 1980]. The great majority of patients with mesothelioma have a history of exposure to asbestos, and this has led to its description as a “signal neoplasm” because of its rarity in the absence of exposure to asbestos [Selikoff, 1980].

Among asbestos insulators, over 9% of all deaths were due to malignant mesotheliomas [Selikoff and Seidman, 1991], whereas lifetime expected risk for death from mesothelioma is estimated at 0.05% for the 1955–1959 male birth cohort in the U.S. [Price, 1997]. A latency of 20 years or more is again observed, with most mesothelioma deaths occurring more than 30 years from onset of asbestos work [Selikoff and Seidman, 1991]. The risk of mesothe-

lioma appears to increase in proportion to the third or fourth power of time elapsed since onset of exposure [Finkelstein, 1991].

Diffuse malignant mesotheliomas generally spread rapidly over the surfaces of the thoracic and abdominal cavities and organs, with little invasion of the organs involved. The disease often presents with chest pain and dyspnea, frequently due to pleural effusions, which prompt initial medical attention. The diagnosis is made on the basis of histological examination of cell blocks prepared from pleural fluid or, more commonly, tissue obtained by closed pleural biopsy or by thoracoscopy. Immunohistochemical staining and/or ultrastructural assessment using electron microscopy is often necessary for definitive diagnosis.

Interaction with Cigarette Smoking

Cigarette smoking and exposure to asbestos dust have been shown to interact in a multiplicative (or synergistic) fashion in the causation of lung cancer [Hammond et al., 1979]. In a study of a large group of heavily exposed asbestos insulators, Hammond et al. [1979] found a standardized mortality ratio (SMR) of 5 for lung cancer for non-smokers and 53 for smokers, compared to lung cancer mortality among the American Cancer Society’s large cohort of non-smoking, blue-collar workers occupationally exposed to dust, fumes, gases, chemicals or radiation, but not exposed to asbestos. The SMR for lung cancer among blue-collar cigarette smokers not exposed to asbestos was 11. These results are summarized in Table IV. Other studies have shown a more than additive but less than multiplicative interaction between cigarette smoking and asbestos in the risk of lung cancer [Berry et al., 1985; Saracci, 1987]. The increase in lung cancer risk is proportionate to the degree of exposure to asbestos and the cigarette smoking “dose” [Vainio and Boffetta, 1994]. Cessation of smoking among asbestos-exposed workers has been shown to be associated with a decreased risk of lung cancer, although the risk never falls to the level of never-smokers [Hammond et al., 1979].

The malignancies seen in significant excess among asbestos insulators [Selikoff and Seidman, 1991] other than lung cancer that have been shown to occur at even higher rates among cigarette-smoking asbestos insulators included

TABLE IV. Interaction between Smoking and Asbestos in Lung Cancer Mortality [Hammond et al., 1979]

Group	Exposure to asbestos	Cigarette smoking	Death rate^a	Mortality ratio
Controls	No	No	11.3	1.00
Asbestos workers	Yes	No	58.4	5.17
Controls	No	Yes	122.6	10.85
Asbestos workers	Yes	Yes	601.6	53.24

^aper 100,000/year.

cancers of the esophagus, oropharynx, and larynx. Smoking appears to have no influence on the risk of mesothelioma or cancers of the stomach, colon/rectum, and kidney among asbestos-exposed workers [Hammond et al., 1979].

Smoking has been associated with a greater profusion of irregular opacities evident on chest radiographs among men with asbestosis [Kilburn et al., 1986]. There is considerable evidence for an enhancing effect of smoking on the presence and profusion of radiographically evident, small, irregular parenchymal opacities. There has been debate, however, about the ability of smoking, without exposure to asbestos, to produce the appearance of such interstitial opacities [Blanc, 1991]. Cigarette smoking among asbestos insulation workers has been shown to increase the risk of death from asbestosis [Hammond et al., 1979].

COMPONENTS OF THE MEDICAL EVALUATION

The following elements should be included in the assessment of an individual for asbestos-related disease. The histories, physical examinations, and laboratory assessments described should be viewed as comprising the necessary, but minimum, content of such evaluations. More specific investigation regarding exposure to asbestos and other job-related hazards should be tailored to the conditions of each trade.

Medical History

In addition to a general medical history, questions relating to respiratory symptomatology, the development of dyspnea, and loss of exercise tolerance are important to identify symptoms resulting from asbestos-related disease.

Dyspnea

The key questions on dyspnea include the date of onset, whether gradual or sudden in onset, and if shortness of breath has progressed. It is useful to note if progression is gradual or episodic, e.g., following respiratory infections. A history of orthopnea and paroxysmal nocturnal dyspnea should be sought. Dyspnea on exertion, beginning gradually over years, is typical in patients with asbestosis, but not always present [Selikoff, 1980].

If the patient is still working, inquiry should be made about the level of physical activity on the job, i.e., whether there has been a loss of capacity to carry out certain strenuous tasks that were performed previously without difficulty, beyond what would be anticipated from the effect of ageing alone. The examiner should ask whether restrictions have been placed by an employer on the individual's working conditions (e.g., restriction from exposure to asbestos or from use of a respirator).

For all patients, an assessment should be made of the capacity to perform household activities and to engage in recreation that might entail physical effort. Examples of what the patient can no longer do should be recorded. An inquiry on the effect of dyspnea on sexual activity may reveal a history of shortness of breath not previously mentioned by the patient.

A respiratory questionnaire developed by the Medical Research Council (MRC) of Great Britain to assess symptomatology associated with obstructive airway disease and chronic bronchitis has proven useful as a measure of dyspnea in a number of asbestos studies [Medical Research Council, 1960]. The questionnaire, incorporated into the current OSHA standard [USDOL, 1994], provides a semi-quantitative index of exercise tolerance. Inquiry about the number of flights of stairs the patient can climb without stopping for breath can offer additional semi-quantitative information.

Cough

Cough, not associated with respiratory infections or chronic bronchitis, is a symptom of asbestosis, and information related to the date of onset, the duration if the cough is episodic, and whether the cough produces sputum should be obtained. The cough found in asbestosis is typically a dry cough [Rosenstock and Cullen, 1994]. If there is sputum production, the quantity and character of the sputum should be noted. Any history of hemoptysis, which may be the first sign of lung cancer, should prompt further clinical assessment, which may include sputum cytology and bronchoscopy.

Chest Pain

Chest pain, not associated with physical effort or emotional stress, at times provoked or worsened by respiratory motion, occurs in a small proportion of patients with pleural scarring [Miller, 1990].

Respiratory Infections

A history of unusually frequent or persistent respiratory infections should be sought. Previous medical evaluations for respiratory (and cardiac) problems should be recorded, and results of earlier diagnostic studies included.

Past Medical History

In obtaining the past medical history, the examiner should ask specifically about any confounding illness, such as tuberculosis, pneumonia, chest trauma (especially rib fractures), pleuritis, pleural effusion, cancer of any type, emphysema, childhood or adult onset asthma, chronic

bronchitis, heart disease, collagen-vascular or rheumatological diseases (including systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, and scleroderma or systemic sclerosis), and sarcoidosis. A history of other serious illnesses, hospitalizations, or surgery should be obtained. Drug allergies and medications should be noted. The patient should be asked about routine vaccinations, especially against influenza and pneumococcal pneumonia.

Family Medical History

The individual's family history is useful, especially the parents' ages, family illnesses, and household members' occupations. A family history of lung cancer, lung fibrosis, and other non-neoplastic lung diseases should be sought. Specific inquiry should be made about the possibility of exposure to asbestos dust brought from the workplace into the home by another member of the household, and about residence near a facility that mined asbestos, or produced or used asbestos products.

Review of Systems

There should be inquiry into the presence, onset date, frequency and severity of symptoms that might be manifestations of asbestos-related malignancies. Thus, the patient should be asked about soreness in the mouth or throat, hoarseness, lymph node enlargement, dysphagia, dyspepsia, abdominal pain or swelling, loss of appetite, unintended weight loss, change in bowel function, blood in the stool or melena, recent hernia, and hematuria.

Smoking History

The patient's smoking history, the age at which smoking began, the age(s) at which smoking ceased (including temporary periods of abstinence lasting at least six months), the average number of cigarettes smoked per day, the maximum number smoked regularly, and the smoking status of household members, including the spouse, are important. Pipe and cigar smoking should be included in the history, as well as the use of smokeless tobacco (chewing tobacco or snuff).

Lifetime Occupational History

The lifetime occupational history should elicit information regarding the duration and intensity of past and current exposures to asbestos. Since air monitoring data are rarely available for exposures two and three decades ago, the examiner should obtain descriptions of job tasks performed which may have generated asbestos aerosols, as well as their frequency and duration. In the construction environment especially, work performed in areas where other trades (e.g.,

pipe coverers, pipe fitters, plumbers) may have disturbed asbestos materials can result in clinically significant "bystander" exposures. The nature and sufficiency of local ventilation and the availability and use of personal protective equipment should be recorded.

The examinee should be asked if he or she was aware of the possible hazards of asbestos exposure at the time the work in question was performed. To elicit information on possible contamination of the worker's car or household, the examinee should be asked if he or she changed work shoes and clothing at the end of the shift, if there were shower facilities available and if he or she used them, and whether his or her work clothing was brought home to be laundered. Questions to ask regarding exposure to asbestos include:

- Job title, tasks, and dates of employment.
- Exposure to dusts.
- Mixing, cutting, applying, spraying or removing asbestos materials.
- Work in areas where other trades generated asbestos dust.
- Use of personal protective equipment.
- Ventilation of work areas.
- Awareness that asbestos is a health hazard.
- Whether clothes were changed at home or at the workplace.
- Whether work clothes were washed at home and by whom.
- Whether shower facilities were used at the workplace.
- Military service—especially aboard ship or in ship construction or repair.
- Other jobs or hobbies associated with asbestos exposure.

It is of value to determine the dates of employment in estimating lifetime exposure to asbestos, since the use of asbestos in insulation materials decreased rapidly in the late 1970s, markedly reducing exposures in new construction projects. A history of work in specific asbestos-exposed trades should be asked about, if not already covered in earlier questioning: shipyard experience, brake repair, insulation work, and construction work.

Questions should also be directed toward exposure to other chemical, physical, and ergonomic hazards on the job, and information to be obtained with similar detail when hazards are identified. For jobs without asbestos exposure, the dates and job tasks, as well as hazardous agents encountered should be recorded, with appropriate detail regarding frequency, duration, and conditions of any exposure and/or symptoms experienced. Military service should be recorded, with specific questions regarding service aboard ships (Navy, Marine Corps, and Merchant Marines), the job tasks performed, and whether asbestos exposure may have occurred.

Physical Examination

While focused on the respiratory, cardiovascular, and gastrointestinal systems, the physical examination should also include the elements of a general examination, since the clinical contact with an asbestos-exposed individual may represent a singular opportunity to diagnose conditions that are not related to asbestos and to provide secondary and tertiary preventive interventions.

The blood pressure and the pulse rate and rhythm should be measured. The presence of tachypnea or respiratory distress at rest or with the effort of dressing/undressing should be noted. The assessment should include examination of the oropharynx for possible pre-malignant or malignant lesions. The neck should be palpated for cervical adenopathy. The examination of the chest should include inspection of the chest contour, percussion for dullness and assessment of the mobility of the diaphragm, and auscultation for adventitious or decreased breath sounds. End-inspiratory basilar rales, which persist after cough, are typical findings in asbestosis [Murphy et al., 1984]. In addition to the usual cardiac examination, the assessment of the relative intensity of the second heart sound at the pulmonic area in comparison with the aortic area is important, because pulmonary hypertension is associated with asbestosis [Selikoff, 1980]. The abdominal examination is performed to detect the presence of masses, organomegaly, and intra-abdominal fluid. While digital rectal examination is insensitive as a screening approach to colorectal cancer, a fecal specimen often can be obtained and assayed for occult blood at this time. The size and contour of the prostate should be assessed, because many individuals screened for asbestos-related disease are in the age groups in which the prevalence of prostate cancer rises rapidly. Since clubbing of the distal phalanges can result from asbestos-induced disease, albeit infrequently, the extremities should be examined. The distal pulses should be examined to assess peripheral circulation.

Diagnostic Evaluation

Chest x-ray

The chest x-ray has traditionally been the most useful diagnostic tool in the initial evaluation for asbestos-related lung disease. Asbestosis is characterized by the appearance on the standard posterior-anterior chest radiograph of small irregular opacities in the mid- and lower lung zones, reflecting the presence of parenchymal fibrosis. The technique for taking the chest x-ray and the approach to classifying the film for pneumoconiosis should follow the International Labor Organization [1980] guidelines, utilizing comparison with the standard films provided by the ILO. The guidelines and standard films can be obtained from the

International Labor Public Center Office in Waldorf, MD (ILOpub@tsacol.com).

In addition to the posterior anterior view recommended by the ILO, radiological examination should include a lateral view of the chest. While the lateral chest x-ray provides only limited additional information on asbestos-related scarring, the presence of malignancy is often detectable only on lateral projections, since lesions may lie behind the heart, mediastinal, and diaphragmatic shadows on the posterior anterior film. Right and left anterior oblique views may help define costal pleural abnormalities and will detect some pleural changes not evident on posterior anterior films. Additional views can be requested if necessary in individual cases. Chest x-rays should be read promptly for abnormalities indicating the need for urgent clinical evaluation.

The chest x-ray should be assessed definitively for asbestos-related or other pneumoconiotic changes by a NIOSH-certified "B" reader (a physician who has had specific training and has passed an examination in the interpretation of chest x-rays for pneumoconioses) or other physician experienced in the radiological assessment of asbestos-related disease. The x-ray appearances should be classified according to the ILO-1980 schema.

Computed tomography (CT) of the chest

Computed tomography scanning of the chest, especially the scans utilizing high resolution (HRCT) technique, has demonstrated effectiveness in revealing the presence of pleural thickening in areas of the pleura not readily accessible to the chest radiograph, even when oblique views are available. [Harkin et al., 1996; Neri et al., 1994]. This diagnostic modality has proven useful in distinguishing pleural fibrosis from accumulations of sub-pleural fat, which can mimic pleural fibrosis. The use of HRCT in the assessment of parenchymal fibrosis has demonstrated its superior sensitivity, in comparison to the plain chest x-ray when histological examination was used as confirmation of the diagnosis [Gamsu and Aberle, 1995]. HRCT has particular utility in resolving diagnostic questions posed by equivocal posterior-anterior views, especially in the presence of clinical findings such as dyspnea, basilar rales, a restrictive pattern evident on pulmonary function testing, or a markedly reduced diffusion capacity. This can be accomplished by obtaining "cuts" from only the lower lung zones, with considerable savings in radiation exposure, time, and cost.

Recently, low-radiation-dose "spiral" CT scanning of the chest has been shown to detect non-calcified pulmonary nodules in cigarette smokers with a three-fold greater sensitivity, and to detect lung cancer with a six-fold greater sensitivity than the standard chest radiograph [Sone et al., 1998; Henschke et al., 1999]. While results

are preliminary and an effect on survival rates has not yet been demonstrated, the ability of this technique to find malignant lesions at an earlier stage in their development holds promise for improving the outcome of surgical intervention.

Pulmonary function testing

Routine spirometry can offer important diagnostic information. Measurement of the forced vital capacity (FVC), the forced expiratory volume in the first second (FEV₁), the FEV₁/FVC, and the forced expiratory flow rate at mid-expiration (FEF₂₅₋₇₅) should be obtained, following the guidelines published by the American Thoracic Society [Gardner, 1987], including frequent calibration of the spirometer.

For a more complete assessment of pulmonary functional status, a D_LCO should be performed, offering an assay of the lung's ability to accomplish gas exchange. This may be especially useful when routine spirometric studies reveal no or minimal abnormalities, despite the patient's report of dyspnea. Measurement of static lung volumes should be obtained by body plethysmography or helium dilution, to evaluate the contribution of air trapping to decrements in the forced vital capacity.

Typically, asbestos-related disease causes a restrictive pattern on pulmonary function tests [Bader et al., 1965; Lerman et al., 1988]. A forced vital capacity (FVC), a total lung capacity (TLC), and/or a D_LCO less than the 95% confidence lower limit, suggest the presence of an interstitial fibrotic process consistent with asbestosis. Predicted values are most often based on age, sex, race, and height. Predicted values based also on smoking status are especially useful for evaluation of the D_LCO [Miller, 1986]. Pulmonary physiology laboratories are increasingly reporting 95% confidence intervals for spirometric parameters, D_LCO, and static lung volumes, which provide a statistically more valid basis for defining abnormality than the commonly employed lower threshold values of 80% of predicted and should be used where available. Similarly, a decreased resting PaO₂, or a fall in the PaO₂ with exercise, indicates impaired gas transfer consistent with, but not specifically diagnostic of, parenchymal fibrosis and may result from emphysema as well. Constriction of respiratory bronchioles, with decreased expiratory flow rates at low lung volumes (FEF₂₅₋₇₅), may be the earliest functional impairment [Becklake et al., 1970].

In individuals whose severity of dyspnea exceeds what might be expected from either normal or minimally abnormal pulmonary function test results, cardiorespiratory stress testing (exercise testing) may provide valuable information regarding the individual's ability to meet varying levels of exercise demand. This enables the physician to comment on the patient's fitness for any particular work

task. Patterns of test results can assist in distinguishing exercise intolerance from pulmonary disease, cardiac disease or deconditioning. Except for research purposes, exercise testing is generally reserved for markedly dyspneic patients who do not demonstrate severe impairments on routine spirometric testing.

Routine blood, stool, and urine tests

For patients who do not obtain routine medical care, as is the case for many in the asbestos-exposed trades, the asbestos screening or surveillance examination may be the only opportunity to identify other, treatable diseases. To screen for other medical problems, a complete blood count (CBC) and blood biochemistry screening panel, including measures of kidney function, liver function tests, cholesterol and lipid levels, may be obtained, along with an urinalysis with microscopic analysis of the sediment. The U.S. Prevention Services Task Force [Public Health Service, 1996], however, currently recommends that only a serum cholesterol level and stool analysis for hidden blood be obtained on an annual basis.

Stool specimens collected on three separate days should be tested annually for occult blood, since some studies of populations heavily exposed to asbestos have shown an increased risk of gastrointestinal tract malignancies, including the oropharynx, esophagus, stomach, colon, and rectum. [Selikoff, 1991]. Periodic colonoscopy is recommended as an alternative approach to screen for colorectal cancer.

Diagnostic Criteria

Pulmonary fibrosis (pulmonary asbestosis)

Pulmonary asbestosis is defined as the pneumoconiosis caused by the inhalation of sufficient asbestos fibers to cause diffuse interstitial fibrosis within the lung. Most commonly, the physician must diagnose pulmonary asbestosis in the absence of a histological assessment of lung tissue. The case definition, therefore, is a clinical one, since subjecting a patient to lung biopsy is rarely warranted, except when another, potentially treatable, cause of evident interstitial fibrosis is suspected, when another cause of interstitial lung disease is suggested by evidence on HRCT scanning, or when the clinical course is atypical (e.g., with rapid progression).

To arrive at the diagnosis of pulmonary asbestosis, the minimum criteria include: (1) a reliable history of occupational or other significant exposures to asbestos (either direct or "bystander"), with onset of exposure 15 or more years earlier (appropriate latency), and (2) a chest x-ray showing small, irregular parenchymal opacities in the mid and/or lower lung fields with a profusion classified as abnormal (1/0 or greater) by a "B" reader or experienced

reader, using the ILO-1980 guidelines, or a HRCT scan showing diffuse interstitial fibrotic changes at the lung bases. High resolution CT scanning can assist in the assessment of equivocal cases and is increasingly gaining favor as a screening tool. It should be noted that latency periods shorter than 15 years, especially in heavily exposed individuals, have been reported.

Valuable support for the diagnosis of asbestosis can be derived from a history of progressive dyspnea or loss of exercise tolerance, most often gradual in onset. Many patients with radiographically apparent asbestosis report no shortness of breath, however. This has been seen especially in screening examinations of active workers in asbestos-exposed trades.

In early asbestosis, pulmonary function abnormalities may appear before radiographic evidence of asbestos-related scarring. In addition, it has been shown that diffuse interstitial scarring may be evident on pathological examination of lung tissue even when the chest x-ray appears normal [Kippen et al., 1987]. It is difficult, however, without the benefit of tissue pathology, to make a diagnosis of parenchymal asbestosis in the absence of radiographically demonstrable interstitial fibrotic change either on the plain film or on HRCT.

Asbestos-related pleural fibrosis

To arrive at the diagnosis of asbestos-related pleural fibrosis, the minimum criteria include a history of occupational exposure to asbestos (either direct or “bystander”), with onset of exposure 15 or more years earlier, and a chest x-ray showing thickening and/or calcification of the costal, diaphragmatic or mediastinal pleura. Latency periods shorter than 15 years have been reported, but are uncommon. These areas of scar formation may be restricted to discrete “plaques” involving the costal or diaphragmatic pleura, or may be extensive, diffuse areas of thickening, usually involving blunting of the costophrenic angle on the same side. Calcification of areas of pleural thickening, whether “circumscribed” or diffuse, is commonly found in asbestos-related pleural fibrosis [Hillerdal and Lindgren, 1980]. The presence of bilateral pleural plaques is virtually pathognomonic of asbestos-related pleural disease, but unilateral plaques are common. The differential diagnosis of pleural thickening is presented in Table III.

Benign asbestos effusion

Benign pleural effusions are recognized to occur with increased frequency in asbestos-exposed populations. The criteria for the diagnosis of this entity include a prior history of asbestos exposure, absence of any other predisposing cause for effusion, and spontaneous remission. The effusions may be unilateral or bilateral, and there may be a

single episode or multiple recurrences, on the same or contralateral side. Pleuritic chest pain and/or dyspnea often accompany the onset of fluid accumulation, and there may be fever, elevation of the sedimentation rate and leukocytosis. The fluid is exudative in character and often hemorrhagic, but can be serous or fibrinous. Cultures and cytological examination are negative [Gaensler and Kaplan, 1971].

The diagnosis is, in fact, arrived at by exclusion of other possible causes, with underlying malignancy representing the greatest concern. Cytological examination of aspirated fluid and/or pleural biopsy, usually by thoracoscopy, should be performed to rule out lung cancer and malignant mesothelioma. Continuing observation, with repeat chest x-rays within 2–3 months, of the patients with an apparently benign effusion is necessary to ascertain that the resolution of the effusion persists. There is widespread agreement that “clinically silent” asbestos-related pleural effusions are the precursors of the development of diffuse pleural thickening [Epler et al., 1982].

NOTIFICATION

Notification of Evaluation Results to Examinees

Abnormalities found on chest x-ray, physical examination, or laboratory testing which require urgent clinical assessment should be reported to the examinee as promptly as possible. Often, direct communication with the patient’s primary care physician is warranted.

A summary report should be sent to each examinee, which includes the abnormal findings on physical examination and laboratory evaluation and states the diagnostic conclusion, utilizing both medical terminology and appropriate explanatory language. When applicable, a statement should be made regarding the relationship of the asbestos-related abnormality to the examinee’s history of exposure to asbestos and to respiratory symptoms reported.

Advice to stop smoking should be included in every smoker’s notification letter. Advice never to resume smoking should be offered to ex-smokers. The markedly increased risk of pulmonary functional impairment and lung cancer posed by the synergistic effect of asbestos exposure and cigarette smoking can be emphasized, as well as the considerable reversibility of the smoking-related risk. Assistance in smoking cessation should be offered. The necessity to avoid further exposure to asbestos and other pulmonary irritants should be advised as well.

Examinees who manifest asbestos-related abnormalities on chest x-ray should be informed about the possibility of progression of parenchymal and pleural scarring, even if external exposure has ceased. Those with significant asbestos exposure and no signs of asbestos-related disease

should be advised of the possibility that they may develop asbestos-related scarring in the future. Both groups should be informed that they are at increased risk of the cancers caused by asbestos and that annual medical monitoring is advisable.

Notification to Employers

The OSHA standard for exposure to asbestos in construction requires that the physician provide to the employer written opinions regarding: (1) whether the employee has another medical condition that would place the employee at an increased risk of health impairment from exposure to asbestos; and (2) any recommended limitations on the employee or on the use of personal protective equipment such as respirators.

The physician must also notify the employer that the employee has been informed by the physician of the results of the medical examination and of any medical condition that may result from asbestos exposure, including an increased risk of lung cancer from the combined effects of smoking and exposure to asbestos. The physician is not to reveal to the employer specific findings or diagnoses unrelated to asbestos exposure [USDOL, 1995].

OSHA ASBESTOS STANDARDS— FREQUENCY OF EXAMINATIONS

The OSHA's 1995 Asbestos Standard for the Construction Industry requires employers to provide medical surveillance for employees who work 30 days or more per year in Class I, II, or III jobs (which include tasks relating to the removal of thermal system insulation, flooring and roofing materials, or the disturbance of asbestos-containing materials during repair and maintenance) and for employees who are exposed at or above the short term exposure limit or the permissible exposure limit (0.1 fibers/cm³) [USDOL, 1995]. Medical surveillance is also required prior to the use of negative-pressure respirators.

According to the 1995 standard, beginning with the first year of exposure to asbestos, the medical surveillance examination must be performed annually by a physician and must include a medical and work history, completion of a standardized questionnaire, a physical examination, pulmonary function tests, and any other tests the physician feels is indicated. The cost of the medical surveillance is borne by the employer [USDOL, 1995]. Chest x-ray examinations are now up to the discretion of the evaluating physician.

By contrast, the earlier 1988 OSHA asbestos standard recommended chest x-rays based on the age of the employee and time since first exposure, taking the latency of asbestos-related diseases into account. The schedule recommended in the 1988 OSHA standard was as follows: for employees

with first exposure more than ten years earlier, chest x-rays were recommended annually for workers over the age of 45, once every two years for age 35–45, and once every five years for age 15–35. For workers whose first exposure was less than ten years earlier, chest x-rays were recommended at five year intervals [USDOL, 1988]. Given the latency of asbestos-related disease, this examination schedule better targets those workers who are at greatest risk for clinically detectable illness than a schedule requiring an annual examination starting from the onset of exposure.

While the 1995 OSHA standard focuses on current workers, it is prudent to screen previously exposed workers, including disabled and retired workers, household members exposed to dust brought home by workers in heavily exposed trades, and workers now in other industries. Individuals whose exposure to asbestos occurred as a consequence of working (or residing) in a building with asbestos-containing materials in place, but who have not themselves disturbed these materials nor have been present regularly when others performed maintenance or renovation work, should not be encouraged to undergo evaluation for asbestos-related disease. While any exposure to asbestos may increase the risk of asbestos-related malignancy (in proportion to the cumulative dose), the likelihood that such low-level, intermittent exposures will result in clinically detectable scarring lung disease or pleural fibrosis is minimal.

TREATMENT OF BENIGN ASBESTOS- RELATED DISEASE

There is no effective treatment available for parenchymal asbestosis. Measures used for patients with other forms of interstitial fibrosis, including steroids and colchicine, have not proven beneficial for asbestosis [Rom, 1998]. The hypoxemia associated with advanced fibrosis can be managed with oxygen administration, and cor pulmonale is treated as for other causes of right heart failure. For terminal patients with pulmonary failure due to asbestosis, a last resort option is lung or heart-lung transplantation [Wagner, 1994], although experience with this approach is very limited.

Asbestos-related, circumscribed pleural scarring may be associated with a loss of exercise tolerance [Lilis et al., 1991], but, as with asbestosis, no treatment for this condition is available, and in most cases, no treatment would appear warranted. In cases of extensive, diffuse pleural thickening with entrapment of the lung, pleurectomy may be necessary to permit lung expansion [Miller et al., 1983].

Benign asbestotic pleural effusions are treated as are effusions from other causes, with careful evaluation to rule out the possibility of malignancies by thoracentesis and cytological examination of the aspirate. In cases of multiple,

recurrent effusions, pleurodesis has been utilized to prevent further episodes [Weissberg and Ben-Zeev, 1993].

Treatment of Malignant Asbestos-Related Disease

Asbestos causes all of the common types of lung cancer (squamous cell, adenocarcinoma, small cell and large cell carcinomas). Treatment approaches include the usual methods used against lung cancer from any etiology. [Lordi and Reichman, 1993]. The treatment of lung cancer has had limited success. Overall, 5 year survival rates remain below 10%. Solitary, non-small cell carcinomas are more amenable to surgical resection, with 5 year survival rates as high as 30%.

The prognosis for a patient with mesothelioma is bleak. The overall median survival time after diagnosis is 8–12 months, with fewer than 10% alive at 2 years [Rom, 1998]. Treatment with surgery and chemotherapy (with or without radiation therapy) has been found, in an uncontrolled retrospective study, to prolong survival as compared to supportive care only [Huncharek et al., 1996].

There are indications that trimodality therapy, including surgery, chemotherapy, and radiation, has increased survival for some patients. [Kaiser, 1997]. Other treatments under investigation include oncogene and tumor-suppressor gene modification, immunotherapy, photodynamic therapy, and aggressive debulking combined with continuous hyperthermic peritoneal perfusion using cisplatin [Aisner, 1995; Lechner et al., 1997; Kaiser, 1997; Ma et al., 1997].

Other cancers related to asbestos exposure are also treated as per the usual standard of care for those malignancies.

MEDICO-LEGAL ASPECTS OF ASBESTOS-RELATED DISEASE

Informing a patient that he or she has developed a disease caused by asbestos has legal implications of which the health care professional should be aware, in order to protect the patient's rights under the law. Included among those rights is access to the workers' compensation system, designed to be a "no-fault", non-adversarial mechanism for redressing the medical and financial burdens associated with occupational disease and injury. Compensation for asbestos-related illness may take the form of medical care and/or monetary payment.

There are requirements regarding the time within which an affected worker must file a claim for compensation, whether via the workers' compensation system or through a product liability action, defined by the "statute of limitation". The communication to a patient that he or she has developed any asbestos-related illness marks the beginning of the time period (e.g., two years in New York State)

beyond which the claim can no longer be filed. It is the responsibility of the physician or other health care provider to be familiar with laws affecting access to workers' compensation and other legal remedies, and to inform the patient of the implications of the diagnosis. While it is not the role of the physician to encourage or discourage the filing of a workers' compensation claim (or a product liability action), the practitioner is likely to be the only source of information about the necessity to take such action in a timely fashion.

PREVENTION AND FURTHER MEDICAL SCREENING

Patients with significant asbestos exposure are advised that they are at increased risk of cancer and should obtain regular screening for asbestos-related disease. Given the advances in CT and HRCT techniques and decrease in cost, annual low-dose (spiral) CT imaging of the chest to detect small nodular densities [Henschke et al., 1999], with HRCT at the lung bases to image fibrotic changes, should be considered. It should be noted, however, that data are not yet available which demonstrate the effectiveness of the detection by CT of smaller lung masses in the reduction of lung cancer mortality.

Patients with asbestosis and/or pleural scarring are advised to obtain a pneumococcal vaccine, an annual influenza vaccine, and prompt treatment of respiratory infections. Annual fecal occult blood testing is recommended. The accuracy of detection of blood in the stool depends on testing within a day or two following streaking of the specimen. As an alternative to fecal blood testing, screening colonoscopy once every 7–8 years can be advised to patients over the age of 50 years. As mentioned above, smoking cessation counseling and treatment is very important.

A worker who shows evidence of asbestos-related scarring should be encouraged to eliminate all further exposure to asbestos, other fibrogenic dusts, and airborne irritants, to avoid additional injury to the lung. In many circumstances, this can be accomplished with the use of personal respiratory protection. At times, however, this may require discussion of the advisability of the worker's continuing in his or her trade, if further exposure cannot be avoided.

Reports of current exposure to asbestos, usually occurring during renovation, repair and demolition activities, should prompt industrial hygiene investigation of the worksite and work practices. Laws and regulations exist at the federal, state, and local level which prohibit activities that place workers and the general public at risk of exposure, although these may be unevenly enforced.

Training and educating of workers, supervisory personnel, and employers in the recognition of the hazard and

the health effects of asbestos inhalation are key to preventing further exposure and future disease.

All cases of asbestos-related parenchymal and/or pleural fibrosis in New York State must by law be reported to the Occupational Lung Disease Registry maintained by the New York State Department of Health, although physician compliance is problematic. Reporting of asbestosis is required by public health law in about half of the states in the U.S.

CONCLUSION

There are large numbers of workers whose exposure to asbestos dust prior to the implementation of asbestos regulation and improved control measures places them at risk of asbestos-related disease today. Active screening and surveillance for asbestos-related disease among such workers is an important public health intervention. In addition, workers are still being exposed to significant amounts of asbestos, when asbestos materials in place are disturbed during renovation, repair, or demolition. Given the ubiquity of asbestos-containing products, a sizeable population remains at risk of asbestos-related disease.

Screening exposed workers has multiple benefits. Earlier disease detection may make curative treatment possible, such as for some asbestos-associated cancers. Screening presents an opportunity for education on the health hazards of asbestos and for emphasizing the importance of eliminating further exposure. Prevention of disease can be achieved through the reduction of other risk factors, such as smoking. Workers with asbestos-related fibrosis achieve higher quit rates following smoking cessation counseling [Li et al., 1984; Humerfelt et al., 1998]. Screening is a mechanism for workers to gain access to medical care and appropriate follow-up treatment, and the diagnosis of illness related to asbestos exposure helps workers obtain medical monitoring and other compensation. Screening also assists in epidemiological surveillance of diseases caused by exposure to asbestos.

This article delineates the steps necessary for the comprehensive screening and clinical assessment of individuals for asbestos-related disease, in order to assist physicians in identifying and preventing illness associated with exposure to asbestos among their patients.

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