



Pulmonary Radiology

By Members of THE FLEISCHNER SOCIETY

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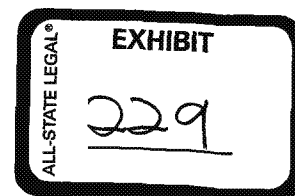
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PNEUMOCONIOSES: RADIOLOGY AND HIGH-RESOLUTION COMPUTED TOMOGRAPHY

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Pneumoconioses are diseases resulting from the accumulation of dust in the lungs. The pneumoconioses can be divided into two groups: the fibrogenic type, in which the dust is capable of causing a tissue reaction that may lead to fibrosis, and the "benign" pneumoconioses that occur after the inhalation of nonreactive dust.

Examples of the fibrogenic type include silicosis, coal worker's pneumoconiosis (CWP), and asbestosis. Examples of the benign type include lung responses following inhalation of iron, tin, and barium.^{1,2} The benign pneumoconioses may produce significant radiographic abnormalities, but they do not lead to symptoms or disability.

Abnormalities on the chest radiograph, and now on the computed tomography (CT) scan, are frequently accepted as the major evidence of dust exposure in worker's compensation situations and similar industrial litigation. The chest radiograph or CT scan is less accurate for the assessment of functional impairment from dust inhalation. The extent of high-resolution CT (HRCT) abnormality does, however, correlate with the results of pulmonary function studies. Radiographs and CT scans nevertheless provide a measure of the amount of dust retained in the lungs, especially in such conditions as asbestosis, silicosis, and CWP, which are discussed in this chapter.

INTERNATIONAL LABOR ORGANIZATION CLASSIFICATION OF THE PNEUMOCONIOSES

The International Labor Organization (ILO) classification of the radiographic appearances of the pneumoconioses is a standardized internationally accepted system that codifies the radiographic changes of the pneumoconioses in a reproducible manner.²⁻⁴ The classification is not intended for clinical practice but is used for epidemiologic studies or for evaluating pulmonary disability.² The advantage of this system is that it provides graphic and morphometric terms to describe diffuse lung patterns. The classification includes conventions of small rounded (nodular) and small irregular

(linear and reticular) opacities (Fig. 7-1). The small rounded opacities are classified according to the approximate diameter of the predominant opacity: *p*, up to 1.5 mm in diameter; *q*, 1.5 to 3 mm in diameter; and *r*, 3 to 10 mm in diameter. Small irregular opacities are divided on the basis of thickness and size: *s*, fine; *t*, medium; and *u*, coarse or blotchy irregular opacities. The irregular opacities also correspond in size to the nodular opacities.

The ILO scheme allows for quantification of the radiographic severity or "profusion" on a 12-point scale (Fig. 7-1). The scale contains four basic categories: 0, normal; 1, slight; 2, moderate; and 3, advanced. In addition, it recognizes the existence of a continuum and provides for additional grading. When there is no doubt, the readings are 0/0, 1/1, 2/2, and 3/3; if during the reading the category above or below is considered a serious alternative, this category is recorded after the slash, for example, 1/2. The distribution and extent of opacities are recorded in six zones. In addition, a convention is provided for large opacities that describes the conglomerate masses identified in some pneumoconioses. The most recent (1980) classification also includes a detailed categorization of pleural thickening, which is quantified and classified as diffuse or circumscribed (plaque).

To ensure consistency in the application of the ILO classification system, standard radiographs are needed for comparison. For epidemiologic surveys a high kilovoltage (peak) (110 to 150 kv[p]) technique, short milliamperage seconds, a fixed grid with a 10:1 grid ratio, and a focal spot no larger than 5 mm are recommended and will result in consistently good-quality radiographs.^{2,5}

COMPUTED TOMOGRAPHY

Although some of the features of the pneumoconioses can be detected with conventional CT techniques, in virtually all circumstances more precise information is obtained with HRCT. The elements that define the technique as high resolution include collimation of 2 mm or less (in our expe-

	R	mm	I	
p		-1,5		s
q		1,5 - 3		t
r		3 - 10		u

A

0		0/0
0		0/1
1		1/0 1/1 1/2
2		2/1 2/2 2/3
3		3/2 3/3 3/4

B

FIGURE 7-1 A, Small rounded opacities (p, q, r) and small irregular opacities (s, t, u). The diameter is in millimeters. The small irregular or linear opacities may be designated as fine (s), medium (t), or coarse or blotchy (u). B, International Labor Organization categories of profusion (severity). With higher profusion the blood vessels tend to disappear. (From Guidelines for the use of ILO international classification of radiographs of pneumoconioses. Vol. 22. Occupational Safety and Health Services. Geneva, Switzerland: International Labour Office, 1980; 43.)

rience, 1 mm is better than 2), a high-resolution reconstruction algorithm, a CT technique that produces a high photon flux, and photography of the images at a reasonable size (we use six-on-one) with slightly more contrast than is used for conventional CT (we use a window level of about -700 and width of 1000 Hounsfield units). These are the same technical factors used for HRCT of the thorax in general.⁶

An important decision in determining a protocol for investigation of the pneumoconioses is the spacing of scans. Nobody has published a definite study in this area. In our

opinion different spacing should be used for suspected silicosis, berylliosis, and CWP than for asbestosis. For asbestosis we suggest prone and supine positioning of the patient with about six scans in each position, spaced throughout the lungs.⁷ In unpublished studies we have found that sufficient information is available from three or four prone scans and that these alone could be adequate for a screening study for asbestosis.

Screening or evaluation by HRCT of pneumoconioses other than asbestosis should be performed with the patient in the supine position only and with 1 cm spacing of the scans.⁸ The same high-resolution techniques should be used.

SILICOSIS

Silicosis is a chronic nodular fibrotic disease produced by the inhalation of crystalline silica. Virtually all cases of silicosis in North America result from occupational exposure in hard rock mining, sandblasting, or foundry work. As a result of protective measures the incidence of new cases is rapidly declining, but silicosis occurs in an estimated 3% of heavy metal miners who have worked as miners for at least 20 years.^{2,9,10} Classic silicosis is nowadays a chronic, slowly progressive disease that affects a minority of the workforce. The clinical and pathologic findings of silicosis may be divided into four types: simple silicosis, complicated silicosis, acute silicosis, and Caplan's syndrome.

The traditional imaging modality used for morphologic assessment of silicosis is the chest radiograph. Simple silicosis is characterized by the presence of multiple small fibrotic nodules that occur in greatest profusion in the apical and posterior regions of the upper and lower lobes. The classic radiographic pattern consists of multiple well-defined nodules of uniform size that ranges from 1 to 10 mm in diameter (Fig. 7-2). Occasionally the nodules calcify. Enlargement of lymph nodes is common and may precede the appearance of diffuse nodularity. The periphery of hilar and mediastinal nodes may calcify; this appearance has been referred to as eggshell calcification (Fig. 7-3). The radiographic changes do not usually appear until at least 10 years after the initial exposure to silica dust.

On CT or HRCT the findings in silicosis consist of silicotic nodules, micronodules, coalescent nodules or masses, and larger masses described as progressive massive fibrosis.⁸ CT is more sensitive than chest radiographs for detecting the profusion of nodules and their extent. Remy-Jardin and co-workers⁸ have shown that CT and HRCT are more sensitive than chest radiographs for detecting not only the usual silicotic nodules, but also micronodules, as well as fibrosis in coal workers. Akira and colleagues¹¹ have shown with HRCT that the smallest opacities tend to center in the middle of the secondary pulmonary lobule. The opacities frequently have low-attenuation foci of centrilobular emphysema around them. Bergin and colleagues¹² reported that in patients with silicosis and obstructive pulmonary function, CT may demonstrate emphysema that was not evident from chest radiographs. Bergin and co-workers¹³

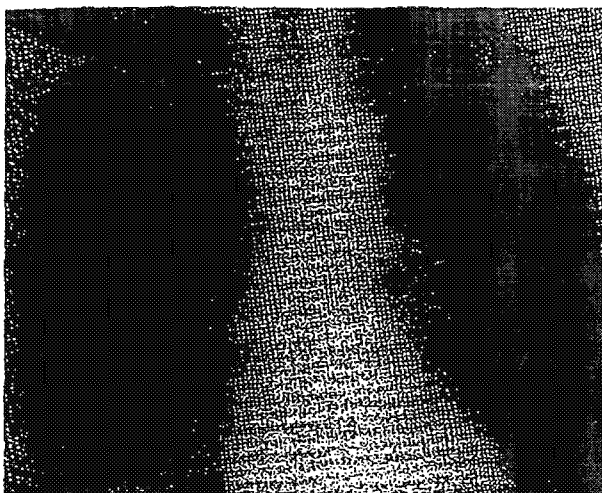


FIGURE 7-2 Simple silicosis. Small rounded opacities (nodules) are distributed throughout both lungs with greatest profusion in the upper lobes. (From McLoud TC. Diffuse infiltrative lung disease. In: Putman CE, ed. *Pulmonary diagnosis: imaging and other techniques*. New York: Appleton-Century-Crofts, 1981; 123-153.)

found that CT also could detect areas of coalescence and larger masses that represented conglomerate silicosis, superimposed tuberculosis, or bronchogenic neoplasm. These masses were not seen with chest radiographs, and because they may not have the typical findings of conglomerate masses of progressive massive fibrosis, further evaluation was necessary in most cases. These investigators suggest that all patients in whom simple silicosis is discovered from chest radiographs be screened with CT to detect occult masses. They do not suggest, however, at what time intervals this screening should be undertaken.

Complicated silicosis is characterized by one or more areas in which the silicotic nodules have become confluent, that is, greater than 1 cm.^{2,4,9} Coalescent small nodules can progress to masses 10 cm or more in diameter. On the chest radiograph these opacities appear in the midzone or in the periphery of the lung in the upper lobes (Fig. 7-4). They tend to migrate to the hilum, leaving overinflated emphysematous lung tissue in the surrounding peripheral lung, particularly at the bases. The more extensive the progressive massive fibrosis, the less the apparent nodularity in the remaining lung.^{2,4} The masses may cavitate as a result of ischemic necrosis. In this situation tuberculosis may supervene, a condition called silicotuberculosis. Findings such as cavitation of conglomerate masses, pleural reaction at the apices, or other rapid radiographic changes suggest complicating tuberculosis. The diagnosis of supervening tuberculosis, however, is bacteriologic rather than radiologic.

The development of a mass in an individual with sili-

cosis raises the possibility of a superimposed bronchogenic carcinoma. When serial chest radiographs do not show stability of the mass, CT is indicated for further investigation. CT cannot always distinguish conglomerate fibrotic masses from other causes of masses in silica-exposed individuals. A mass that shows multiple foci of calcification is likely to be benign. On the other hand, masses with cavities suggest a malignant neoplasm or active tuberculosis and require biopsy. The presence of enlarged mediastinal lymph nodes also suggests an active infection or neoplasm. With progressive massive fibrosis the fibrotic mass is often surrounded by a border of paraseptal emphysema, attesting to a chronic process and making neoplasms less likely. However, this finding is not common enough to be considered diagnostic. In such instances further workup, including percutaneous needle aspiration biopsy of the mass, may be required for diagnosis.

The precise diagnostic role of CT in patients with suspected silicosis or CWP has yet to be defined. At present it appears that CT should be performed in individuals with symptoms, chest radiographic abnormalities, or impaired pulmonary function. CT may detect underlying masses, which may be benign or malignant. It also may demonstrate the cause of symptoms or functional impairment such as emphysema or lung disease unrelated to the pneumoconioses. In compensation cases HRCT can demonstrate nodules that are invisible on chest radiographs. In the future, limited HRCT may become a routine screening test for detection of radiographically occult silicosis or CWP.

Acute silicosis is a rare condition related to heavy exposure to respirable free silica in enclosed spaces in which

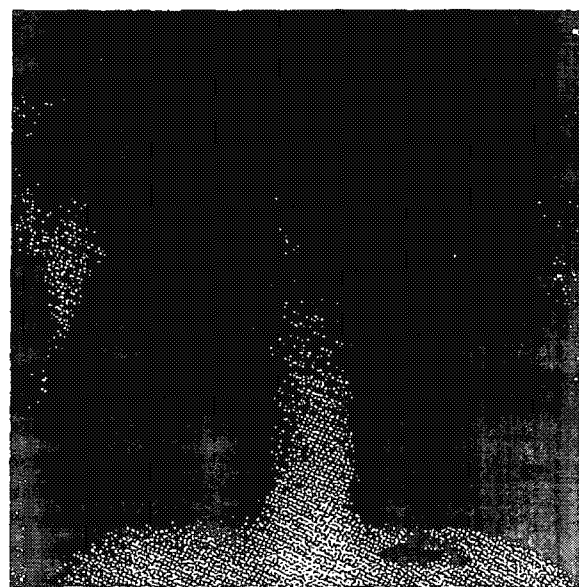


FIGURE 7-3 Silicosis with eggshell calcification in hilar and mediastinal nodes (arrows).

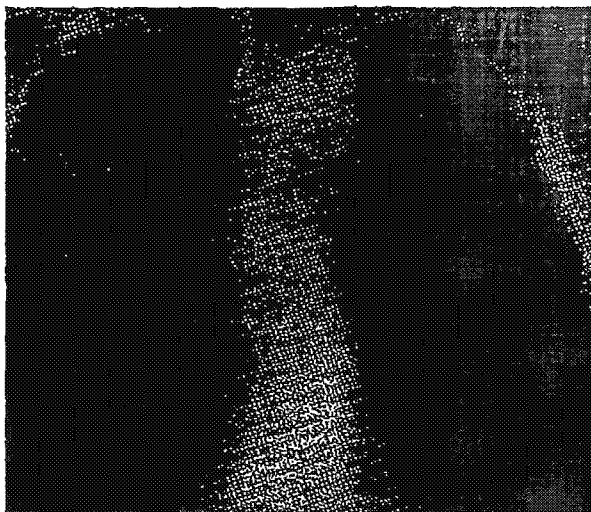


FIGURE 7-4 Silicosis with progressive massive fibrosis. Fibrotic masses are present in the upper lobes bilaterally; they have a somewhat vertical orientation. Overinflated emphysematous lung tissue is present at the bases.

there is minimal or no protection from the silica.^{2,9,14} Sporadic outbreaks still occur but are relatively rare; they are most common in sandblasters and tunnel workers. The disease is often rapidly progressive, with death caused by respiratory failure. On pathologic examination the appearance is that of alveolar proteinosis. The radiographic appearance of acute silicosis or silicoproteinosis is quite different from classic silicosis. Acute silicosis is associated with a pattern of diffuse airspace or "ground glass" disease in a perihilar distribution with air bronchograms.^{2,4,15} Tuberculosis and

infection with atypical mycobacteria are common complications.¹⁴

Caplan's syndrome or rheumatoid lung is characterized by large necrobiotic nodules (rheumatoid nodules) superimposed on a background of simple silicosis. It is seen more commonly in CWP than in silicosis.² The nodules measure from 0.5 to 5 cm; they may cavitate and may calcify (Fig. 7-5). Although they most often develop concomitantly with joint disease, they may precede the onset of arthritis by months or years.²

COAL WORKER'S PNEUMOCONIOSIS

Pneumoconiosis caused by coal dust inhalation is now defined as CWP. It is recognized in the United States as a compensable occupational disease and seems to be common, particularly among underground miners, although it may occur in other workers exposed to coal. Prevalence studies performed in the 1960s and 1970s have shown a prevalence among coal miners ranging from approximately 10% to 25%.^{14,16} Simple CWP results from the retention of coal dust alone. On pathologic study the hallmark of CWP is the coal macule, which consists of aggregations of dust around dilated respiratory bronchioles.^{2,4} Only minimal fibrosis is identified. The chest radiograph is characterized by the presence of nodules, predominantly in the upper lobes. The appearance corresponds to that seen in simple silicosis.

Complicated CWP or progressive massive fibrosis (PMF) occurs on a background of simple CWP. The ILO radiographic classification recognizes the presence of PMF when a single opacity on the radiograph exceeds 1 cm in diameter. PMF may develop many years after exposure has ceased and may progress in the absence of further exposure.¹⁷ The radiologic features consist of large opacities

FIGURE 7-5 Caplan's syndrome. Multiple large pulmonary nodules are present in a man who had worked for 15 years in an underground coal mine. He had clinical rheumatoid arthritis with radiographic evidence of joint changes. (From McLoud TC. The pneumoconioses. In: Freundlich IM, Bragg DG, eds. A radiologic approach to diseases of the chest. Baltimore: Williams & Wilkins, 1991; 293.)



identical to those described in silicosis. Caplan's syndrome is more common in CWP than in silicosis, and a high prevalence of rheumatoid factor is found among miners.¹⁸

ASBESTOS-RELATED DISEASE

The term "asbestos" describes the group of hydrated fiber silicates that share the properties of heat resistance. They can be classified into two large groups, the serpentines and the amphiboles. Cryotile, the only asbestos-formed mineral in the serpentine group, accounts for more than 90% of the asbestos used in the United States.^{2,19} Among the amphiboles, crocidolite is the most dangerous form of asbestos because of its association with malignant mesothelioma.

The two major sources of exposure to asbestos dust are the primary occupations (mining asbestos and processing it in a mill) and the secondary occupations (such as insulation manufacturing, textile manufacturing, construction, shipbuilding, and gasket and brake lining manufacturing and repair). As with the other pneumoconioses, the development of the disease appears to depend on both the degree and length of exposure. Clinical manifestations usually do not appear until 20 years after the initial exposure.

Inhalation of asbestos fibers causes disease in the pleura and lung parenchyma and at distant sites. The pleural disease includes plaques (circumscribed thickening), diffuse pleural thickening, pleural effusion, and mesothelioma. The lung parenchymal abnormalities include pulmonary fibrosis or asbestosis, lung neoplasms, and cicatricial lung masses. Bronchogenic carcinoma, pleural and peritoneal mesothelioma, laryngeal cancer, and gastrointestinal cancers are well-recognized or suspected hazards of occupational exposure to asbestos.²⁰⁻²²

The significance of intensity and length of exposure and of the time elapsed from exposure to disease was apparent as early as the 1930s. In the last 25 years these connections have been convincingly established, and their variations for the different manifestations determined.

Pleural Disease

PLAQUES (CIRCUMSCRIBED THICKENING)

Pleural abnormalities are common in patients with asbestos exposure. The most frequently seen lesions are pleural plaques, which are discrete, elevated, opaque, shiny, fibrous areas of pleural thickening. They are found most commonly on the parietal pleura. Generally plaques do not appear until 20 or more years after initial exposure.^{4,23,24} The plaques themselves do not produce any symptoms, and unless extensive, they are not associated with functional impairment.

On the chest radiograph, plaques are identified as localized, limited, plateaulike, smooth and nodular areas of pleural thickening.^{4,23,25} On a posteroanterior (PA) radiograph a well-developed pleural plaque has either a "profile" or an "en face" presentation.^{4,25} The plaque in profile appears as a sharply margined dense band of soft tissue density, from 1 to 10 mm thick, paralleling the inner margin of the

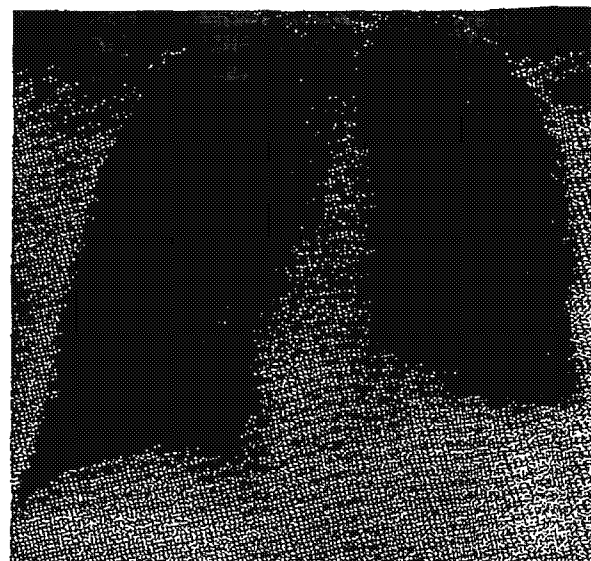


FIGURE 7-6 Pleural plaques. Bilateral plaques can be seen both in profile (*small arrows*) and en face (*large arrow*). En face plaques may simulate pulmonary nodules.

lateral thoracic wall (Fig. 7-6). Plaques are usually bilateral, often symmetric, and more prominent in the lower half of the thorax between the sixth and ninth ribs. When seen en face, a pleural plaque appears as a faint, ill-defined, veil-like opacity with irregular edges. In addition to the standard PA radiograph, oblique views may be helpful to confirm suspected pleural plaques and to detect additional plaques not visible on a PA radiograph.²⁶ Early plaque formation must be distinguished from normal companion shadows of the chest wall or from extrapleural fat, a distinction that is sometimes impossible without CT.^{27,28} Despite these difficulties it is clear that noncalcified plaques occur often enough to be regarded as virtually diagnostic of asbestos exposure and asbestos-related pleural disease. They are an excellent indication of significant exposure, although extensive exposure and lung disease can occur without plaques being evident on chest radiographs. Unless extensive, plaques alone are unassociated with functional lung impairment.

CT and HRCT are more sensitive than chest radiographs for detection of pleural plaques.^{7,29} On CT they form linear white densities of various thicknesses. They might be flat or irregularly margined (Fig. 7-7). On HRCT they must be differentiated from the normal endothoracic fascia and from the subjacent intercostal vein.²⁸ In our studies in 100 occupationally exposed subjects, HRCT detected asbestos-related pleural disease in at least 25% more cases than chest radiographs (Table 7-1).²⁹

A most striking radiographic abnormality is calcification of pleural plaques. Calcified plaques vary from small linear or circular shadows to complete encasement of the

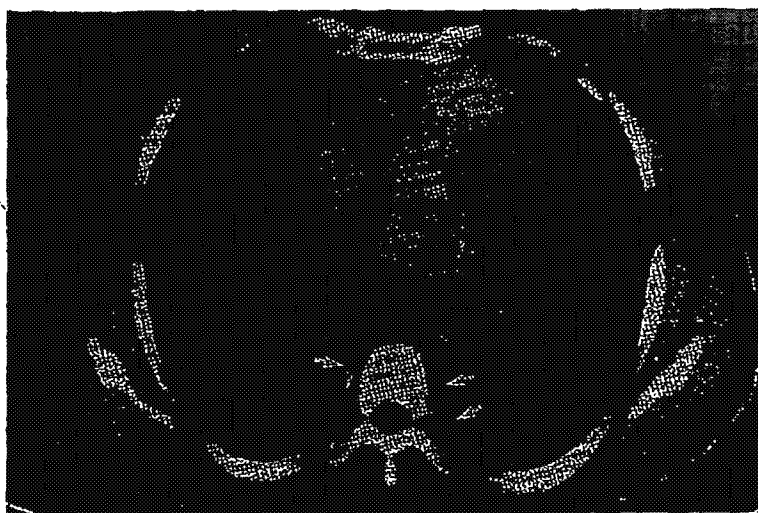


FIGURE 7-7 Asbestos-related pleural plaques. Computed tomography demonstrates plaques along the rib margins on both sides anteriorly, posterolaterally, and posteriorly. Several plaques demonstrate foci of calcification. The larger plaques will be visible on conventional chest radiographs. The smaller and paraspinal plaques (arrows) will not be seen on radiographs.

lower portion of the lungs. When calcification is minimal, CT greatly facilitates its visibility (Table 7-1). No part of the pleura is immune to calcification, although the most common site is over the diaphragm.

DIFFUSE PLEURAL THICKENING

Diffuse pleural thickening is less frequently seen than pleural plaques following exposure to asbestos. It is characterized by uniform, homogeneous density, by smooth contours, and frequently by obliteration of the costophrenic angle (Fig. 7-8).^{4,23,26} Diffuse pleural thickening is most frequently the result of a previous benign asbestos effusion.³⁰

Diffuse pleural thickening is more easily appreciated on CT than on chest radiographs. It is of lower density than pleural plaques, is usually thicker and more irregular, and forms a continuous sheet at least 8 to 10 cm in a craniocaudal direction and 5 cm in a lateral direction (Fig. 7-9). The

posterior and paraspinal regions are the most commonly affected sites. Extensive diffuse pleural thickening, unlike pleural plaques, is commonly associated with restrictive pulmonary function and symptoms. On HRCT most patients with diffuse pleural thickening demonstrate volume loss in the subjacent lung and extensive bands of scar tissue extending into the lung parenchyma.

ASBESTOS PLEURAL EFFUSION

Benign asbestos pleurisy frequently occurs before other manifestations of asbestos-related disease and is commonly seen during the first 20 years after exposure.^{4,23,31} Most effusions are small. They may be recurrent, and two thirds are asymptomatic.^{4,23,31} The diagnosis can be made only after exclusion of other possibilities, particularly tuberculosis and mesothelioma. Most benign asbestos effusions are easily detected radiographically on standard PA and lateral chest radiographs. Residual diffuse pleural thickening, as described previously, usually with a blunted costophrenic angle, occurs in about half of patients with benign asbestos effusion as the effusion resolves.³¹

TABLE 7-1 ASBESTOS-RELATED PLEURAL DISEASE IN 100 SUBJECTS

Abnormality	Chest Radiograph	Computed Tomography	High-Resolution Computed Tomography
Plaques	49	56	64
Calcification	13	16	20
Diffuse thickening	4	6	3

Parenchymal Disease

ASBESTOSIS

Asbestosis is the term reserved for pulmonary fibrosis caused by the inhalation of asbestos fibers.³² The fibrotic process usually starts at the centrilobular bronchiolar level and is multifocal with normal parenchyma intervening. Subpleural areas, predominantly in the lower lobes, are usually

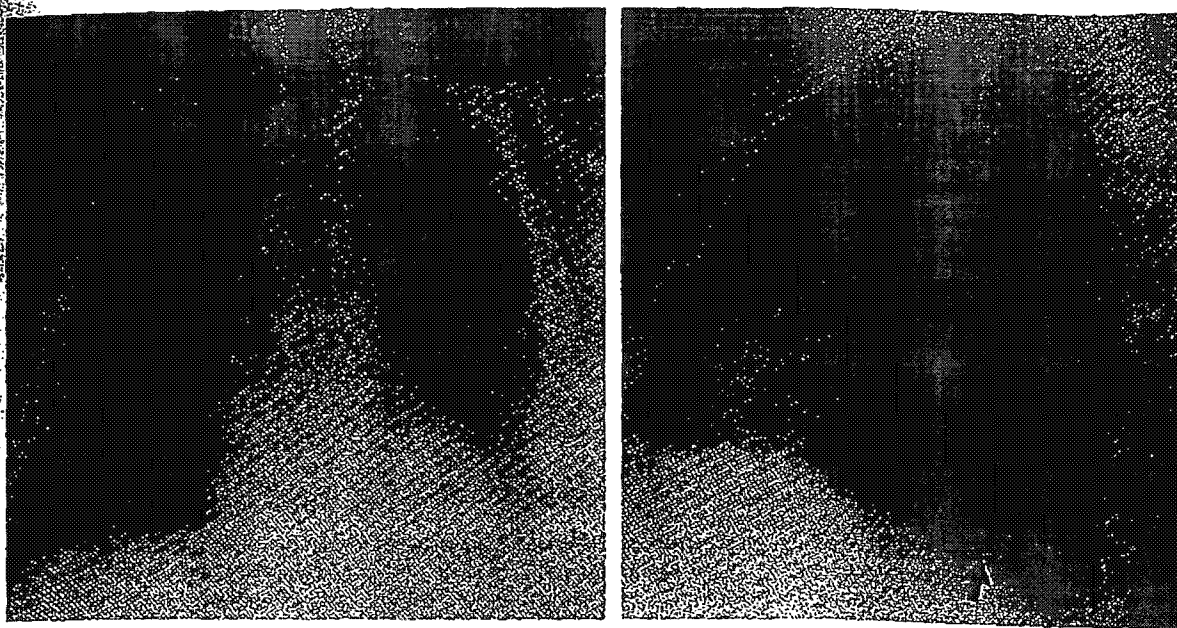


FIGURE 7-8 Diffuse pleural thickening. The thickening on the left side is smooth and uninterrupted and involves both the lateral and the posterior costophrenic angles. Pleural calcification can also be identified along the diaphragm (*arrow*). (From McLoud TC. The pneumoconioses. In: Freundlich IM, Bragg DG, eds. A radiologic approach to diseases of the chest. Baltimore: Williams & Wilkins, 1991; 297.)

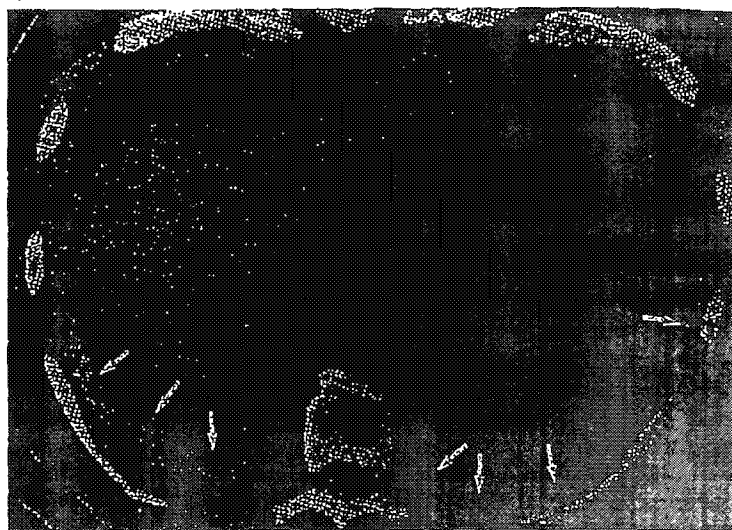


FIGURE 7-9 Bilateral diffuse pleural thickening. High-resolution computed tomography demonstrates diffuse pleural thickening (*arrows*) on both sides laterally and posteriorly. The thickening is of lower density than pleural plaques, is more irregular, and forms a continuous sheet. Increase in thickness of the extrapleural fat reflects lung retraction.

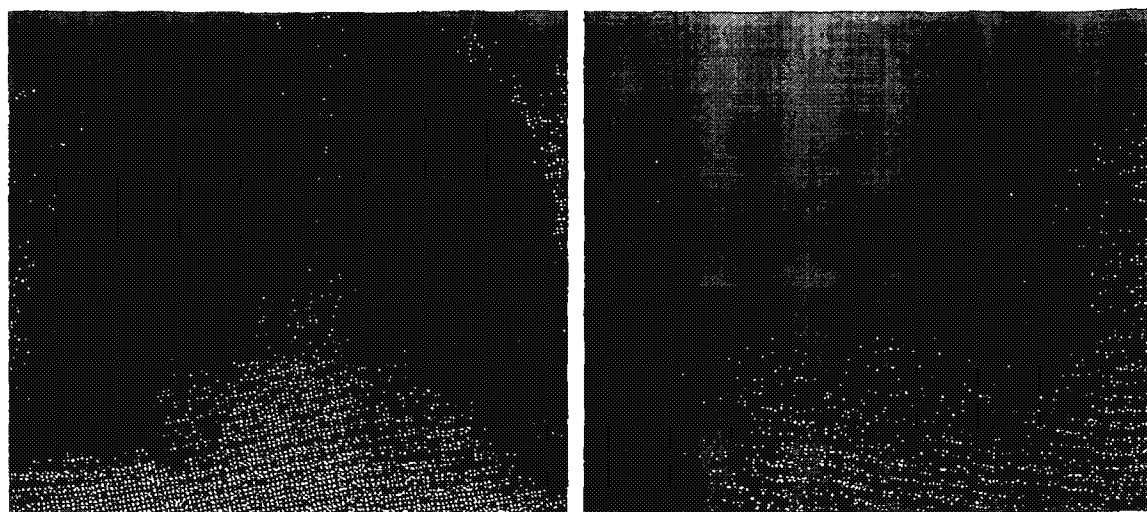


FIGURE 7-10 Advanced asbestosis. Coarse linear opacities with honeycombing are present at both bases and are associated with pleural thickening. (From McLoud TC. Diffuse infiltrative lung disease. In: Putman CE, ed. Pulmonary diagnosis: imaging and other techniques. New York: Appleton-Century-Crofts, 1981; 125-153.)

involved first. Subpleural honeycombing may be a prominent feature, but it is seen in only about 10% of patients with asbestosis. The incidence of asbestosis has been declining, and in newly diagnosed cases the disease is relatively mild and does not progress.³³ There is, however, a definite dose-effect relationship.¹⁹

The radiographic changes consist of small irregular opacities or a linear pattern that predominates in the lung bases.^{4,23} A fine reticulation eventually progresses to a coarse linear pattern with honeycombing (Fig. 7-10). The combination of parenchymal and pleural changes leads to partial obscuration of the heart border, the so-called shaggy heart sign.

Although chest radiography is the imaging method of choice both in surveillance studies for detection of asbestosis and in the follow-up of patients with confirmed disease, evidence indicates that CT might be useful in detecting early asbestosis. Early CT studies by Katz and Kreel³⁴ demonstrated abnormalities about twice as commonly as radiographs. The abnormalities included honeycombing, reticulonodular densities, and fine linear densities. This increased sensitivity for detection of parenchymal abnormality has been confirmed by several groups.^{29,35-37}

More recently, several investigators have applied HRCT to the detection and characterization of asbestosis.^{7,27} When the chest radiograph shows abnormalities, the HRCT findings of asbestosis may be present. Of our occupationally exposed subjects with normal chest radiography findings, about one third had abnormal HRCT scans indicative of parenchymal asbestosis (Table 7-2).²⁹

Patients with asbestosis display many of the features of interstitial fibrotic lung disease on HRCT. These include

septal and centrilobular thickening (Fig. 7-11), long scars or parenchymal bands traversing the lung (Fig. 7-12), honeycombing (Fig. 7-13), subpleural lines (Fig. 7-14), and architectural distortion. The interstitial changes frequently occur adjacent to the areas of pleural plaques and are usually most severe in the posterior lower lobes.

In asbestos-exposed individuals the HRCT diagnosis of asbestosis should require a multiplicity of abnormalities on both sides and at several levels that conform to known pathologic abnormalities. Although the incidence of asbestosis without radiographically visible pleural plaques is about 15%, we have found that virtually all patients with parenchymal disease have at least some plaques visible on HRCT. In occupationally exposed individuals the incidence of HRCT abnormality suggestive of asbestosis, when the chest radiograph shows normal lung parenchyma, varies with the age of the population and the extent of exposure (Fig. 7-15). In our experience in a population with heavy shipyard exposure and long latency, the percentage is between 15%

TABLE 7-2 COMPARISON OF CHEST RADIOGRAPH AND HIGH-RESOLUTION COMPUTED TOMOGRAPHY IN 100 PATIENTS WITH ASBESTOSIS

Chest Radiograph	High-Resolution Computed Tomography	
	Abnormal	Normal
Normal	34	1
Abnormal	39	26



FIGURE 7-11 Asbestosis with interstitial fibrosis. High-resolution computed tomography with the patient in a prone position shows multiple sites of interlobular septal thickening (*arrows*) and intralobular core structure thickening (*open arrows*).

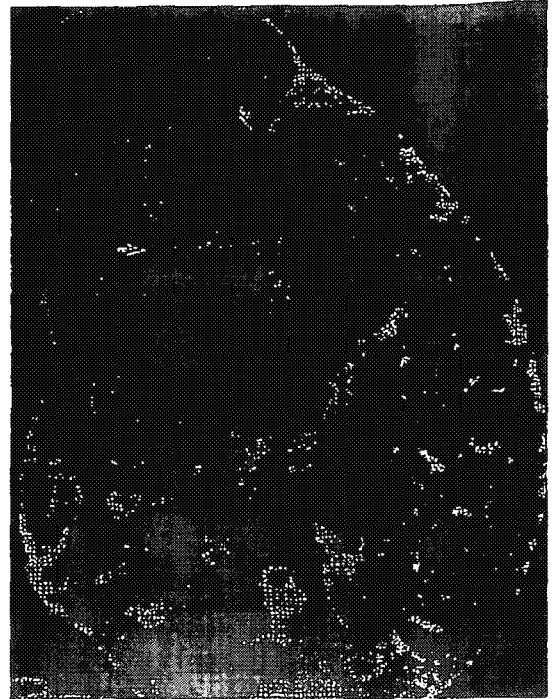


FIGURE 7-12 Specimen radiograph from a patient with asbestosis. High-resolution computed tomography of an inflated lung specimen with asbestosis demonstrates a multiplicity of abnormalities, including honeycombing and long scars (parenchymal bands) (*arrows*).

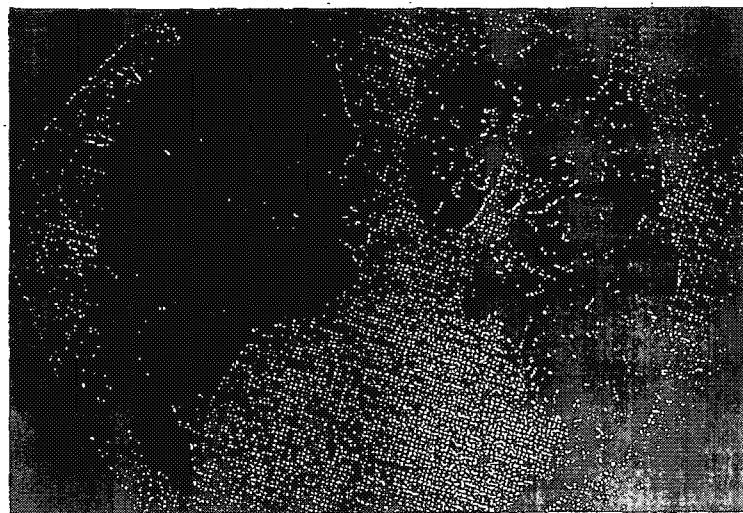


FIGURE 7-13 Asbestosis with honeycombing and interstitial disease. High-resolution computed tomography with the patient in the prone position demonstrates extensive honeycombing on the right side. Interstitial septal thickening is present at the left base in nondependent lung (*arrows*).

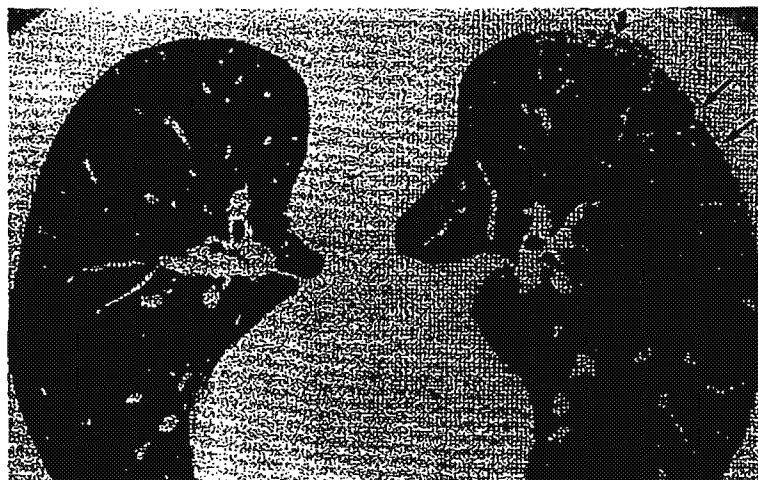


FIGURE 7-14 Asbestosis with interstitial fibrosis. High-resolution computed tomography with the patient in the prone position demonstrates a distinct subpleural line (arrows) on the right side in nondependent lung. In addition, focal subpleural honeycombing (curved arrow) is present on the right side.

and 25%. This range concurs with the known incidence of pathologic fibrosis not seen on chest radiographs in this type of population.

Staples and colleagues³⁸ studied asbestos-exposed individuals with normal lung parenchyma on chest radiographs to determine the functional importance of parenchymal dis-

ease found only with HRCT. HRCT of the lung parenchyma was normal in 76 patients and suggestive of asbestosis in 57 patients. The 57 with abnormal findings on HRCT had significantly reduced lung function, suggesting restrictive defect. Thus HRCT may be sensitive in detecting mild asbestosis not visible radiographically. In a recent unpublished pathologic correlative study, we found that HRCT had about a 65% sensitivity for detecting asbestosis. However, the specificity in this occupationally exposed population was 100%.

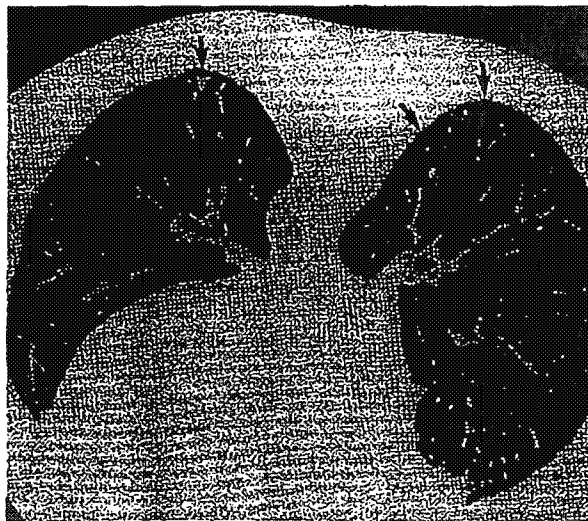


FIGURE 7-15 Asbestosis with a normal chest radiograph and minimal abnormalities. High-resolution computed tomography with the patient in the prone position demonstrates focal centrilobular and interlobular (arrows) thickening at both lung bases. The abnormalities are mild in extent and severity. These findings were judged insufficient to diagnose interstitial lung disease. Open lung biopsy, however, revealed interstitial fibrosis and asbestos bodies.

Thoracic Masses

BENIGN MASSES

Individuals with occupational exposure to asbestos are at risk for benign and malignant intrathoracic masses. These masses include fissural pleural plaques, rounded atelectasis, fibrous masses, mesothelioma, and bronchogenic carcinoma.³⁷ One type of benign mass is rounded atelectasis, which is a form of peripheral lobar collapse that develops in patients with pleural disease.^{39,40} On the standard chest radiograph, rounded atelectasis appears as a round, sharply marginated mass abutting the pleura. Pleural thickening is always present and frequently is of greatest dimension near the mass. The mass often has a curvilinear tail referred to as the comet tail sign.⁴¹ The tail is produced by crowding together of bronchi and vessels that extend from the lower border of the mass to the hilum. CT may be useful in evaluating such features when they are not obvious on standard films. CT may in addition show that the area of rounded atelectasis has a low-density center containing air bronchograms and calcifications. Volume loss in the lobe containing the masses is also more readily appreciated on CT (Fig. 7-16).⁴²

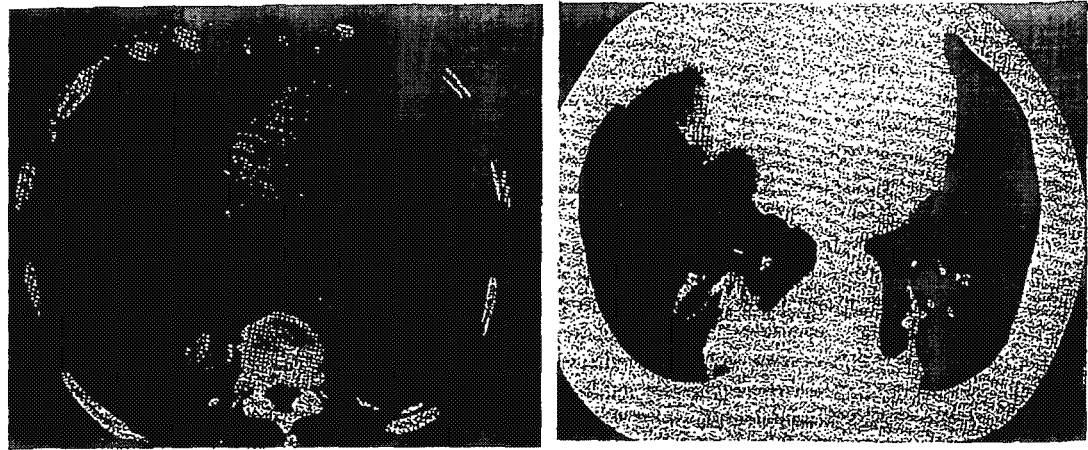


FIGURE 7-16 Rounded atelectasis. Computed tomography through the lung bases demonstrates bilateral posterior circumscribed masses. At soft tissue window settings (*left*), diffuse pleural thickening and pleural calcifications are seen adjacent to the posterior masses. At lung window settings (*right*), vessels and bronchi converge on the masses in a comet tail configuration. Both lower lobes are reduced in volume.

CT has identified a spectrum of fibrotic or cicatricial masses that do not have the typical features of rounded atelectasis in patients with asbestos exposure and diffuse pleural thickening. These masses must be viewed with suspicion for malignancy. On CT such benign masses tend to be contiguous with abnormal pleural thickening and have multiple long fibrotic scars radiating from them.

MALIGNANT MASSES

Patients with asbestos exposure are at risk for lung carcinoma and mesothelioma. Adenocarcinoma arising in the lower lobes is the most common malignancy and may be difficult to detect when pleural and parenchymal fibrosis is present. The increased risk of lung carcinoma has been observed in asbestos workers who smoke, since smoking

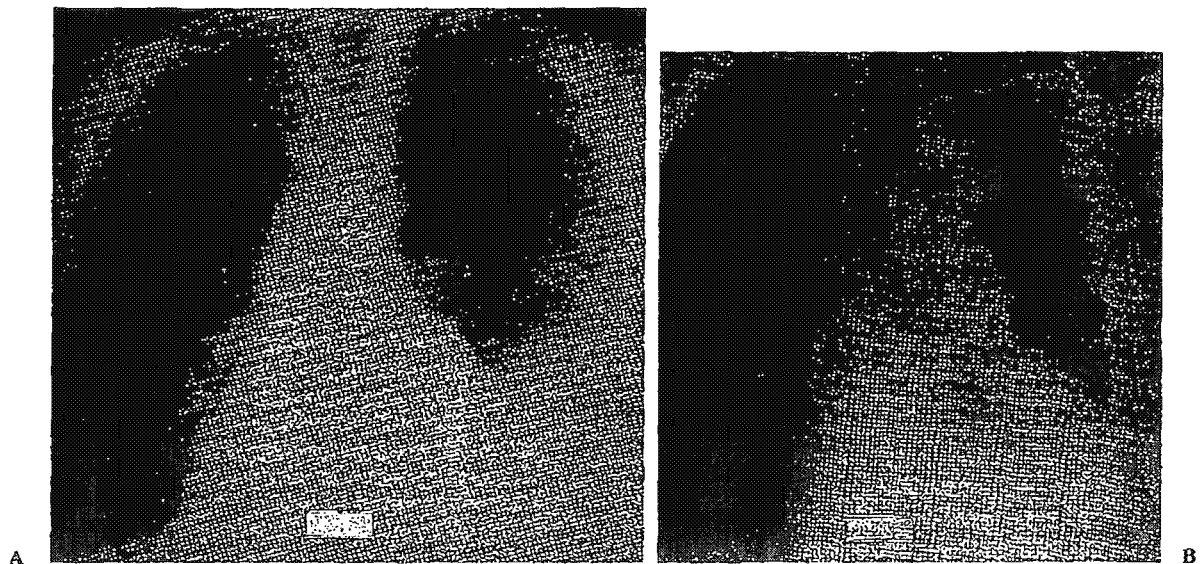


FIGURE 7-17 Malignant mesothelioma. **A**, On the initial study a left pleural effusion can be seen. **B**, Nine months later diffuse thickening of the left pleura has occurred both on the lateral and on the mediastinal surfaces. The trachea is slightly shifted to the left.

and asbestos exposure interact in a multiplicative manner.⁴³ When a mass is detected on CT or a chest radiograph, a high index of suspicion for malignancy must be maintained. Masses that do not have the features of benignity previously described should be biopsied.

Diffuse malignant mesothelioma is an uncommon and fatal neoplasm of the serosal lining of the pleural cavity or peritoneum. The risk of mesothelioma to any asbestos worker is approximately 10% over his or her lifetime.^{4,18} Again, a latency period of approximately 20 to 40 years usually occurs from exposure to detection of tumor. The tumor commences as nodules on the visceral or parietal pleura that produce a thick rind, encasing and constricting the lung. The tumor may extend into the fissures and interlobular septa with superficial invasion of the underlying lung. Advanced cases show invasion of the chest wall, pericardium, diaphragm, and abdomen. The prognosis is universally poor. Radiographic features include a moderate-sized to large pleural effusion with or without a pleural mass or diffuse pleural thickening with or without an effusion. About one third of patients have contralateral plaques as a manifestation of asbestos exposure.⁴⁴ Malignant mesothelioma usually envelops the entire pleura and encases the underlying lung. This results in a shift of the mediastinum toward the side of disease in most cases (Fig. 7-17).

CT may be helpful both in detecting underlying signs of asbestos exposure, such as pleural plaques, and in determining the extent of tumor. The latter indication may be important in a small minority of patients who are chosen for surgical resection. It may also be useful in determining response to other treatment modalities such as radiation therapy and chemotherapy. CT features of mesothelioma include pleural thickening of varying extent and nodularity of the pleura.⁴⁵ The hemithorax is frequently contracted as a result of the pleural disease. CT is particularly useful in determining direct extension of the tumor to the pericardium or invasion of the diaphragm with spread to the abdomen.⁴⁵

SUMMARY

Both standard radiography and CT play an important role in the imaging of asbestos-exposed individuals. Chest radiographs should be used for initial screening of exposed populations and for evaluation of symptomatic workers.

The role of HRCT for evaluating asbestos-related diseases is both medical and legal. Many asbestos-exposed patients are being evaluated for compensation. In asymptomatic patients with unequivocal radiographic and functional abnormalities, CT probably does not add additional information. In occupationally exposed subjects with equivocal abnormal or normal chest radiographs, abnormalities can be found in appreciable number. CT may clearly demonstrate pleural and parenchymal disease when chest radiographic results are normal. It may also be helpful in explaining symptoms or abnormal findings of pulmonary function studies by showing diseases, such as unsuspected emphysema, unrelated to the asbestos exposure. In exposed

patients with a suspected lung mass, CT can help characterize the mass and provide assistance for biopsy of lesions suspected to be malignant.

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