



FIGURE 311-2 Acute silicosis. This high-resolution computed tomography scan shows multiple small nodules consistent with silicosis but also diffuse ground-glass densities with thickened intralobular and interlobular septa producing polygonal shapes. This has been referred to as “crazy paving.”

(Chap. 315). The chest radiograph may show profuse miliary infiltration or consolidation, and there is a characteristic HRCT pattern known as “crazy paving” (Fig. 311-2). The disease may be quite severe and progressive despite the discontinuation of exposure. Whole-lung lavage may provide symptomatic relief and slow the progression.

With long-term, less intense exposure, small rounded opacities in the upper lobes may appear on the chest radiograph after 15–20 years of exposure, usually without associated impairment of lung function (*simple silicosis*). Calcification of hilar nodes may occur in as many as 20% of cases and produces a characteristic “eggshell” pattern. Silicotic nodules may be identified more readily by HRCT (Fig. 311-3). The nodular fibrosis may be progressive in the absence of further exposure, with coalescence and formation of nonsegmental conglomerates of irregular masses >1 cm in diameter (*complicated silicosis*). These masses can become quite large, and when this occurs, the term *progressive massive fibrosis* (PMF) is applied. Significant functional impairment with both restrictive and obstructive components may be associated with PMF.

Because silica is cytotoxic to alveolar macrophages, patients with silicosis are at greater risk of acquiring lung infections that involve these cells as a primary defense (*Mycobacterium tuberculosis*, atypical mycobacteria and fungi). Because of the increased risk of active tuberculosis, the recommended treatment of latent tuberculosis in these patients is longer. Another potential clinical complication of silicosis is autoimmune connective tissue disorders such as rheumatoid arthritis and scleroderma. In addition, there are sufficient epidemiologic data that the International Agency for Research on Cancer lists silica as a probable lung carcinogen.

Other, less hazardous silicates include fuller’s earth, kaolin, mica, diatomaceous earths, silica gel, soapstone, carbonate dusts, and cement dusts. The production of fibrosis in workers exposed to these agents is believed to be related either to the free silica content of these dusts or, for substances that contain no free silica, to the potentially large dust loads to which these workers may be exposed. Some silicates, including *talc* and *vermiculite*, may be contaminated with asbestos. Fibrosis of lung or pleura, lung cancer, and mesothelioma have been associated with chronic exposure to talc and vermiculite dusts.

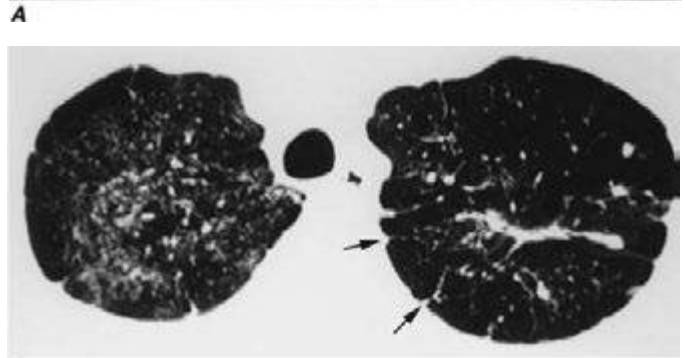
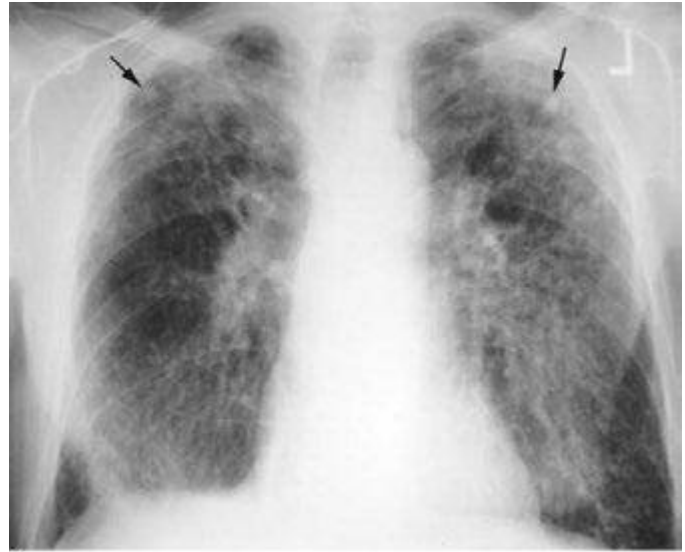


FIGURE 311-3 Chronic silicosis. **A.** Frontal chest radiograph in a patient with silicosis shows variably sized, poorly defined nodules (arrows) predominating in the upper lobes. **B.** Axial thoracic computed tomography image through the lung apices shows numerous small nodules, more pronounced in the right upper lobe. A number of the nodules are subpleural in location (arrows).

COAL WORKER’S PNEUMOCONIOSIS (CWP)

Occupational exposure to *coal dust* can lead to CWP, which has enormous social, economic, and medical significance in every nation in which coal mining is an important industry. Simple radiographically identified CWP is seen in ~10% of all coal miners and in as many as 50% of anthracite miners with more than 20 years of work on the coal face. The prevalence of disease is lower in workers in bituminous coal mines.

With prolonged exposure to coal dust (i.e., 15–20 years), small, rounded opacities similar to those of silicosis may develop. As in silicosis, the presence of these nodules (*simple CWP*) usually is not associated with pulmonary impairment. In addition to CWP, coal dust can cause chronic bronchitis and COPD (Chap. 314). The effects of coal dust are additive to those of cigarette smoking.

Complicated CWP is manifested by the appearance on the chest radiograph of nodules ≥ 1 cm in diameter generally confined to the upper half of the lungs. As in silicosis, this condition can progress to PMF that is accompanied by severe lung function deficits and associated with premature mortality. Despite improvements in technology to protect coal miners, cases of PMF still occur in the United States at a disturbing rate.

Caplan syndrome (Chap. 380), first described in coal miners but subsequently in patients with silicosis, is the combination of pneumoconiotic nodules and seropositive rheumatoid arthritis. Silica has immunoadjuvant properties and is often present in anthracitic coal dust.