migrating macrophages and thereby initiate an immune response to components of the particulates or to self-proteins modified by the particles or both.
- Finally, certain types of particles activate the inflammasome (Chapter 3) when phagocytosed by macrophages. These innate and adaptive immune responses amplify the intensity and the duration of the local reaction.
- Tobacco smoking worsens the effects of all inhaled mineral dusts, but particularly those caused by asbestos. The effects of inhaled particles are not confined to the lung alone, since solutes from particles can enter the blood and lung inflammation invokes systemic responses.

In general, only a small percentage of exposed people develop occupational respiratory diseases, implying a genetic predisposition to their development. Many of the diseases listed in Table 15-6 are quite uncommon. Hence only a selected few that cause fibrosis of the lung are presented next.

Coal Workers' Pneumoconiosis

Coal workers' pneumoconiosis is lung disease caused by inhalation of coal particles and other admixed forms of dust. Dust reduction measures in coal mines around the globe have drastically reduced its incidence. The spectrum of lung findings in coal workers is wide, varying from asymptomatic anthracosis, to simple coal workers' pneumoconiosis with little to no pulmonary dysfunction, to complicated coal workers' pneumoconiosis, or *progressive massive fibrosis,* in which lung function is compromised. Contaminating silica in the coal dust can favor progressive disease. In most cases, carbon dust itself is the major culprit, and studies have shown that complicated lesions contain much more dust than simple lesions. Coal workers may also develop emphysema and chronic bronchitis independent of smoking.

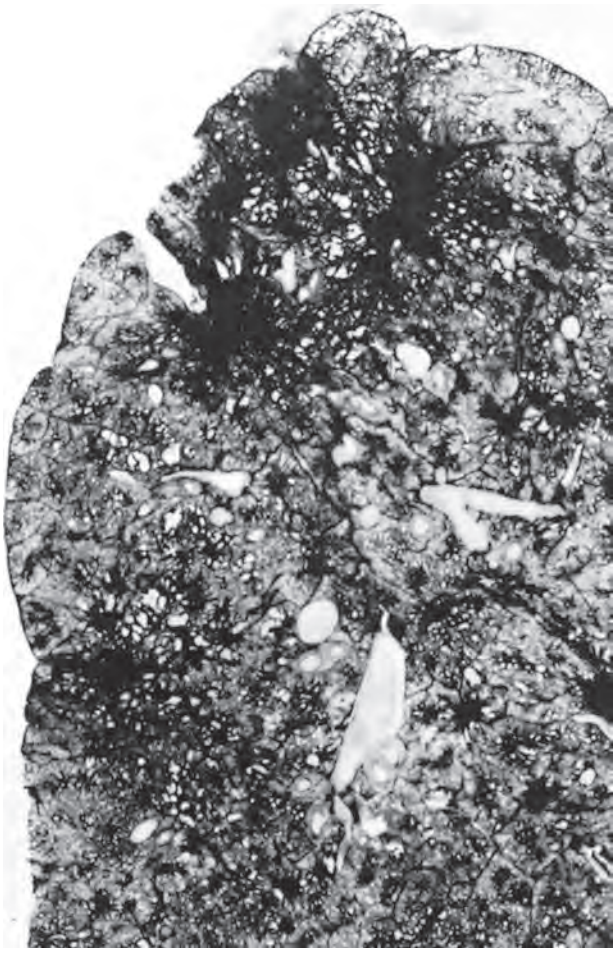

Figure 15-17 Progressive massive fibrosis superimposed on coal workers' pneumoconiosis. The large, blackened scars are located principally in the upper lobe. Note the extensions of scars into surrounding parenchyma and retraction of adjacent pleura. (Courtesy Drs. Werner Laquer and Jerome Kleinerman, the National Institute of Occupational Safety and Health, Morgantown, W.Va.)

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Anthracosis is the most innocuous coal-induced pulmonary lesion in coal miners and is also seen to some degree in urban dwellers and tobacco smokers. Inhaled carbon pigment is engulfed by alveolar or interstitial macrophages, which then accumulate in the connective tissue along the lymphatics, including the pleural lymphatics, or in organized lymphoid tissue along the bronchi or in the lung hilus.

Simple coal workers' pneumoconiosis is characterized by **coal macules** (1 to 2 mm in diameter) and somewhat larger **coal nodules**. Coal macules consist of carbon-laden macrophages; nodules also contain a delicate network of collagen fibers. Although these lesions are scattered throughout the lung, the upper lobes and upper zones of the lower lobes are more heavily involved. They are located primarily adjacent to respiratory bronchioles, the site of initial dust accumulation. In due course dilation of adjacent alveoli occurs, sometimes giving rise to **centrilobular emphysema**.

Complicated coal workers' pneumoconiosis (progressive massive fibrosis) occurs on a background of simple disease and generally requires many years to develop. It is characterized by intensely blackened scars 1 cm or larger, sometimes up to 10 cm in greatest diameter. They are usually multiple (Fig. 15-17). Microscopically the lesions consist of dense collagen and pigment. The center of the lesion is often necrotic, most likely due to local ischemia.

Clinical Course. Coal workers' pneumoconiosis is usually benign, causing little decrement in lung function. Even mild forms of complicated coal workers' pneumoconiosis do not to affect lung function significantly. In a minority of cases (fewer than 10%), progressive massive fibrosis develops, leading to increasing pulmonary dysfunction, pulmonary hypertension, and cor pulmonale. Once progressive massive fibrosis develops, it may continue to worsen even if further exposure to dust is prevented. Unlike silicosis (discussed below), there is no convincing evidence that coal dust increases susceptibility to tuberculosis. There is also no compelling evidence that coal workers' pneumoconiosis in the absence of smoking predisposes to cancer. Domestic indoor use of "smoky coal" (bituminous) for cooking and heating is, however,