quently shown to be a phase in the development of other pleural changes.

*Pathology.* On macroscopic examination at thoracotomy, the pleural surfaces show an active exudative process, characterized by increased vascularity and symphysis. Associated pleural plaques do not appear to be a feature. Microscopic examination shows variable pleural thickening, with "pleural drift of carbon and other dusts and iron-positive granules" (131). Other features include regenerating mesothelium and extensive collateral circulation (131), and there is one report of a granuloma (134). The underlying lung tissue shows varying degrees of interstitial pneumonitis, from mild, low grade inflammation to organized interstitial fibrosis, in which asbestos bodies and fibers are usually, but not invariably, found. Electron microscopic analysis for uncoated fibers has not been reported in this type of case, but it can be assumed that these would be found.

*Clinical features.* The usual presentation is one of recurring pleural effusion of unknown cause, associated with chest pain (131). The effusion may be unilateral, bilateral, or one side may follow the other in sequence. The presentation may be acute, with fever, leukocytosis, and an increased sedimentation rate, or chronic, with minimal systemic reaction. The pleural fluid is frequently blood stained (red blood cell counts ranging from 5,000 to 50,000 cells per $mm^3$) and, in most cases, is an exudate. The clinical course varies from that of a benign and self-limiting illness (132, 133) to the development of chronic pleural thickening that requires decortication (131). The patient's association with asbestos may be current, or more often may have been brief and in the remote past (131).

*Differential diagnosis.* The diagnosis of a benign asbestos pleural effusion should be considered a diagnosis only by exclusion. The chief alternative to be excluded is malignant mesothelioma, one of the manifestations of asbestos exposure that may not develop until many years after the first exposure. The pleural effusions that commonly complicate the latter tumor may precede by months or years the definitive diagnosis of the tumor. Indeed, one could argue against accepting the diagnosis of benign asbestos pleural effusion until all of the present reported cases are followed to death, and death is shown to be attributable neither to carcinoma of the lung nor to mesothelioma. However, as follow-up becomes longer (134), the justification for this diagnosis increases, and asbestos exposure can reasonably be added to the long list of causes of benign, recurrent pleural effusion.

### Diffuse Interstitial Pulmonary Fibrosis (Asbestosis)

#### *Definition*

Diffuse interstitial fibrosis of the lung associated with asbestos exposure was recognized in the early years of the twentieth century, the first asbestos-related disease to be so recognized (1); however, the term "asbestosis" to describe this pneumoconiosis was not suggested until 1927, when Cooke (136) used it to describe the case of a female asbestos textile worker. It is usual to include fibrosis of the associated visceral pleura under this term, but not that of the parietal pleura (10). There is merit in maintaining this specific usage in line with the widely accepted use of the term pneumoconiosis (137), rather than to use the term "asbestosis" in a generic sense to describe all asbestos-related diseases of the lung and pleura, even neoplasms (10).

#### *Pathology*

*Macroscopic appearances.* The main pathologic features that had been described with care by the 1930s in individual case reports (138, 139) were reviewed by Hourihane and McCaughey (116) in the light of their own pathologic material, based on 69 cases of clinical asbestosis examined at the London Hospital. Macroscopic changes ranged from small areas of basal fibrosis (if sufficiently localized, these may escape recognition by the naked eye) to the fully developed case of a diffuse, fine fibrosis affecting both lungs. Lung size tends to reflect the extent of the fibrosis; when this is diffuse, the lungs tend to be small. Cut surface shows the fine, grey-colored fibrosis that generally appears to affect subpleural areas first, often quite extensively, before advancing into other lobes with the extension of the disease process. Lower lobes tend to be affected first, then middle lobes, and eventually, upper lobes (10, 116, 140). Small honeycomb cysts may be seen, in the lower lobes particularly, and fibrosis and honeycombing also tend to be concentrated subpleurally (140). Emphysema, centrilobular or bullous, is frequently found, with characteristics essentially the same as in emphysema not associated with asbestosis (140, 141). The pleural surface in relation to the fibrosis is invariably involved in