Vorwald[18] that usually the disease will not occur with fibers less than 20μ in length or a concentration below five million particles per cubic foot of air.

The pathologic processes resulting from the inhalation of asbestos particles are believed to be due not to their chemical nature but, rather, the consequence of mechanical irritation from fibers lodged in the respiratory tree.[18–20] The inhaled particles are, in general, too large to pass beyond the respiratory bronchioles and so they remain there to initiate a foreign body reaction which eventually leads to fibrosis.[21] The pathologic sequence of events can be considered as occurring in three stages: (1) desquamation and exudation, (2) formation of asbestosis bodies and (3) fibrosis and scarring.

The long fibers traumatize the epithelial cells lining the smaller bronchioles and the constant irritation and friction cause the cells to desquamate. Macrophages pour forth in an effort to phagocytize the fibers. In our case fragmented asbestosis bodies were also seen within macrophages and lymphatics. A second reaction to the asbestos fiber in the lung is the production of the so-called "asbestosis body."[22–24] This results from a reaction occurring between the asbestos particle and surrounding tissues. It is a thickening of the fiber due to the deposition along its course of a protein matrix containing iron which probably serves to reduce the chronic irritation.[25] These bodies may be found in the sputum, lung, pleura, lymph nodes and spleen.[25] Their presence is held to be evidence of exposure to asbestos but by themselves are not necessarily an indication of asbestosis.[17,27,28]

The third and most significant tissue response is the production of fibroblasts and the deposition of collagen about the distal bronchioles and alveoli. There ensues a diffuse fibrosis which compresses the alveoli and capillaries, resulting in complete obliteration of the involved pulmonary tissue. This process is more pronounced in the lower lobes of the lung for it is there that the particles are most abundant. By x-ray one sees a fine, ground glass or granular pattern in the lower lobes and frequently emphysema in the upper lobes.

The sequence of pathologic events described previously occurs slowly. In man the fibrosis tends to progress even after the exposure has ceased; however in animals this does not seem to be the case. It may be that intercurrent infection contributes to the progression in man.[13]

In general there is a delay of five to seven years between the initial exposure to high concentrations of asbestos dusts and the onset of clinical asbestosis. The average interval reported by Merewether is eleven years.[17] While most patients with asbestosis have had an exposure of ten to sixteen years, it is important to realize that the disease has occurred with as short an industrial exposure as 0.5 years.[1,2]

Usually no symptoms appear until a large part of the respiratory reserve has been reduced by the fibrosis. Merewether has frequently commented how markedly the lungs can be affected and yet the patient be fairly comfortable.[17] However, when symptoms once begin and significant dyspnea becomes apparent, there is usually a definite and rapid progression. Then productive cough, anorexia, weight loss and fatigue are the common complaints. Death eventually results from intercurrent infection, cor pulmonale or carcinoma of the lung.

The case herein presented demonstrates many of the significant features in the pathogenesis, symptomatology and natural course of asbestosis. The patient had worked for twelve years in an atmosphere having a concentration of asbestos particles known to be sufficient to produce pulmonary pathology. However, it was only during the last year of life that dyspnea, cough, anorexia and weight loss manifested themselves. Clubbing had been present for at least five years. He had a very rapid downhill course, due undoubtedly to the two associated factors—the asbestosis and carcinoma of the lung. The physical findings of clubbing, cyanosis and dullness at the lung bases were all consistent with asbestosis as were the x-ray findings in the lungs, apart from the evidence suggesting neoplasm. The outstanding symptom, the severe and progressive dyspnea, was attributed to a combination of pulmonary fibrosis, superimposed and spreading lung neoplasm, pulmonary infection and finally congestive failure on the basis of cor pulmonale.

As indicated in the case history, the ten-day period of ACTH therapy was accompanied only by euphoria but objective measurements revealed no significant changes. This was not surprising for two reasons: (1) the fibrosis had obviously been of long duration and therefore one would not expect it to change much at this time; and (2) he had superimposed bronchogenic carcinoma. It is of interest to compare these results to patients with chronic beryllium