bined with cotton and woven as a textile for fireproof and heat-resistant clothing and other substances. Lanza[82] estimates that there are about 10,000 persons exposed to asbestos in the United States. Most observers feel that the incidence of asbestosis in American asbestos workers is quite low. Asbestosis has, however, been reported more frequently in England and Canada.

Lanza, McConnell, and Fehnel[83] concluded from their study of asbestosis that prolonged exposure to asbestos dust causes pulmonary fibrosis different from that produced in silicosis and demonstrable by roentgenogram. Clinically it resembles silicosis in that it is not disabling in the early stages, but it may be markedly disabling when advanced, and frequently leads to death by right heart failure. Although tuberculosis has been shown to be no more prevalent in persons with asbestosis than in the general population, lung cancer is under suspicion as occurring more frequently with asbestosis, especially in England.

Symptoms. The onset of symptoms of asbestosis, as of silicosis, is slow although symptoms in advanced cases are apt to be somewhat more marked than in silicosis. As in silicosis, dyspnea is the cardinal symptom. Anorexia occurs frequently in advanced stages, and cyanosis and clubbing of the fingers are apt to appear with greater constancy in asbestosis.

Pathology. The fibrosis in asbestosis is diffuse and tends to predominate in the basal portions of the lungs in contrast to the generalized nodular fibrosis of silicosis with a predominance in the upper portions of the lung. Bronchiectasis and bronchiolectasis are frequent, especially in the more fibrous portions. Whereas the proliferation of fibrous tissue is caused by chemical action in silica exposure, it is induced by mechanical action in asbestos exposures. Gardner[84] found that the fibrosis-producing character of asbestos could be almost eliminated by grinding the fibers so that no particles more than 20 μ in length were present. As previously mentioned, asbestos is classified among the inert dusts. Vorwald, Durkan and Pratt[85] report that the typical bronchiolar fibrosis is best produced by fibers ranging from 20 to 50 μ in length but not by fibers shorter than 20 μ. The significance of this finding on prevention of the disease will be discussed below.

Microscopic anatomy. Johnstone[86] described the microscopic appearance of the lungs somewhat as follows: In the early phases of the disease there is thickening of the alveolar septa which results from fibroblastic proliferation. The alveolar spaces contain numerous phagocytes. With progression of the disease fibrosis becomes more marked; the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few alveoli that remain in the area of fibrosis are lined with low cuboidal epithelium giving them an almost glandular appearance.

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