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 asbestos 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 said 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, Dunkan 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 spots 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.

[82] A. J. Lanza, *J. Am. Med. Assoc.*, **106**, 368 (1936).
[83] A. J. Lanza, W. J. McConnell, and J. W. Fehnel, *U. S. Pub. Health Repts.*, **50**, [illegible] (1935).
[84] L. U. Gardner, *Ind. Med.*, **9**, 45 (1940).
[85] A. J. Vorwald, Experimental studies of asbestosis, *Arch. Ind. Hyg. and Occupational Med.*, **3**, 1 (1951).
[86] R. T. Johnstone, *Occupational Diseases*. Saunders, Philadelphia, 1942.

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures described first by McDonald.[87] These bodies are 20 to 100 μ in length and are bulbous on one or both ends so that they appear club- or dumbbell-shaped. They are brownish in color, do not stain, and give a Prussian-blue reaction for iron. Simson[88] has produced these bodies in guinea pigs by experimental exposure to atmosphere containing asbestos. Lynch[89] concludes that these "curious bodies" signify exposure to asbestos dust but do not necessarily indicate asbestosis. In other words, they are more properly referred to as "asbestos bodies" and not "asbestosis bodies."

X-ray examination. For an excellent review of roentgenographic findings in both silicosis and asbestosis the reader might well refer to Pendergrass.[90] Characteristic differences in the roentgenographic findings in silicosis and asbestosis are tabulated below:

Asbestosis	Silicosis
Fibrosis diffuse (film may have ground glass appearance from pleural involvement).	Fibrosis nodular.
Findings may be either bilateral or unilateral.	Findings characteristically bilateral.
Lesions largely in the lower one half or two thirds of the lung fields.	Lesions predominantly in the upper two-thirds of the lung fields.
Emphysema in upper portion of lung fields.	Emphysema in lower portion of lung fields.
Borderline degree difficult to recognize.	Borderline degree more easily recognized.

Control. Prevention of asbestosis depends largely on preventing exposure to sufficiently high concentrations of long fibers to produce the characteristic reaction. Also, it is only long fibers cause the disease, fine dust respirators are not necessary. The more comfortable gauze respirators appear to be perfectly adequate filters for the long fibers. Up to this time, however, they have not been approved by the United States Bureau of Mines for this purpose.

Allowable concentrations. Dreessen, DallaValle, Edwards, Miller, and Sayers[91] have found evidence to indicate that 5 million particles per cubic foot of air is a satisfactory figure for the maximum permissible atmospheric concentration of asbestos to which workers may be exposed. However, in setting up this figure only fibers less than 10 μ in length were counted and the longer ones were disregarded. It now appears that the shorter fibers may have to be disregarded and permissible concentrations of long fibers established, which has not been done.

The prognosis with early to moderately advanced asbestosis is good, provided the inhalation of asbestos fibers is materially decreased. Unlike silicosis,

[87] J. McDonald, *Brit. Med. J.*, **2**, 1025 (1927).
[88] J. W. Simson, *Brit. Med. J.*, **1**, 885 (1928).
[89] K. M. Lynch, *J. Am. Med. Assoc.*, **109**, 1947 (1936).
[90] E. P. Pendergrass, "Roentgen-Ray Diagnosis in Silicosis and Asbestosis," in A. J. Lanza, ed., *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.
[91] W. C. Dreessen, J. M. DallaValle, T. I. Edwards, J. W. Miller, and R. R. Sayers, *U. S. Public Health Bull.* No. **241**, 1938.