majority of asbestos miners in the Ural Mountains are said to have on the inner surfaces of the right hand three to five or more papular, non-inflammatory, wart-like formations of round or polygonal shape reaching the size of a pea (Dewirtz). They consist of hyperkeratoses sometimes associated with acanthosis and prolongation of epithelial cell pegs. There is some lymphocytic infiltration in the cutis. Between the cornified masses asbestos needles are found. Many mitoses may be present in the thickened malpighian layer, and large giant cells with centrally located multiple nuclei may be present in this stratum. These lesions will persist unless the asbestos needles are removed. The warts do not exhibit any appreciable proliferative tendencies.

Pulmonary Lesions (Asbestosis). The asbestotic pneumoconiosis, resulting from a prolonged occupational inhalation of asbestos dusts and fibrils, is a condition more serious than the cutaneous manifestations. The first fatality of asbestosis was reported by Murray in 1899. It was not until 1914 that a record of an asbestosis death was published in Germany (Fahr). Fatalities from asbestosis as the main or contributory cause of death were published in the United States by Lynch; Stewart, Bucher and Coleman; and Shull. The Metropolitan Life Insurance Company recorded, between 1924 and 1936, seventeen cases in which asbestosis contributed to the lethal outcome of the main disease.

In Germany there was no appreciable interrelation between asbestosis and tuberculosis of the lung, such as exists regarding silicosis. In England there were 30 cases of asbestosis complicated by tuberculosis in a series of 100 cases of this type of pneumoconiosis reviewed by Wood. Similarly, in the United States a certain relation to tuberculosis was apparent, but the coexistence of the two diseases was not as frequent as in the case of silicosis (Stewart, Bucher and Coleman; Shull; and Lanza, McConnell and Fehnel). The development of a pulmonary asbestosis depends, according to Bauer, upon the duration and degree of exposure to asbestos dust, and on a certain personal disposition. A similar observation was made by Sweany, who remarked that some people develop an asbestosis rapidly, while others may be exposed to the same conditions for years without detriment. Individual differences in the filtering qualities of the nasal passages are mentioned by Lehman as possible reasons for such discrepancies in susceptibility.

Asbestosis usually develops after an exposure of three to fifteen years (Martz), but may not produce subjective symptoms until many years after the cessation of exposure to asbestos material. The first symptoms are dyspnea and a cough with expectoration. Later there is pallor, cyanosis, palpitation of the heart, loss of appetite and weight, and piercing pains in the chest during breathing. The shortness of breath becomes aggravated during physical exertion. In the beginning the physical examination reveals scarcely any pulmonary changes; but later there appears a diminution of resonance, especially over the lower parts of the lung (particularly the right side), where dry and