

This considerable gap in the theory impedes the solution of such major practical problems as the standardization of asbestos-bearing dust, the individual preventive medicine and therapy of asbestosis.

For many years the special fibrous structure of asbestos was examined as the basic reason for its pathogenic effect. Gardner, Cummings (1931), Mottura (1939), King et al (1946), Behrens (1949), Vorwald et al (1951) and many others consider that only rigid, relatively long asbestos fibers, introduced in the tissue of the lung, cause its traumatization and, as a consequence, sclerosis.

Seemingly testifying in favor of such hypotheses are such facts as the more considerable fibrous changes during the introduction of long filaments (15-20 μ) and the insignificant changes during the introduction of particles of respirable fractions (< 7 μ) (Vorwald, 1951, Szimczikiewicz, Wicek, 1962, Klosterkötter, 1968).

Another argument is the development of fibrosis predominantly around the place of the implantation of filaments, peribronchially, which is explained by the mechanical trauma of tissue during respiratory movements. Also given are such facts as the absence of fibrosis in lymph nodes and the development of fibrosis under the effect of brucite, a fibrous mineral of nonsilicate nature ($MgO \cdot H_2O$).

At the same time the particles of serpentine, the rock which carries asbestos, according to Vorwald et al, are not capable of causing fibrosis. The situation comes down to the fact that the industrial dust taken at Canadian factories "gave rise to," according to the head physician of Canadian enterprises Cartier (1949, 1955), a large number of patients sick with asbestosis.