alveolar ducts in which the walls of the associated air spaces were very thick, owing to swollen collagen framework. Connective tissue and Foot-Bielschowsky silver preparations revealed complete loss of capillary bed locally. Outside the collagen was a thin layer of epithelial cells. This did not resemble the "adenomatoid" change characteristic of guinea pig asbestosis. Near the lesions the air spaces were filled with phagocytes containing gray to yellow particulate dust and a rare, long, naked asbestos fiber. Careful search failed to reveal even a suggestion of an asbestosis body. Pleurisy was absent. The tracheobronchial nodes showed compact focal collections of monocytic cells at 12 months and, at 20 months, some diffuse thickening of the reticulum. In a few rats there was definite fibrosis along the margins of the node, extending into the mediastinal areolar tissue.

Results of chemical analyses made on the white rats are given in table 7, and the average values have been recorded in table 8 for comparison with similar values for rats inhaling other dusts. It will be noted that the values for asbestos are lower than those for quartz or chert but approximate those for the gypsum-quartz mixture, in which atmospheric agglutination tended to reduce the amount of dust inhaled. This condition prevailed even though the atmospheric concentration of asbestos dust was essentially the same as that of the quartz, was one-half that of the gypsum-quartz mixture and was one-fifth that of the ferruginous chert. Since the values for asbestos are low, it might be inferred that the total quantity of that dust actually inhaled was small or that it had been eliminated from or dissolved within the lungs. Evaluation of these possibilities is not feasible on the basis of the observations derived from this study.

*Reaction in Cats.*—Twenty cats were used in this inhalation experiment with the short fiber asbestos. Eighteen were kept in the dust room continuously until put to death, the exposure period ranging from one month to nearly 54 months. The other two were removed to normal air after a dust exposure of 31 months; one of these was killed five months, and the other 24 months, later. In general, the tissue response was confined to microscopic foci of fibrosis, which were in the walls of groups of subpleural alveoli rather than in the peribronchiolar areas. In one animal the change was extensive enough to be visualized on gross inspection of the section. Only in the animal with the longest exposure—54 months—did the roentgenogram reveal definitely abnormal shadows. A roentgenogram made after 30 months revealed no abnormality; after 45 months, a faint mottling could be detected throughout both lungs. At autopsy, nine months later, there was only microscopic fibrosis in the subpleural zone plus heavy lymphocytic infiltration about small bronchioles. Asbestosis bodies were rare. On prolonged search a few yellow atypical bodies, smooth and without haustrations, were found in two animals exposed for more than a year.

*Reaction in Rabbits.*—Eight rabbits were exposed to dust for periods extending from one to more than five years; the last animal was removed from the dust room and left in normal air six months before being killed. There was never enough pulmonary fibrosis to be detected grossly, and there was no chronic adhesive pleurisy. Microscopic evidence of alveolar wall thickening was first detected in one animal after about three years of exposure and was seen in all five animals examined thereafter, including the one removed to normal air. One animal that died of paralysis after nearly four years of exposure exhibited a reaction visible on gross inspection of tissue sections. The possibility of pulmonary infection in this animal could not be excluded. In another animal dying two years later the focal fibrosis was not nearly as obvious or as advanced. Areas of involvement, which were largely visualized because of phagocytic reaction within the air spaces, tended microscopically to become more fibrous with the passage of time, but there was never much encroachment on the lumen of air spaces and the structure of the lung was preserved. Asbestosis bodies were not detected in rabbits that died early in the experiment but were seen in all animals that had been exposed to the dust for more than three years.