continuously, until sacrificed, 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 sacrificed 6 months, and the other 34 months, later. In general, the tissue response was confined to microscopic foci of fibrosis 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 X-ray reveal definitely abnormal shadows. After 30 months the picture was negative; after 46 months, a faint mottling could be detected throughout both lungs. At autopsy, 9 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 6 months before being sacrificed. 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 3 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