lived in a normal atmosphere for an additional 24 months. Exposure for 14 months was sufficient to produce cellular accumulations of phagocytes around terminal bronchioles and peripheral arterioles together with compact collections of similar cells in the tracheobronchial lymph nodes. At that time there were no typical asbestosis bodies, but smooth, pointed, yellow fibers were seen very rarely. With continued exposure, up to 42 months, reaction in the locations noted progressed to the formation of cellular connective tissue which made well-defined sheaths about the respiratory bronchioles and arterioles, marked lymphoid hyperplasia and lymphoid infiltration of bronchiolar walls (fig. 16). Typical asbestosis bodies were not formed although there was an occasional fiber, smooth, yellow and pointed. Pleurisy was not present. The reaction was similar in location to that in the guinea pigs, but fibrosis was much slower in development. Roentgenograms of cats made after exposure periods of 25, 33 and 42 months, respectively, failed to demonstrate evidence of pulmonary pathology.

Reaction in Rats. Although 20 rats were placed in the dust room, many died from pneumonia and were not suitable for study. Five animals, of which one was exposed for 19 months and four for 25 months, were free from pulmonary infection and offer a basis for tentative conclusions. In the 19-months animal, the reaction was just beginning. All four animals sacrificed at 25 months showed a well-marked peribronchiolar fibrosis. After a long search only two small smooth asbestosis bodies were found in the 19-months animal and none was found in the 25-months animal. Thus, these animals exhibited fibrosis without asbestosis bodies or fibrosis accompanied by only a very infrequent asbestosis body.