installed, the dust counts were higher, and the over-all average for the remaining 21 months was about 150 million.

Size-frequency studies of atmospheric dust collected inside the animal cages revealed that nearly 99 per cent of the components suspended in the air could be classified as clumps or particles; only about 1 to 1.5 per cent was fibers. One third to one half of the fibers were longer than 10 microns, indicating a concentration of long fibers of about 0.8 million. This figure is about one-half the estimated value of 1.4 million for the short fiber experiment.

Guinea pigs, rats and mice were used in the inhalation experiment with the 100 per cent ball-milled asbestos dust. The results are summarized in table 9.

Reaction in Guinea Pigs.—The experiment was started with 100 guinea pigs. As the dust exposure proceeded, there were 39 accidental deaths, 32 of these being due to pneumonia in an epidemic. The 61 pigs remaining exposed to the dust were killed at intervals during exposure, except for 16 guinea pigs transferred to normal air after 28 months of dusting. For the first year of exposure practically the only reaction to the dust was the presence of scattered phagocytes and an occasional minute asbestosis body. At 16 and 20 months no gross response was visible on the tissue section, but microscopically peribronchiolar foci of inflammatory cells

TABLE 9.—*Summary of Inhalation Experiment with 100 per Cent Ball-Milled Asbestos Dust*

Nature of Experiment	Animals	Maximum Dust Exposure, Mo.	Maximum Survival After Dust Exposure, Mo.	Results
Dust exposure continuous throughout life	84 guinea pigs	24	0	No appreciable pulmonary reaction
	40 rats	20	0	No suggestion of asbestosis
	24 mice	12	0	No suggestion of asbestosis
Dust exposure followed by prolonged residence in normal air	16 guinea pigs	28	12	Fibrosis typical of asbestosis was present 12 mo. after exposure ceased in an amount sufficient to be visible grossly; smaller foci could be seen microscopically at 2 mo. and 8 mo. after termination of exposure

could be seen. At 24 months (fig. 6*A*) there was still no change large enough to be seen with a hand lens, although microscopic examination revealed cellular accumulations about terminal bronchioles and many more asbestosis bodies, chiefly within cells. The lungs of animals exposed for the full dusting period of 28 months and afterward living in normal air for two months revealed the changes described above and also very slight peribronchiolar fibrosis. For exposed animals living eight months in normal air the findings were similar, but at 12 months three of four animals showed grossly visible characteristic peribronchiolar fibrosis with adenomatoid change (fig. 6*B*).

The tracheobronchial nodes were essentially normal until exposure had been continued for more than a year and a half. Animals killed at 12 months and at 16 months revealed a few minute collections of phagocytes containing particles but practically no fibers large enough to be recognized as such. After 20 months of exposure many monocytes filled with yellow granules were present. At 30 months there had been a slight increase in reticulum but no fibrosis. No further changes occurred in the nodes. Asbestosis bodies were not seen in the nodes of any of the guinea pigs.

Minute asbestosis bodies were observed in the lungs as early as three months after exposure began, but they did not become numerous until 16 months had elapsed. The bodies were short and practically all were intracellular, although at 20 months some were long enough to project beyond the cell borders. It is