with little or no fibrosis continued to progress. It is assumed that following attainment of fibrotic maturity, the same process of contraction would ensue as was noted for the mature lesion.

G. The formation of asbestosis bodies represents a coating of the fibers by blood and tissue elements, which results in loss of ability of the fiber to produce fibrosis.

Intratracheal injection of asbestosis bodies failed to produce the typical asbestotic tissue reaction in experimental animals. The cessation of progressive reaction to inhaled asbestos dust soon after exposure terminates may be due to the formation of asbestosis bodies.

H. Aluminum hydroxide failed to neutralize the fibrosing action of the long-fiber asbestos.

Intratracheal injection of aluminum hydroxide with chrysotile asbestos did not retard or prevent the development of asbestosis in rats.

I. Inhalation of asbestos dust did not alter significantly the final outcome of experimental tuberculosis in two series of guinea pigs exposed to the dust.

The apparently mild influence of asbestos dust is in distinct contrast to the stimulating effect of inhaled quartz upon a tuberculous process in the lung. The interpretation must remain tentative, however, since it is based upon an investigation limited to two series of guinea pigs exposed to only one kind of asbestos, namely, King's floats. Reference to table 4 shows that when the infection was coincidental with the onset of dust exposure, there