lesions, due to contraction of the fibrous tissue. In contrast, an immature tissue response, evidenced primarily by cells 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 observed 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.

Aluminum hydroxide added to the suspension of chrysotile asbestos prior to intratracheal injection 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 exerted by inhaled quartz on a tuberculous process in the lung. The interpretation must remain tentative, however, since it is based on an investigation limited to two series of guinea pigs exposed to only one kind of asbestos, namely, King's floats: Table 4 shows that when the infection was coincidental with the onset of dust exposure, there was temporary progression of the infectious process, with subsequent healing; when infection was initiated after 26 months of dust exposure, the course of the tuberculosis was not appreciably altered. The latter finding is quite different from our usual experience with quartz dust or with mixed dusts containing quartz, wherein the adverse influence of quartz on a tuberculous infection is manifested most strikingly when infection is initiated after a period of dust exposure, viz., superimposed on a background of established silicosis. As indicated above, this more sensitive test, when applied to asbestos dust, failed to demonstrate that the latter had an adverse influence on a tuberculous infection. The inability of asbestos dust in that experiment to affect unfavorably the tuberculous process furnishes strong support for the interpretation that inhaled asbestos dust has no more than a mildly unfavorable effect on pulmonary tuberculosis.

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