16 months a delicate fibrosis made its appearance. The process evolved gradually and the number of fine intercellular collagenous fibers steadily increased. As this fibrosis contracted, it partially closed and distorted the alveoli, and with this change the alveoli became lined with cuboidal cells. The result was an adenoma-like appearance which frequently accompanies chronic pulmonary inflammation resulting from many causes. Willis[10] described a similar structure in the lungs of guinea pigs inhaling silicon carbide. The longer exposures to asbestos resulted only in more thickening of the walls of the air spaces, largely due to an increase in the amount of fibrosis. The fibrous tissue always remained cellular and failed to show the hyalinization characteristic of silicosis. Asbestosis bodies (fig. 3), first seen in the lungs of the guinea pigs that had inhaled dust for about 2 months, became more numerous and more distinctly segmented with increasing exposure.

The reaction produced in guinea pigs exposed for 6 and 9 months did not progress significantly during a subsequent period of 35 and 37 months when the animals lived in a normal atmosphere (figs. 6 and 7). Between 8 and 11 months after exposure ceased, the cellular reaction in the lung had been completely replaced by thin strands of fibrous tissue. At later periods, the scar tissue was less in amount but in the last animal sacrificed, 37 months after discontinuing dust exposure, some fibrosis was still visible.

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10. Willis, H.S. and Brutsaert, P.: Tumor-like Structures in the Lungs of Guinea Pigs Artificially Exposed to Silica Dust. Am. Rev. Tubere. 17: 268, 1928.