

were bundles of smooth muscle that often constituted masses 50μ to 100μ in thickness (Fig 6). These muscle bundles were sharply delimited and related to the wall of thickened alveolar ducts. They had no connection with blood or lymph vessels nor could a relationship to the muscularis of a terminal bronchiole be demonstrated. In the newly formed connective tissue about some of the alveolar ducts and respiratory bronchioles, there were scattered gland-like structures. These pseudo-glands represent surviving units of the evaginating alveoli that have become lined by tall columnar epithelium, occasionally ciliated. No acid-insoluble mineral ash was demonstrable in the area occupied by the proliferated muscle fibers. The regions, pervaded by the more cellular and active inflammation, some containing leukocytes, were also devoid of demonstrable mineral ash.

Hamsters (Intratracheal Injection).—Of 19 hamsters, 16 died as a result of the asbestotic inflammation that followed the intratracheal injection of chrysotile dust. The three survivors had received only one injection and were killed 21 months after the imposition of the lung dust burden. The average survival of the hamsters that had one intratracheal injection was $18\frac{1}{2}$ months, with a range of $13\frac{1}{4}$ to $20\frac{1}{4}$ months. The animals that had received two intratracheal injections survived, on the average, $14\frac{1}{4}$ months from the time of the first injection, with a range of $12\frac{1}{2}$ to $16\frac{1}{4}$ months. The three survivors that were killed after 21 months were emaciated and also had extensive asbestotic disease.

Many of the lung sections showed extensive consolidation with relatively few patent air spaces. In other lung sections, the consolidation was patchy, seemingly concentrated about larger air spaces with relatively little aerated tissue between the consolidated portions. The consolidated portions were composed of masses of cells with vesicular round or oval nuclei—apparently alveolar cells (Fig 7). Leukocytes were not seen. Alveoli or larger air spaces usually could not be identified in these regions except after silver impregnation (Fig 8). The argyrophilic mural stroma of all air spaces in the solidified regions was greatly thickened by arborescent fibers that formed a loose network

encroaching upon the lumens. Occasionally, the argyrophilic stroma extended into the lumen of an alveolar duct or a bronchiole in a polypoid manner, occluding it. The condensation of thin argyrophilic fibers to form parallel thick fibers devoid of arborescence and the transformation into collagen, characteristic of the lesions in rats, was not seen at all or to a very minor extent in these hamsters. For the most part, the occlusion of the air spaces was caused mainly by massed alveolar cells, but the contribution of the argyrophilic stroma should not be dismissed. It should also be emphasized that in contrast to the situation in rat lungs where the involvement was sharply limited to the alveolar duct and its evaginating alveoli, there was no such delimitation in the hamster lung. Here, the cellular as well as the stromal proliferation extended without abatement to the peripheral alveoli, thus accounting for the diffuseness of the consolidation.

Following incineration, abundant acid-insoluble mineral dust was observed in the involved racemi. Many individual fibers could be identified; some straight or wavy, others coiled and forming tangled masses. Compared with the amount of dust found in rat lungs, the chrysotile dust in the hamster lungs was much more abundant and more diffusely distributed (Fig 9). Asbestos bodies were found in abundance throughout the affected regions. They were considerably smaller than those seen in human lungs but were readily recognized. They were best seen under dark-field illumination with dry objectives in unstained sections (Fig 10). However, there was no parallelism between the number of luminescent asbestos bodies seen in the unstained sections and the amount of dust found in the same field after microincineration. The amount of dust present was much greater than might be suspected from the number of asbestos bodies observed. Most of the dust and the asbestos bodies were situated in the air spaces. When observed within tissue, the asbestos bodies and the dust were found trapped within the network of inflammatory argyrophilic fibers and associated cells that tended to occlude the air spaces.

Guinea Pigs (Inhalation).—Barely recognizable, minimal mural thickening of an occasional alveolar duct and respiratory bron-