Pathology and morbid anatomy of asbestosis

In crushing operations, fiberizing and disintegrating, in opening, spinning, weaving and the filling of asbestos mattresses, production of fine thistledown dust occurs. This consists of fine needles or spicules of asbestos, which are inhaled by the workmen. Most of the dust inhaled never reaches the lungs, but is arrested in the upper respiratory tract and trachea, from which it is rejected in the nasal mucus and sputum. Whereas only the minute particles of silica gain entrance and are retained within the lungs, much larger particles of asbestos, when inhaled, because of their physical form, are mechanically trapped. Fibres up to 200 microns have been detected in the air passages, but dangerous fibres measure up to 20 microns. In the lung no local necrosis results, nor is there any leucocytic reaction. As a result of mechanical irritation the pulmonary epithelial cells are desquamated, and fibroblasts appear round bronchioles and alveoli, in interlobular septa and in the subpleural tissue. Finally, collagenous fibres appear around the distal ramifications of the bronchial tree.

Gardner and Cummings (1931) have shown, by animal-inoculation experiments, that the chief site of asbestos-dust localization is the respiratory bronchiole.

They describe the early lesion, in the guinea-pig exposed to inhalation of asbestos dust, as follows:

> "The asbestos dust is carried by the inspired air only to the distal respiratory bronchioles . . . The mononuclear phagocytes from the adjacent connective tissues enter the lumen of the air passages and engulf the particles. The phagocytes with their contained fragments of dust are frequently pushed aside into the alveoli which pouch out of the sides of the bronchioles, where many of them remain indefinitely. More dust entering the lung is held up by the partial obstruction created, and further reaction is largely proximal to the point of original localization."

At 60 days the authors describe the dust-cells of the terminal bronchioles as being increased, and the lumina of some of the lateral alveoli completely blocked with mononuclear phagocytes and giant cells. At the end of 2 months small asbestosis bodies appear. After 90 days' exposure it was found that the amount of cellular reaction had not kept pace with the number and size of the asbestosis bodies. Hyperplasia of the lymphoid tissues was now visible. At the end of a year the walls of the bronchioles proximal to the alveolar ducts were thickened from excessive local accumulation of phagocytes, but definite proliferation of fibroblasts was not noted until after 550 days. This was confined to the walls of the air spaces adjacent to the deposits of dust phagocytes. At the end of a further 100 days a true fibrosis developed, contracting the air spaces, which now appeared to be filled with compact masses of dust cells and phagocytes, fibres and bodies. After 2 years the mischief had reached the periphery of the lung, and sub-pleural greyish-white nodules were uniformly distributed over the surface.

King, Clegg and Rae (1946) have recorded the results of experiments in rabbits exposed to the intratracheal injection of asbestos fibres of varying length. In

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