1. The characteristic early asbestotic lesion is situated in, and sharply localized to, the proximal portion of the pulmonary racemus which, in the rat, consists of a very short respiratory bronchiole and adjoining alveolar ducts.

2. Asbestosis in rats, caused by chrysotile dust, is nonprogressive.

3. The evidence for the nonprogressiveness of chrysotile asbestosis in rats consists of the observations that the minimal asbestotic lesion is limited to the wall of the respiratory bronchiole and of the adjoining alveolar ducts and does not extend into adjoining normal alveoli and that the minimal asbestotic lesions heal by becoming collagenized and hypocellular.

4. The localization of the minimal asbestotic lesions is attributed to the proximal transport of dust from peripheral air spaces by the alveolar clearance mechanism and subsequent stagnation of the transported dust in the proximal portion of the racemus.

5. In rats chrysotile asbestotic lesions heal in the absence of demonstrable (optical microscope) asbestos bodies and in the presence of chrysotile fibers, the latter becoming trapped in the scar tissue.

6. There is a considerable reduction in the amount of chrysotile dust found in the lung sections of rats one year or more after the imposition of the lung dust burden as compared with the amount of dust present shortly after the dust burden was imposed.

7. The reaction of hamster lungs to chrysotile dust is the antithesis of that observed in rats. The lesion is progressive, extending into peripheral air spaces so as to involve the entire racemus and thereby producing extensive consolidations leading to the death of the animals. The lesions do not heal, since the precollagenous stroma and the high cellularity persists through the two-year period of observation. At the same time, the amount of asbestos dust demonstrable in the sections remains large and diffusely distributed.

8. The progressiveness of the chrysotile asbestotic lesion in hamsters is ascribed to a less effective pulmonary clearance mechanism and to a greater reactivity of the pulmonary tissue than exist in rats.

9. The early chrysotile asbestotic lesion in guinea pigs resembles that of rats, but it has not been adequately studied to permit further characterization.

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