chiole was found in the lungs of guinea pigs that had been exposed for three months to an atmosphere containing 20 mg/cu m of chrysotile dust. Similar to the appearance of such lesions in hamsters, the mural thickening consisted of proliferated alveolar cells and their supporting stroma of interconnecting argyrophilic fibers. The lung sections of guinea pigs that had inhaled high concentrations (86 mg/cu m) of chrysotile dust for two weeks or longer, manifested similar minimal mural asbestotic thickening in the proximal portion of the racemus. However, the number of units (racemi) involved was much greater than that in the animals exposed to the lower dust concentration for three months. No evidence of healing (significant reduction in cellularity of the lesion and collagenization of the stroma) was seen in animals examined up to seven months after the beginning of the exposure. Following microincineration and treatment with acid, the amount of ash seen in the guinea pig lung sections was comparable to that noted in rats that had inhaled chrysotile dust. Asbestos bodies found in the lung sections were few and very small.

Comment

A chronic pulmonary inflammation may be termed progressive if it extends from its original site into adjoining, previously normal air spaces, and if it remains active, retaining its argyrophilic precollagenous stroma and high cellularity. Such a pulmonary inflammation may be considered healed if its argyrophilic stroma has been completely collagenized while its cellularity has become considerably reduced.

In the rat that has inhaled high concentrations of chrysotile asbestos fibers for only a few months or has been injected intratracheally with this dust, the asbestotic inflammation remains sharply limited to the proximal portion of the racemus and heals by becoming transformed into a hypocellular collagenous scar. Thus, it would appear proper to classify asbestosis caused by chrysotile dust as nonprogressive in the rat.

It is of basic interest that appreciable amounts of asbestos fibers are demonstrable within the scars of healed or healing inflammation. This would suggest that the disappearance of chrysotile dust from the lesion, either by dissolution or by transport, is not a sine qua non of healing; but that healing, at least in the rat, does take place—even in the presence of this dust. One would like to think of the formation of the asbestos body as a protective mechanism by which the asbestos fiber becomes sequestrated and the tissues safeguarded from further irritant action by the fiber. To what extent this mechanism may apply is not known; but it does seem that in rats, in which asbestos bodies are not demonstrable (with the optical microscope),[6] healing occurs in the presence of apparently naked fibers.

The diminution, in time, of the amount of dust demonstrable in the tissue and its apparent disappearance in some of the scars, poses an interesting question in regard to the mechanism by which the asbestos fibers disappear. It is commonly believed that chrysotile fibers have a relatively high solubility in tissue fluid. This would seem to account for the inability to demonstrate asbestos fibers in some of the asbestos bodies found in human asbestotic lungs. However, dissolution of the fibers, particularly of the coarser ones, would require a long time—so that it would be difficult to explain the failure of peripheral alveoli to become involved by inflammation. Furthermore, high solubility would not be consistent with the sharp inflammatory localization of the asbestotic lesion in the proximal portion of the racemus in rats. It is much more reasonable to explain this sharp localization of the inflammation on a fairly prompt removal of the inhaled or injected irritant dust from the peripheral alveoli and the subsequent stagnation of the dust in the proximal portions of the racemus.[7] The transport of the chrysotile dust from the peripheral alveoli is effected by the alveolar clearance mechanism consisting of a proximally moving film of alveolar fluid.[8] This transport is dramatically illustrated by the increase in, and concentration of dust in the lumen of the alveolar duct within 72 hours after the intratracheal injection of chrysotile dust (cf Fig 1 and 2). It may be of interest at this point to note that the localization of the early asbestotic lesion to the proximal portion of the racemus was first described by Vorwald et al,[6] and recently confirmed by Holt et al.[9]