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Experimental Asbestosis

Studies on the Progressiveness of the Pulmonary Fibrosis Caused by Chrysotile Dust

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THE unreliability of symptoms as a criterion for the progression of pulmonary fibrosis was underlined in a medical and psychiatric study of coal miners with respiratory complaints. Here, a very significant association was observed between a recognizable psychoneurotic factor and an apparent worsening of the clinical condition: of 40 patients studied, only 12.5% had disability on "physical" grounds alone; 35% had disability on psychiatric grounds alone; and the remainder on both.[1]

Furthermore, discerning clinicians recognize that opinions concerning the progression of a pulmonary fibrosis based upon the symptomatology (eg, shortness of breath) and a roentgenologic evaluation, even when reenforced by lung function studies may be quite erroneous. In the presence of a superadded coincidental pulmonary illness, such signs and symptoms may be reversible in whole or in part between such episodes, which include covert and overt acute pneumonitides, pulmonary edema, and allergic or chronic bronchitis—with or without emphysema, or emphysema without bronchitis. Such diagnostic difficulties were the subject of a recent symposium on emphysema in industry.

As uncertain as are the clinical criteria of progression of pneumoconiosis, very little help has come from postmortem studies of human lungs or those of experimental animals in defining the criteria by which progression of pneumoconiosis may be determined. This is particularly true of asbestosis.

It is the purpose of this paper to define some of the anatomic stigmata of progression of asbestosis and to determine whether or not asbestosis caused by chrysotile dust is progressive.

Methods and Materials

Lung burdens of chrysotile asbestos dust were imposed upon rats, hamsters, and guinea pigs by exposure in inhalation chambers as well as by intratracheal injection. Most of the animals exposed to dust in an inhalation chamber and reported in this study were part of a larger investigation that will be reported separately.

The inhalation chamber in which all animals (except four guinea pigs) were exposed to dust, measured 8 × 8 × 8 feet. The animals were housed in wire cages that were suspended in racks. Periodically, these cages were rotated so that inequalities in dust exposure caused by position were largely obviated. The exposures were for six hours per day, five days per week. The total exposure varied, as listed in the table.

The chrysotile asbestos was ballmilled and then fed into a hammermill (modified from a design by Holt and Young[2]). This was provided with inlet and outlet tubing so arranged that the comminuted asbestos was fed back continually into the hammermill. At the same time, the ultrafines were allowed to waft upward into the inhalation chamber. Two of these hammermills provided the chamber with sufficient dust to average 86 mg/cu m with a range of 42 mg/cu m to 146 mg/cu m. The asbestos dust cloud was evaluated by sampling with a two-stage size selective device, similar in design to that proposed by Wright[3] and operated at 20 liters/minute. The first stage of the instrument was a horizontal elutriator with selector characteristics recommended by the Johannesburg Pneumoconiosis Conference of 1959, namely acceptance of all particles having terminal settling velocities greater than that of a 7.1μ sphere of density 1 gm/cu cm, 50% acceptance

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