exposure to high concentrations of dust cannot be ruled out. Three cases were found with 3, 3½ and 4½ years work in asbestos, respectively, but in each there was previous exposure to other dusts (see p. 10).

Support for this view is provided by (1) Professor Beattie's experiments previously mentioned, which demonstrated that the lungs of guinea-pigs exposed for 43 and 67 hours to asbestos dust showed "definite cellular proliferation, though not very extensive, and this is certainly a preliminary stage in the production of fibrosis"; (2) Simson's case mentioned above, and his report in the same paper on the lungs of a guinea-pig exposed by Mavrogordato to asbestos dust for 100 hours during a period of 56 days. On the death of the animal (from causes other than asbestosis) some 32 months later, sections of the lungs showed a slight generalised fibrosis. In commenting on the amount of fibrosis found, he states that "a comparison between the human cases and the experimental animal showed that the fibrosis was more rapid and extensive in the human cases than in the experimental animal," and again "the amount of fibrosis in two of the human cases was quite rapid, and if due to the presence of asbestos dust, the initial rate of production was rapid when compared with present day non-infective silicosis on the Rand"; (3) data from this investigation, in which 21 of the 363 workers showed signs suggestive of commencing fibrosis (p. 10). Of these 12 had been employed for less than 7 years, and 6, for between 4 and 5 years.

To sum up, therefore, it appears probable that concentration of dust and length of exposure as factors in the production of fibrosis are interdependent within certain limits. While it seems necessary for the production of generalised fibrosis of the lungs that a definite minimal quantity of dust must be inhaled, the lower the concentration of dust in the air breathed, the longer the lapse of time before the fibrosis is fully developed, and within a certain limit, the higher the concentration of dust, the sooner the fibrosis becomes fully developed and the more intense the involvement of the lung tissue.

If this hypothesis is correct, and the evidence points to it, the practical inferences are of very great importance, since it follows that the application of measures resulting in the reduction of the concentration of dust in the air in the neighbourhood of dusty asbestos processes will cause, firstly a great increase in the length of time before workers develop a disabling fibrosis, and secondly, the almost total disappearance of the disease, as the measures for the suppression of dust are perfected.

Disablement produced by the Asbestos Fibrosis.—Regarding the amount of disablement produced by the development of pulmonary fibrosis in asbestos workers,—for a number of years this is surprisingly slight, even more so than is generally the case in silicosis. This is partly due to the character of the disease, and partly to the nature of the work, which in the majority of these processes does not involve much physical exertion. The affected person may, and often does, continue at work with occasional intermissions, latterly, due to exacerbations of bronchitis, until the condition is advanced, although he suffers increasing inconveniences from shortness of breath, on exertion. Sometimes a terminal broncho-pneumonia, or other acute infection, commences while still at work, and there is no long period of invalidism.

There is no doubt but that fibrosis of the type produced by asbestos can of itself lead to complete disablement and to a fatal termination, and this in the absence of a superadded tuberculous infection.

Particulars have been collected up to the end of 1929 of 10 cases in which an advanced degree of the asbestos fibrosis without tuberculosis was the primary cause of death. In 9, the cause of death was verified by post mortem examination and in the 10th, repeated clinical, radiological and sputum examinations confirmed the diagnosis. In an 11th case, post mortem examination showed that a lobar pneumonia had supervened upon lungs already the seat of a moderate degree of the asbestos fibrosis. With one exception, all these deaths occurred in the years 1927-29.

The length of exposure to asbestos dust in these cases varied between 9 and 24 years.