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in an asbestos cloth factory ($2{\cdot}25$–$4{\cdot}0 \times 10^6$ particles (1–2 μ) per litre of air) complained of dyspnoea on exertion and was somewhat cyanosed. Radiographically the fibrotic condition progressed in the middle and lower lobes until he died 7 years later. Necropsy findings were of the classical kind: diffuse fibrosis, thickened pleurae, no nodulation, many asbestosis bodies with giant cells and many mineral particles in the lungs.

Dust particles were found also in the vessels of the liver, kidney and spleen. No mention is made of metaplasia or neoplastic changes. These authors discount the possibility of a chemical influence of asbestos particles on the pulmonary tissues.

We have been at some pains, but without success, to elicit any statement or evidence in the literature of asbestosis which could be interpreted as more than a verbal attribution of the effects of asbestos particles to irritation. For the fibrotic effects there does not seem any reason to seek causes other than sharp "insoluble" foreign bodies larger than a critical size. The work of King, Clegg and Rae (1946) in which they pursued the mechanical action theory of Gardner, presented a reasonably full picture of the response of the lungs of rabbits to insufflation of small ($2{\cdot}5$ μ) and large (15 μ) asbestos fibres. Gardner and his school had emphasized that, as we have already mentioned, with asbestos particles size is everything, small particles being innocuous, big particles hazardous, and that this distinguishes their effects from those of silica and quartz which are entirely the result of small particles. Nevertheless, King, Clegg and Rae did find some effect from the inhalation of small ($2{\cdot}5$ μ) particles of asbestos, which might be described as a diffuse fibrous tissue reaction affecting mainly the interstitial tissue and alveolar walls with hyperplasia of the bronchial nodes and some nodular areas of giant-cell reaction. Phagocytosis of these small particles was probably easy enough to restrict the reaction to the locations mentioned. With larger fibres (15 μ), the foreign body giant-cell processes were apparently overlapped by a more permanent reaction, nodular in distribution, which fixed the asbestos particles *in situ*.

King, Clegg and Rae make the significant comment that these nodular, somewhat acellular, areas of intra-alveolar connective tissue were strictly comparable to, if less intense than, those produced by quartz, which means that the lung does not distinguish them.

In respect of small particles they suggested that if fibres $< 2{\cdot}5$ μ in size were insufflated, they would be completely removed from the alveolar wall and fail to produce interstitial fibrosis. It seems possible that in the case of Luton and his colleagues the smaller particles were phagocytosed and discharged into the circulation in the way probably envisaged by King and his co-workers, since they report having found dust particles in the vessels of the liver, kidney and spleen.

Summing up the many years of research of Gardner and his co-workers on the effects on animals' lungs of asbestos particles administered by