upon an embarrassingly obvious structural characteristic of asbestos, it is supported by uncontested documented experimental findings as well as by data from some promising preliminary experiments. The latter, however, require confirmation.

Theory and Supportive Data

The new theory is simply this: the locus of pathogenicity of asbestos dust resides in the polyfilamentous structure of its fibrous particles. Various data support this theory.

Man-made mineral fibers, in contrast to polyfilamentous asbestos fibers, are monofilamentous. The dust of those monofilamentous fibers (fibrous glass,[1] ceramic aluminum silicate,[2] and silicon carbide whiskers[3]) that have been studied have proved to be nonfibrogenic when inhaled or injected intratracheally.

Synthetic chrysotile is nonfibrogenic when it is injected intratracheally. It is monofilamentous. It differs from natural chrysotile in that its tubular crystals are discrete and not in close parallel apposition to each other, as is the case with the natural product.

When a grinding force is applied to asbestos fibers, they tend to split longitudinally rather than to fracture transversely. Consequently, asbestos that has been ground to a fiber length of less than 5μ will consist largely of fibers that are monofilamentous. Those bundles of fibers still remaining will be greatly reduced in thickness and will approach the monofilamentous state. It is the fact that most of the fibers in such finely ground asbestos are either monofilamentous or nearly so, that offers a logical explanation of its nonfibrogenicity.

When asbestos is heated to 1,000 C, its pathogenicity is either lost or severely reduced (P. Gross, MD and R.A. Harley, Jr., MD, unpublished data). At this temperature, the fibrils composing the bundles tend to become fused into unitary structures, ie, the fibers tend to become monofilamentous (Fig 1). Although chemical changes do occur as a result of the heat, such changes have no bearing on the resulting loss of pathogenicity since it has been shown (with synthetic chrysotile) that the chemical composition of asbestos is not responsible for its pathogenicity.

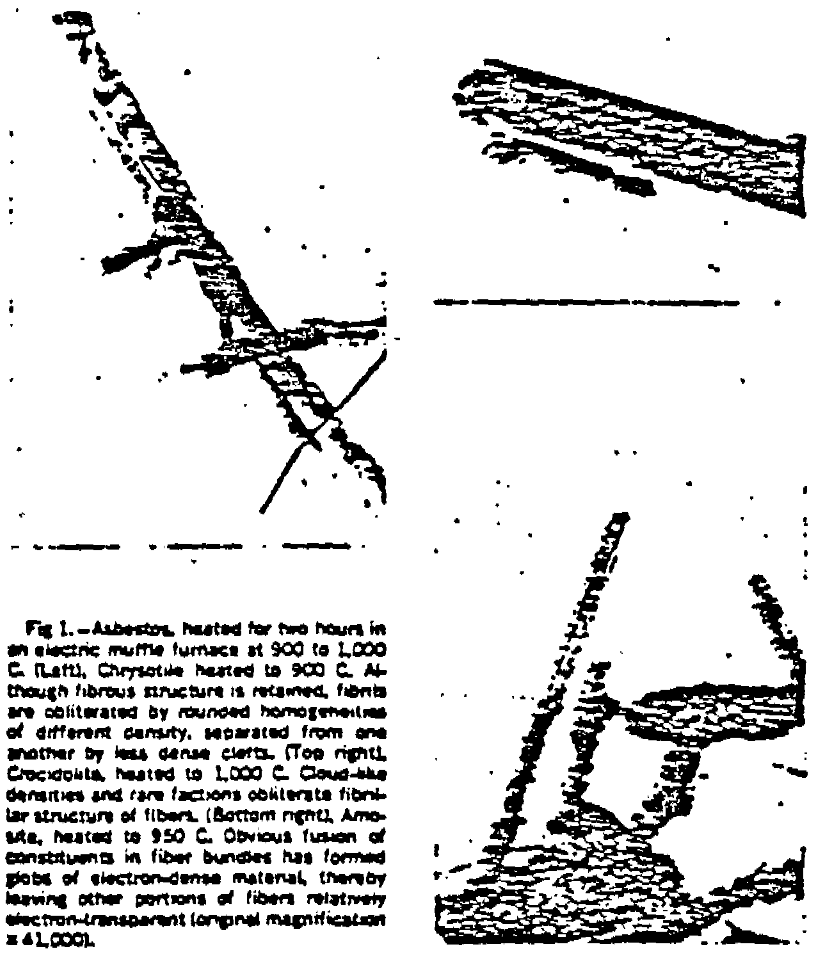
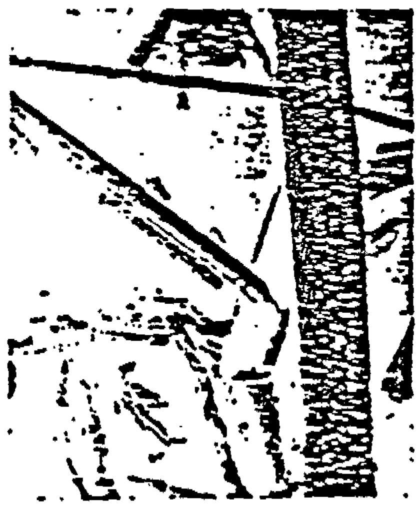


Fig 1.—Asbestos, heated for two hours in an electric muffle furnace at 900 to 1,000 C. (Left), Chrysotile heated to 900 C. Although fibrous structure is retained, fibrils are obliterated by rounded homogeneities of different density, separated from one another by less dense clefts. (Top right), Crocidolite, heated to 1,000 C. Cloud-like densities and rare factions obliterate fibrillar structure of fibers. (Bottom right), Amosite, heated to 950 C. Obvious fusion of constituents in fiber bundles has formed globs of electron-dense material, thereby leaving other portions of fibers relatively electron-transparent (original magnification x 41,000).

By cementing the fibrils of asbestos fiber bundles together chemically, it is possible to convert the polyfilamentous structures into monofilamentous ones. This has been successfully accomplished with various silicates such as those of lead, iron, and magnesium. The use of other water-insoluble cementing substances, such as those of magnesium and calcium pyrophosphate, calcium hypophosphate, and calcium metaphosphate, is contemplated.

The method has been to suspend finely ground asbestos in a dilute solution (1% to 5%) of an appropriate soluble metal salt (lead nitrate, iron chloride or magnesium chloride). After washing the asbestos free of all excess salt, it is suspended in a 1% solution of sodium silicate. The latter

Fig 2.—Chrysotile fibers cemented with lead silicate. Presence of lead silicate indicated by electron-dense stippling within lumen of the fibrils and elsewhere (original magnification x 143,000).



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