represent a general reaction to filamentous particles and are *not* a specific reaction to asbestos fibers. To what extent our findings may have application to the ferruginous bodies that are being found with increasing prevalence in human lungs [1-4] remains to be determined. The applicability depends, of course, upon the identity of the central fiber in the ferruginous bodies. The method of isolation and concentration of uncoated fibers and ferruginous bodies from human lungs which is described in this paper may help in the study of the nature, identity, and prevalence of inhaled filamentous particles and associated pulmonary ferruginous bodies.

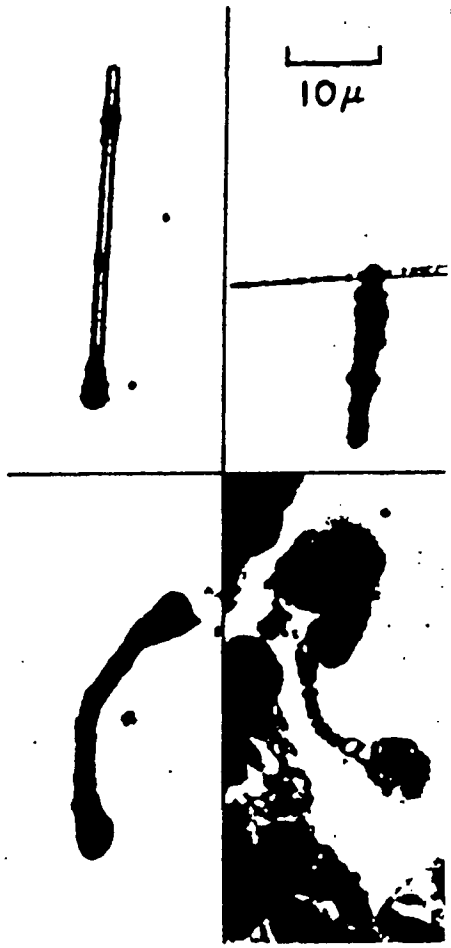
It is highly probable that the composition of the ferruginous body is altered by the method used for isolating it, since it is not likely that with the destruction of proteins leading to the liquefaction of all tissue elements in the lung, the protein in the ferruginous bodies should be spared. In all likelihood, the protein constituent of the bodies is destroyed, and only the iron and other inorganic components remain to retain and maintain the form of the bodies. The fragility of this chemically altered ferruginous coating is indicated by the ease with which it is removed when subjected to ultrasonic vibrations, thereby rendering the central fiber naked. This may further facilitate the identification of these fibers.

There appears an increasing tendency to consider all filamentous dusts in the same category as asbestos with regard to their pathogenic potential. This reasoning is probably based on a mechanistic concept of the pathogenicity of fibrous dust in general, and asbestos dust in particular. Such a mechanistic concept holds that the pulmonary fibrosis in asbestosis is the tissue response to mechanical trauma produced when pulmonary cells are perforated or impaled by the fine points of the asbestos fibers.

It need only be recalled that a similar mechanistic concept of the pathogenicity of crystalline silica was abandoned many years ago because of overwhelming evidence against its validity. Just as the proven biologic "inertness" of diamond dust was the coup de grace for the mechanistic pathogenetic concept of silicosis,[9] so should the proven biologic "inertness" of filamentous aluminum silicate [10] have discredited the mechanistic pathogenetic concept of asbestosis.

Extremely thin flakes of glass may also be considered to have sharp cutting edges; yet, glass has also been found biologically

Fig 4.—Ferruginous bodies formed in response to other filamentous dusts and stained for iron (Perls' test). The central fiber (top, left and right) is silicon carbide; and (bottom left) tremolite (talc) (× 1,110).



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