supplied as a felt, this was finely ground before injection.

2. Aluminum silicate, an uncoated ceramic fiber with a median diameter of 2μ. Fifty percent of the fibers were under 75μ in length and many filaments were shorter than 15μ. No free silica was detected in the fibers.

3. Silicon carbide whiskers, 99.5% silicon carbide. Fiber diameter ranged from 0.5μ to 3μ, and fiber length ranged from 100μ to 750μ.

4. Elastin, a commercial sample consisting of relatively short thick fibers with diameters of 5μ to 15μ and lengths from 50μ to 200μ.

Observations

When the animals were examined six weeks after injection, it was found that all test materials had caused the formation of large granulomas. These were fairly evenly distributed over both visceral and parietal pleura and the diaphragm. The histological patterns of these granulomas varied, however, with the different materials. Granulomas produced in response to glass or aluminum silicate were very cellular and consisted mainly of giant cells mixed with free macrophages and some fibroblasts. There was some deposition of reticulin fibers in these lesions from about two weeks after injection, but collagen was limited to a capsule around each granuloma. In the case of elastin, however, although the foreign material was quickly encased in a thin collagenous capsule, the cellular response around the elastin fibers was poor. In some areas, macrophages and fibroblasts infiltrated between the individual elastin fibers, but other areas remained quite acellular.

Perl's stain revealed that with both glass fiber and aluminum silicate, some of the fibers were coated with iron-containing material as little as two weeks after injection. By six weeks many ferruginous bodies, some of them beaded, were present in the granuloma, and they appeared in all respects similar to asbestos bodies produced in guinea pigs by the intrapleural injection of chrysotile asbestos (Fig 1 to 3). In the lesions produced by elastin, Perl's staining appeared negative after two weeks, but by six weeks some of the fibers were beginning to stain blue. In this case, however, the elastin fibers were much larger than asbestos bodies and could not be confused with them.

Electron miscroscope examination revealed that the cellular response to fine glass fiber and aluminum silicate was exactly similar to that induced by asbestos dust. Although the glass fiber used had an average diameter of 0.05μ to 0.99μ, much of the dust was thinner than this and some of it was as small as 250 A in diameter. These particles were, therefore, almost as small as crystals of chrysotile asbestos. The aluminum silicate dust was much coarser and very little of it was below 0.5μ in diameter.

All sizes of dust were taken up into macrophage or giant cell phagosomes, and these quickly contracted either to surround the dust with a closely apposed membrane or to form dust containing secondary lysosomes, as previously reported for asbestos dust by one of us (J.M.G.D., 1967). As with asbestos dust, it was found that the coating of dust to form ferruginous bodies was the exception rather than the rule, and it would appear that well below 1% of dust was ever involved in this process. Ferruginous bodies were always intracellular, usually in giant cells, and the deposition of their coating appeared in all respects similar to the formation of asbestos bodies.

The body coating consisted of dense granules approximately 60 A in diameter which probably represent ferritin (Fig 4 to 6). Most frequently the granules were deposited in a single dense layer up to 5μ thick (Fig 7), but occasionally they were arranged in layers of uneven thickness and density. In many cases there was a distinct membrane between the ferruginous body and the surrounding cell cytoplasm (Fig 6), but this was not always present (Fig 4). It can be seen in Fig 4 that the good preservation of many cytoplasmic membrane structures in close proximity to the ferruginous body precludes the possibility that a surrounding

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