OCT 11 2005 15:41 FR CISTI ICIST 613 952 9303 TO 12145201181 P. 04/16

The possibility that what appear to be "asbestos bodies" may have other than an asbestos core has been accepted almost as long as asbestos bodies have been known,[11] especially when exposure to asbestos is not documented. When other exposures are identified, appropriate designations are useful ("talc bodies," "graphite bodies," "fibrous glass bodies," etc). When exposure is not documented, one can consider implicit the bodies' nonspecificity[12,13] or even use a descriptive term which is noncommittal, as "ferruginous body."[14] In such circumstances, the uncertainty concerning the nature of the fibrous core of random "asbestos bodies" has hampered evaluation of the significance of their demonstration in the lungs of individuals in the general population. Do they or do they not reflect nonoccupational community asbestos exposure?

Investigations have been pursued to obtain further information on the problem. These investigations have taken two directions: first, the study of asbestos bodies by more refined analytical techniques, as electron microprobe analysis[15,16] or electron diffraction[17]; second, the appreciation that the unique morphology of chrysotile asbestos allows its specific identification by high-magnification electron microscopy. Both lines of study have centered on chrysotile, since more than 90% of asbestos used in the United States is of this variety, and both have sought to answer the question: is chrysotile asbestos to be found in the lungs of individuals not known to be occupationally exposed to asbestos, and, if it is, how frequently?

It was recently reported that analyses were made of 28 asbestos bodies micromanipulated from the lungs of 28 individuals in the general population of Pittsburgh.[17] It was stated that chrysotile was not found in any instance, and it was inferred that asbestos contamination of urban air was unlikely to be responsible for the formation of the asbestos bodies. Unfortunately, since few details were given, it is difficult to fully evaluate the report.

We have investigated the problem in another way and come to another conclusion; chrysotile was specifically identified in lung tissue in 24 of 28 consecutive cases examined at autopsy in New York city. This suggests that it is commonly present in the lungs of urban dwellers at this time.

Mineral Fibers: Their Occurrence in Lung, Recovery, and Species.—It has been frequently noted that there is lack of correspondence between the ease of recovery of chrysotile asbestos from the lungs of experimental animals and the difficulty experienced in its recovery from the lungs of humans occupationally exposed to this mineral. This observation may be explained by a number of interrelated factors.

The first factor is the amounts of asbestos involved. In a series of experiments by W. E. Smith, MD, and associates (unpublished data), 25 mg of chrysotile asbestos were injected intratracheally into golden Syrian hamsters. Six hundred and fifty-one days later, some of the animals were killed and lung tissue forwarded to our laboratory for mineralogical analysis. The tissue was ashed in a low-temperature ashing device, and the unashed residue was water dispersed and examined by means of polarized light microscopy and electron microscopy. Everywhere within the partially digested tissue debris, chrysotile fibers were observed. The optical properties remained remarkably similar to the natural material before injection; asbestos bodies tended to form about the thinner fiber bundles. By electron microscopy (Fig 1, *A*), a large number of thick fibers were found that were not broken open into individual fibrils. Several of the larger fibers demonstrated incipient coatings. Electron diffraction patterns obtained on the thicker fibers (Fig 1, *B*) indicated that the chrysotile had apparently not altered structurally. High-magnification examination of the fibers with the electron microscope demonstrated the ultimate structure in the form of the internal capillary (Fig 1, *C*).

The recovery of chrysotile from experimental animals is technically simple. The 25-mg dose injected into the lungs of a hamster of a young or a modest age (50 to 100-gm range of animal weight) is massive. Considering the weight of a hamster and its ratio of lung weight, the amount of chrysotile injected would equal several percent of its lung weight. (This would correspond to huge amounts of chrysotile asbestos in the lungs of humans, in the order of 4 to 5 gm per human lung.) This massive dose, con-

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