malignant pleural or peritoneal tumors. We are left with a strong body of evidence that individuals with light, moderate, and heavy exposures to crocidolite, amosite, or mixtures of amosite and chrysotile show tumors of a type that is much less common in the general population.

Studies in animals in 1962 by Wagner[31] in Wales and in 1965 by Smith[32] and others in New Jersey have led to production of mesothelial tumors in hamsters in which various types of asbestos were introduced intropleurally. The difficulty in preventing cross contamination in laboratories has complicated some of these animal studies, so that differences in response to different types of asbestos and co-factors remain unclear. Nevertheless, the carcinogenic potential of asbestos minerals appears to have been confirmed, although the role of co-factors has not been settled.

Ferruginous Bodies

The final question is, is it true that from a fourth to a half of the general population harbors asbestos fibers in its lungs? The basis for such a statement is the fact that a number of studies of routine consecutive autopsy specimens in recent years have demonstrated "asbestos bodies," or as Gough[22] would prefer to call them, "mineral-fibre-bodies," or Gross, "ferruginous bodies." As far back as 1928, Stewart[21] had shown that smears of the cut surface of the fresh lung at autopsy would reveal large numbers of asbestos bodies in the asbestotic. If anyone tried this in a supposedly unexposed population before studies done in South Africa in the 1960's, it was not published. In 1963, Thomson et al[7] showed 26.4% in 500 consecutive autopsies in Cape Town and later, in 1966,[8] 27.2% in 400 autopsies in Miami. These and other studies are summarized in Table 2. The facts that have emerged from these are as follows:

1. Morphologically and in staining properties, these meet all criteria for asbestos bodies.

2. In no studies so far published, have there been supporting data to indicate whether all, most, or some of the bodies were asbestos.

3. In most series, about 50% to 60% of

Table 2.—*Ferruginous Bodies in Human Lungs in Autopsy Series**

Place	Year	[illegible]
Cape Town[7]	1963	[illegible]
Miami[8]	1966	[illegible]
Pittsburgh[22]	1965	[illegible]
Johannesburg[28]	1966	[illegible]
Finland[24]	1966	[illegible]
Montreal[5]	1966	[illegible]
San Francisco†	1966	[illegible]

* Based on lung smears except [illegible] where thick sections were examined.

† W. C. Cooper and I. R. Tabershaw, unpublished data, 1966.

the positive reports were on the basis of relatively few asbestos bodies per case.

4. In those with many bodies there were often but not invariably occupational or residential clues as to a source of asbestos; they were most commonly in males.

5. In none of the series, all small were correlations were attempted with data was there any apparent association with malignancies or other specific causes of death.

6. There is ample evidence in the literature that so-called asbestos bodies can be found in workers with exposures other than asbestos, eg, graphite workers, soft coal miners, diatomaceous earth workers, etc.

7. Ferruginous bodies can be produced experimentally in guinea pigs with other dusts. Presumably, they represent a nonspecific response to any relatively insoluble fibrous material in the lungs.

Speaking of this, Cooke[4] said, "... there is no reason why any fine stranded mineral should not have colloidal material deposited around it and become modified into a curious body. But as no other mineral dust is fibrous, this occurrence must be so rare as to be negligible from a diagnostic point of view."

The question now, is Cooke's statement 40 years ago true today? Or, are we adding fibers of many kinds to our environment giving a confusing picture of our lung samples?

If, as is probable, many of the bodies that are being seen are really asbestos bodies, there is need to determine their significance. It is unwarranted to draw [illegible] from what we now know that [illegible] ly present an exchange of [illegible]

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