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July 24, 1961

Dr. Kenneth W. Smith
Medical Director
Johns-Manville Corporation
22 E. 40th Street
New York 16, N.Y.

Dear Ken:

I am enclosing my report on Edward Bullock with a carbon copy. I am always glad to see material of this type. Thanks for the opportunity of studying this slide.

I hope you have had an enjoyable trip abroad and have come back better prepared to handle your various headaches (maux à la tête?), than when you left.

I am enclosing a letter from Dr. Spain which is self-explanatory. I have in mind that a good many of your cases would lend themselves admirably for this purpose. What do you think?

With best regards.

Sincerely yours,

Paul Gross, M.D.
Research Pathologist

pg:la

P.S. I am returning your slide.

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July 24, 1961

MEMORANDUM TO: Dr. Kenneth W. Smith
Medical Director
Johns-Manville Corporation

FROM: Dr. Paul Gross
Research Pathologist
Industrial Hygiene Foundation

RE: Microscopic Findings in the Case of
Edward Bullock, Toronto

The following findings are based on a slide, numbered 2272/61, which was received from Johns-Manville Corporation, 22 East 40th Street, New York 16, New York.

Sections of lung (three sections on one slide) exhibit generalized, fairly uniform, cellular and fibrillar thickening of septal walls. This thickening has progressed to the formation of nodular masses which obliterate air spaces. The nodules are poorly circumscribed and contain a variable number of foreign body type giant cells or Langhans-type, or both. The nodules are composed of a fibrillar tissue suggestive of reticulin rather than collagen. The cells present include epitheloid type nuclei, lymphocytoid type and occasional plasma cells. Occasional macrophages are also

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noted. The latter may contain yellow to yellow-brown pigment granules. By confluence of nodular masses some fairly extensive consolidation is formed. No necrosis is present anywhere. The giant cells contain vacuoles of irregular character. Inclusions of other types are not found.

The septal wall thickening is caused by a loose, fibrillar-type of tissue suggestive of reticulin, and proliferated alveolar cells as well as scattered lymphocytoid-type nuclei.

There is subserosal thickening of the pleura by an edematous fibrillar tissue scantily infiltrated by lymphocytes, monocytes, and occasional eosinophils.

Bronchi show no significant disease. Blood vessels are not thickened.

Isolated asbestos bodies are found singly, in pairs, and in small groups. Naked asbestos fibers can not be found. The asbestos bodies are found incorporated in fibrillar connective tissue, independent of giant cells or the granulomatous nodules.

In addition, separate from the asbestos bodies, scanty intracellular dark olive pigment deposits are noted.

<u>Diagnosis:</u>

1. Sarcoidosis of lung with minimal asbestos bodies but no evidence of asbestosis.

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**Comment:**

All the changes present in these sections, except asbestos bodies, can be accounted for on the basis of a granulomatous inflammation suggestive of sarcoidosis. The only other diagnosis which deserves serious consideration is berylliosis. I confess that I cannot distinguish the pulmonary lesion of the latter disease from pulmonary sarcoidosis. If berylliosis can be ruled out clinically, only sarcoidosis need be considered.

This is not asbestosis because:

1. The granulomatous character of the inflammation
2. The fairly extensive granulomatous consolidation
3. The absence of emphysema
4. The delicate, fibrillar character of subpleural fibrosis
5. Absence of vascular sclerosis
6. Paucity of asbestos bodies.

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