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Mount Sinai School of Medicine • The Mount Sinai Hospital

RECEIVED

Irving J. Selikoff, M.D.  
The Mount Sinai Medical Center  
19 East 98th Street, [Box 1059]  
New York, N.Y. 10029

October 12, 1987

(212) 244-7809, 7811

Mr. Robert E. Sweeney  
Suite 950  
55 Public Square  
Cleveland, OH 44113

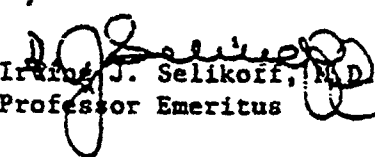
Dear Mr. Sweeney:

In the continuing academic discussions concerning short-fiber vs. long fiber asbestos toxicity, I'm not sure that we mentioned the nice original work by Dr. Paul Gross and his colleagues who studied the questions. This was likely of some importance, in view of Dr. Gross's special role at the Industrial Hygiene Foundation Inc. and the value placed on the work of his group by the asbestos industry.

They gave a single intratracheal injection of short fiber chrysotile experimentally and observed resulting pulmonary fibrosis. (Granulomatous pneumonitis caused by short-fiber asbestos dust (chrysotile), American Journal of Pathology 31:579, 1955) "Our findings are confirmatory of the pathogenicity of short-fiber asbestos dust reported E. J. King, Clegg and Rae in rabbits."

With all best wishes.

Sincerely yours,

  
Irving J. Selikoff, M.D.  
Professor Emeritus

IJS:cl

cc: Mr. Ronald L. Motley ✓

PLAINTIFF'S EXHIBIT

SA-485

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VOLUME XXXI

1955

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whether a left-to-right shunt or pulmonary stenosis was present. The incidence was similar in both groups: increased with advancing age, and in both abnormal groups was 100 per cent in age groups over 10 years.

(2) Since it is reasonable to assume that the average of the pulmonary arterial pressures in patients with left-to-right shunt was higher than in the patients with pulmonary stenosis, it is apparent that hypertension was not a primary factor in the production of pulmonary arteriosclerosis in these cases. However, the severity of arteriosclerotic lesions was significantly greater in cases with left-to-right shunt than in those with pulmonary stenosis, suggesting that increased pressure and blood flow in these vessels may be etiologic factors of accessory importance.

(3) Fibrous intimal lesions were associated with pulmonary arterial thrombi in 70 per cent of the cases regardless of the type of anomaly present, suggesting that thrombi may be a primary factor in the production of these arteriosclerotic lesions.

(4) Pulmonary arterial thrombi were of similar frequency in cases with left-to-right shunt (25 per cent) and in cases with pulmonary stenosis (35 per cent).

(5) Because potential sources for emboli were found in 10 cases, an embolic origin of the arterial thrombi can be postulated.

(6) The transformation of a fresh thrombus to an arteriosclerotic lesion could occur through organization of the thrombus and subsequent contraction. Lesions which irresistibly suggest the stages in this process could be easily found.

EMBOLIZATION OF CEREBRAL TISSUE TO LUNGS FOLLOWING SEVERE HEAD INJURY.  
James E. McMillan (by invitation), Ohio State University, Columbus, Ohio.

Three cases are reported in which microscopic emboli of cerebral tissue were found in pulmonary arterioles. All 3 patients died following severe head injury with skull fractures, gross lacerations of the brain, and traumatic intracerebral hemorrhage. Lung sections showed numerous emboli of brain tissue distending the pulmonary arterioles, and in one case the embolization was accompanied by infarcts of the lung. In all cases glial cells were well preserved, and neurons and vascular structures could be identified in several of the emboli. A total of 100 complete necroses were performed during a 17-year period on patients dying with severe head injuries giving an incidence of 1.5 per cent for this lesion. Although the literature stresses the great rarity of its occurrence, the present report would indicate that traumatic embolization of cerebral tissue is not too uncommon. Careful histologic examination of numerous sections of the lungs is necessary, and two of the present cases were discovered only after careful restudy of the necropsy material. The validity of this report is further substantiated by a series of animal experiments.

GRANULOMATOUS PNEUMONITIS CAUSED BY SHORT-FIBERED ASBESTOS DUST (CHRYSOTILE). Paul Gross and (by invitation) Marian L. Westrich and James M. McMerney. Industrial Hygiene Foundation, Mellon Institute, Pittsburgh, Pa.

By a single intratracheal injection in rats of slightly less than 10 mg. of chrysotile fibers substantially shorter than 5  $\mu$ , but also including a few longer fibers, we have produced nodular granulomatous lesions leading to pulmonary fibrosis. Our findings are confirmatory of the pathogenicity of short-fibered asbestos dust reported by E. J. King, Clegg, and Rae in rabbits.

EXPERIMENTAL ASPIRATION PNEUMONIA: INFLAMMATORY AND REPARATIVE CHANGES PRODUCED BY INTRATRACHEAL INJECTIONS OF AUTOLOGOUS GASTRIC JUICE AND HYDROCHLORIC ACID. T. J. Moran, Presbyterian and Woman's Hospitals, Pittsburgh, Pa.

The effects of intratracheal injection of homologous gastric juice, hydrochloric acid, pepsin, and steapsin in rabbits have been studied. The changes produced in-