

Jerome Gelman, Esq.
for GROSS, Matthew
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PLAINTIFF'S
EXHIBIT

K-281

the past four years had been exertional dyspnea. This had become considerably worse for the past one or two years and was now affecting him in moderate activities. Reference to my notes during my examination in 1950 indicate that no significant exertional dyspnea was then present. This shortness of breath was without apparent cause insofar as he was concerned, was moderately disabling, was not present at rest and was progressive.

Review of his family history, system review, etc. revealed nothing of significance apart from a back injury many years ago while at Union Asbestos with occasional recurrent pain in the back since. There were also moderately frequent headaches, moderate alcoholic intake and moderate smoking.

Mr. Grossi was carefully reviewed by myself and my associates, Dr. Robt. H. Joelson and Dr. David Roth. Complete physical examination failed to reveal anything of significance apart from the lungs. Here, there were coarsened breath sounds and moderate rales at both bases. Weight, without clothes, was 148-3/4 lbs. and height was 70". The remainder of the gross physical examination was not significant except for rather marked clubbing of the fingers. Blood studies done at the Barnert Memorial Hospital in Paterson revealed a hemoglobin of 15 gms., red blood cells 5, 850,000, white blood count 9,550 with 51% neutrophil polys and 48% lymphocytes. Hematocrit was 44%. Examination of his sputum showed no acid fast bacteria. Electrocardiogram done on Nov. 27, 1954 was not remarkable and serological tests for syphilis were negative. Decholin circulation time was 18.5 seconds.

X-ray of the chest is distinctly abnormal. Films taken on Nov. 25, 1954 shows a very fine nodular infiltration predominantly in the lower third of the lung field, diminution of the vascular markings in the middle and lower thirds of the lung fields, irregularities along the medial halves of the diaphragmatic leaves, as well as moderate reticulation of the lung fields in the outer thirds. There was some prominence of the vascular marking of the hilae. The heart contour is normal.

When this film is compared to one in my possession, taken on December 21, 1950, there has been considerable progression of the disease. At that time, there was little significant change in the chest film, at least of a specific nature. The changes in the x-ray at the present time, however, are quite specific for asbestosis, being the definitive roentgenological findings in this disease.

Appended to this report is a report on pulmonary function studies performed on Dec. 17, 1954 by Dr. Mortimer E. Seder, M.D. and Richard A. Seder, M.D. at the Cardio-pulmonary Research Laboratory of the Mt. Sinai Hospital. Very complete detailed studies were done and the data obtained is, as noted, appended hereto. Although it is well known that minor or occasionally even moderate respiratory disease may fail to be reflected in

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pulmonary function studies with techniques presently available, definite abnormality was found in the case of Mr. Grossi. Here, although ventilation is apparently still adequate and alterations thereof not easily yet detected, gas exchange through the lungs is found to be defective. There was a definite diminution in the oxygen saturation in the arterial blood on exertion. The conclusion of Dr. Mortimer E. Sader and Dr. Richard A. Sacer is quoted. "Such a finding is distinctly abnormal and reflects impaired gas exchange under the stress of exercise." Mr. Grossi is suffering from pulmonary asbestosis. This is moderately severe. I would not be particularly concerned with his cough or with the drawing pain and tightness felt across the anterior chest. Of much greater import is the shortness of breath upon exertion that this man has for the past several years and which has become much worse in the past year or two. At rest, he has no respiratory difficulties. This is reflected in the normal pulmonary function studies obtained at rest. However, with exertion he was short of breath. This again is reflected in the abnormal pulmonary function studies obtained during exertion and found to be due to a defect in his ability to affect gas exchange (oxygen) through his lungs. This parallels very well with the x-ray findings of considerable disseminated pulmonary damage. At the present time I would estimate that Mr. Grossi is disabled to the extent of 75% of his total disability. I do not anticipate, if he stays away from asbestos dust, that this disability will increase. We may state that it is now permanent. However, I am not completely sanguine about his prognosis. There is now distinctly recognized that the danger of carcinoma of the lungs is considerably increased in people with pulmonary asbestosis, as is the danger of the development of pulmonary tuberculosis. Thus, in addition to his significant pulmonary disability peculiar to the asbestosis, there is the considerably increased danger of cancer of the lung and tuberculosis in this man. For these possibilities, however, no statement of disability can be made at the present time.

Very truly yours,

Irving J. Selikoff, M.D.

JS:sl

cc/ Dr. Fall

Drs. M. E. and R. A. Sader

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Irving J. Selikoff, M.D.
David Roth, M.D.
Robert H. Joelson, M.D.

Report on Matthew Gross!
Studied: Dec. 17, 1954
in Age 42
B.S.A. 1.86 H2

Lung volumes, Maximal Breathing Capacity and Index of Intrapulmonary Mixing Lung volume.

	Predicted	Lying	Standing
Expiratory reserve volume		1400 cc	2380 cc
Inspiratory capacity		3570	
Vital capacity (calc.)	4480cc	4970	
" " (obs.)	4480	5050	5220
Residual volume	1450	2060	
Total capacity	5940	7110	
<u>Residual Volumes</u>			
Total capacity	24.1%	29%	
Maximum breathing capacity	120 L/min	140 L/min	146 L/min
Alveolar Nitrogen	2.5%	1.11	
After 7 minutes oxygen		1.29	

Breathing reserve after 1 minute standard exercise

	<u>RECOVERY</u>					
	1 min. Ex.	1 min.	2 min.	3 min.	4 min.	5 min.
Minute Ventilation	25.2 L/min	20.1	16.2	12.7	8.10	6.46
Breathing reserve Ratio	83%	85	88	91	94	95

Dyspnea time following exercise - less than one minute.

6. Gas Exchange: Arterial oxygen content, capacity and saturation normal at rest. On exercise the oxygen saturation in the arterial blood to 85%. This is abnormal. There is no evidence of retention of carbon dioxide. The respiratory dead space is slightly increased.

Comment: The only significant abnormal finding is the arterial O_2 saturation on exercise. This suggests impaired ventilation-perfusion relationships during exercise. Such a finding is distinctly abnormal and reflects impaired gas exchange under the stress of exercise.

(signed) Mortimer E. Bader, M.D.
Richard A. Bader, M.D.