



Mr. Jerome Gelman
45 Church St.
Paterson, N.J.

re: PETTERSON- John W.

Dear Mr. Gelman:

Mr. John W. Peterson of 118 Jefferson Street in Haskell was examined by me on November 24, 1954, December 3, 1954 and December 17, 1954. In addition he underwent a number of examinations at the Barnert Memorial Hospital Laboratory and at the Cardio-Respiratory Research Laboratory of The Mt. Sinai Hospital. Reference will be made to the findings of these laboratories in subsequent paragraphs.

Mr. Peterson is a 53 year old fork-truck operator whose occupational history is not remarkable until his present employment. In adolescence, he worked as a farmhand and then, in sequence, he worked as a lumber-worker, horse-driver, a riveter, a production-worker at Dufont Powder Works, pipe-stem classifier and as a truck driver for 18 years. On December 8, 1942 he went to work for Union Asbestos, at first handling bags of fiber for several years and then as a tow-motor driver. In this latter job, the exhaust of the motor, hitting onto the ground, would stir up the dust thereon and he was frequently, therefore, in a dusty atmosphere. He usually did not wear a mask, although on entering a particularly dusty area, he sometimes did utilize this protective device.

At the time of my examination he had ceased work at Union Asbestos on November 15, 1954. He was asymptomatic insofar as his lungs were concerned. There was no exertional dyspnea. There was little cough except with cigarettes and no chest pain apart from presterinal gas. No sputum was raised and he appeared a generally healthy man.

Apart from lungs, physical examination was not remarkable. There was some "indigestion" relieved by belching, especially after fried foods. An occasional boil on the skin almost completed his inventory of complaints. Again, apart from the lungs, he had no significant abnormal physical findings apart from a slightly enlarged prostate gland, which enlargement was benign. Also, blood pressure was 170/110, evidence of a mild, benign, essential hypertension. Urine was negative, as were aerological studies. Electrocardiogram was normal as was circulation time, done with dachalin, of 18 seconds. Hematological examination at the Barnert

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Bernart Hospital Laboratory did reveal some polycythemia, but not remarkably so. A smear for acid fast bacteria was negative.

However, these negative findings do not tell the whole story. X-ray of the chest showed a mild infiltration in the lung fields with flattening of the vascular markings in the lower lung field. There was some infiltration especially in the right pericardiac area of the diaphragm. This roentgenographic change was not excessive, but it was present. Also, a search of the sputum revealed the presence of asbestos bodies. This was done at the Bernart Memorial Hospital Laboratory and reported on December 8, 1954. A very careful pulmonary function studies, performed by Dr. Mortimer Sader and Dr. Richard Sader at the Cardio-respiratory Research Laboratory of The Mt. Sinai Hospital in New York on January 5, 1955 did reveal evidence of pulmonary damage. The report from this Laboratory is appended hereto. It will be noticed that there is significant reduction in the vital capacity and in the total capacity. This evidence of retraction in the lung volumes, in the absence of the airway obstruction, suggest parenchymal damage, and the reduced oxygen saturation of the arterial blood at rest, similarly indicates impairment of the ventilation-perfusion relationship. The conclusion of the research laboratory should be noted, "there is definite evidence of pulmonary involvement with impaired lung function".

This man is not as badly damaged by pulmonary asbestosis as some other cases I have recently seen. However, he does suffer from pulmonary asbestosis, of a moderate degree. This is evidenced not only by the x-ray findings but also by the presence in the sputum of asbestos bodies and by the findings, on careful study, of impaired lung function. The pattern of impairment is similar to that found in other cases of pulmonary asbestosis.

I would estimate Mr. Peterson to be disabled to the extent of 25% of total disability as a result of pulmonary asbestosis which is closely related to his exposure in his occupation.

Very truly yours,

Irving J. Saltzoff, M.D.

IJS:ed
encs.

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Report on John W. Peterson
 Age 50
 D.S.A. 1.86
 Studied: January 5, 1955

LONG FUNCTION STUDIES

	Predicted	Lying	Standing
Expiratory Reserve Volume		305 cc	500 cc
Inspiratory Capacity		1940 cc	2315 cc
Vital Capacity (calc)	4190 cc	2245 cc	2175 cc
(obs)	4190 cc	2175 cc	2945 cc
Residual Volume	1350 cc	1355 cc	
Total capacity	5540 cc	3500 cc	
<u>Residual Volume</u>			
Total capacity	24.4%	40.9%	
Max. Breathing capacity	105 L/min	117 L/min	140 L/min
Residual alveolar nitrogen after 7 min. breathing O ₂	2.5%	2.4%	

Breathing Reserve following 1 minute standard exercise.

		<u>RECOVERY</u>				
Exercise		<u>1 min</u>	<u>2 min</u>	<u>3 min</u>	<u>4 min</u>	<u>5 min</u>
Minute						
Ventilation (L/min)	9.46	24.3	15.8	12.2	11.9	11.2
Breathing Reserve	130.5	82.7	91.2	104.8	105.1	105.2
ratio	72.5%	70.6%	77.8%	89.5%	89.6%	90.3%

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State	<u>Exercise</u>		
	<u>Resting</u>	<u>Resting</u>	Room Air
Inspired O ₂	21%	16%	
V _{O₂} (oxygen consumption cc/min)	261 cc	245 cc	
V _{O₂} /A ²	140	132	
V _{CO₂} (CO ₂ output cc/min)	211	196	
V _{CO₂} /A ²	114	105	
R.Q.	.81	.80	
VE (Minute ventilation L/min)	7.37 L/min	7.34 L/min	
VE/I ²	3.96 Lit/min	3.94 Lit/min	
Respiratory rate	14	14	
Tidal volume	526	524	
Expired % CO ₂	3.44%	3.20%	
Expired pCO ₂	24.7 mm Hg	23.0 mm Hg	

ARTERIAL BLOOD STUDIES

pH	7.39	7.39	7.36
O ₂ content	21.3 vols%	19.9	22.0
O ₂ capacity	23.1 "	22.5 vols%	23.0 vol%
O ₂ saturation	93%	89%	97%
CO ₂ content (whole blood)	44.1 vols%	42.3 vols%	42.6 vol%
CO ₂ content (serum)	55.2 "	53.0 "	53.0 "
pCO ₂ (mm Hg)	40 mm Hg	39 mm Hg	41 mm Hg
Respiratory dead space	136 cc	150 cc	
Dead space ventilation % of total volume	25.8%	28.6%	

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INTERPRETATION:

1. LUNG VOLUMES - there is a significant reduction in the vital capacity and total capacity. The residual volume is slightly increased, and when compared with the reduced total capacity, results in an increase in the ratio of residual volume to total capacity. There is no evidence of impairment in the intrapulmonary mixing of inspired gases.
2. THE MAXIMUM BREATHING CAPACITY - is normal and there are no significant findings to indicate airway obstruction.
3. SPIROMETRY - reveals no evidence of trapping.
4. THE INDEX OF INTRAPULMONARY MIXING - is normal indicating no significant impairment of the distribution of inspired gases, as measured by this test.
5. GAS EXCHANGE - There is a slight degree of arterial unsaturation at rest, but none on exercise. The arterial blood pO_2 and pCO_2 are within the normal range. Dead space ventilation is within normal limits.

COMMENT:

There is evidence of significant reduction in the lung volumes viz. vital capacity and total capacity, which in the absence of airway obstruction, suggests parenchymal involvement. The reduced oxygen saturation of the arterial blood at rest suggests impairment of the ventilation-perfusion relationship. Improvement of oxygen saturation on exercise might be due to better ventilation in areas which were perfused but hypoventilated at rest. There is definite evidence of pulmonary involvement, with impaired lung function.

/s/ Mortimer E. Bader, M.D.
Richard A. Bader, M.D.

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