

DIVISION OF PULMONARY AND
CRITICAL CARE MEDICINE

August 30, 1981

University of
Arkansas
for Medical
Sciences

Department of Industrial Relations
Division of Industrial Accidents
Information and Assistance Bureau
ATTENTION: Ms. Diane Bustillos
525 Golden Gate Avenue
San Francisco, CA 94102

REDACTED

RE: [REDACTED]

Dear Ms. Bustillos:

Slot 555
4301 W. Markham
Little Rock, Arkansas
72205

[REDACTED] was seen in our Chest Clinic on June 12, 1981 for evaluation of asbestosis as requested in your letter of March 26, 1981. [REDACTED] related that he was first exposed to asbestos beginning in 1948 when he began working as a journeyman logger. The following year, he became a mechanic and remained in that occupation for 1½ years during which time he had no asbestos exposure. From 1951 through 1956 he returned to his previous job as a logger, continuing the same kind of exposure he had had previously. He joined Dupont in 1956, and had heavy primary exposure to asbestos until 1967. About two years ago [REDACTED] began having exertional dyspnea, which has been slowly progressing. Currently he states that he is able to ascend one flight of stairs, shovel dirt about five minutes, or walk about two blocks before being limited by dyspnea. He described his dyspnea as a feeling that he can move air appropriately, but "cannot get enough oxygen". He stated that his dyspnea resolved after about half hour of rest. He denied cough, sputum production, hemoptysis, and wheezing. He smoked approximately two packages of cigarettes daily for 30 years, stopping two years ago when he was informed of the high risk of malignancy due to smoking and asbestos exposure together. He denied allergies. He had a past history of peptic ulcer disease, for which he had taken cimetidine, but he was taking no medications at the time that he was seen.

(501) 661-5525

Physical examination revealed a well-developed, well-nourished white man who appeared to be his stated age and was in no distress. Examination of the chest revealed no adventitious sounds, normal breath sounds, and no abnormalities on percussion. The liver spanned 10 centimeters to percussion, and there was mild right upper quadrant tenderness. The abdomen was otherwise normal. There was no clubbing of the fingers, cyanosis, nicotine staining of the fingers, or edema. There were no other abnormalities noted on physical examination.

PA and lateral chest x-rays revealed an extensive area of thickened pleura on the left, which had slightly increased in size in comparison with an x-ray made in 1979. There was a small approximately 1½ x ½ centimeter area of pleural thickening on the right, and calcification of the diaphragm was visible on the right side. Resting arterial blood gases were PaO₂ of 87 mmHg, PaCO₂ of 34 mmHg and pH of 7.41. Spirometry and lung volumes done by helium dilution are shown on the enclosed report. The lung volumes were normal; the FEV₁/FVC ratio was slightly decreased to 73%. The studies were interpreted as showing a borderline obstructive defect. An electrocardiogram demonstrates incomplete right bundle branch block without other abnormalities.

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DIVISION OF PULMONARY AND
CRITICAL CARE MEDICINE

August 30, 1981

RE: [REDACTED]

REDACTED

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An exercise test was performed on June 19, 1981 at Little Rock Veterans Administration Hospital by Dr. McKendree E. McNabb, a member of this Division. A copy of the report is enclosed. The pertinent findings were that [REDACTED] had normal cardiopulmonary function, including arterial oxygen saturation during exercise, and that he had a normal tolerance to exercise. During the exercise test he reached only 53% of the maximum breathing capacity predicted on the basis of the FEV₁. It is notable that during initial steady state testing at low work rates he had abnormally high ventilation, and that during subsequent testing at the same work rate he had normal ventilation. This suggests a psychogenic etiology for the initial hyperventilation.

A single breath carbon monoxide diffusing capacity was performed at Little Rock Veterans Administration Hospital on June 19, 1981. The results was 26 ml/min/mmHg, the predicted value being 30. This is considered to be a normal value.

Arterial blood gases drawn within two seconds of cessation of maximal exercise were as follows: PaO₂ 97 mmHg, PaCO₂ 34 mmHg, and pH 7.41. The change from the resting blood gases represents a normal response to exercise. On the basis of his chest x-ray findings and his history, it is clear beyond any reasonable doubt that [REDACTED] has asbestosis. The slight decrease in FEV₁/FVC ratio noted is more likely related to his long history of cigarette smoking than to his asbestosis exposure. In any case, that degree of obstructive spirometric defect would not be expected to result in disability or, indeed, in significant symptoms.

I believe that [REDACTED] experiences the symptoms he describes, and that his life is significantly altered by them. Thorough evaluation does not reveal a physiologic basis. I believe that they are largely psychogenic in origin, and are related to [REDACTED]'s realization that he has asbestosis which he finds extremely threatening. I think his symptoms are probably increased by his relative inactivities causing deconditioning, which, in turn, increases his symptoms. In summary, I think his symptoms are due to his asbestos exposure and the resulting asbestosis, although I think the mechanism of symptom production is psychologic, not physiologic.

I would recommend follow up examinations in the future, to primarily consist of chest x-ray's. Obviously, the reason for this is that mesothelioma cannot be completely excluded in any point in time, and the only practical way to detect its occurrence would be to note a change on serial chest x-rays. In addition, it provides a means for detection of bronchogenic carcinoma, for which [REDACTED] is at increased risk. The only treatment other than the follow up examinations I would recommend would be an effort to overcome the respiratory symptoms probably. I should think that the best way to approach this would be

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DIVISION OF PULMONARY AND
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August 30, 1981

RE: [REDACTED]

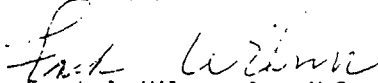
REDACTED

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through an exercise program combined with some incidental supportive therapy by a sympathetic physician. I have discussed this with [REDACTED]. He is skeptical that he can be helped by an exercise program, but I have urged him to undertake one. It seems very likely that his exposure to asbestos between 1957 and 1967 was most significant in causing his asbestosis, since it was much larger in duration and degree than his previous exposure.

If this does not adequately answer the questions of interest to you, please let me know.

Sincerely yours,



Frank J. Wilson, Jr., M.D.
Assistant Professor of Medicine

FJW/bas

Enclosures

cc: Hospital Chart
Chest Clinic Corr.

DUP 0947277

DU 038571

CLINICAL RECORD

CONSULTATION SHEET

REQUEST

PULMONARY FUNCTIONS

FROM: (Requesting word, unit, or address)

DATE OF REQUEST

EXERCISE PHYSIOLOGY

EXERCISE PHYSIOLOGY

INSTRUMENTATION:

Quinton electromechanical cycle ergometer	Avionics valve
Beckman Metabolic Measurement Cart	Beckman valve
Singer dry gas meter	Modified Otis-McKerron valve
Medaw mass spectrometer	Neoprene bags
Hewlett-Packard ear Oximeter	E.C.G. by H-P
Hewlett-Packard digital pneumotach	Mine Safety monoxolyzer

PROCEDURE:

Incremental work rates to maximum tolerance; increments were 100 Kilogram-meters per minute, work rate was incremental each minute.
Submaximum work rate, 6 minutes each

200	400	600	800
300	500	700	900

REDACTED

PATIENT COOPERATION:

Good

SUBJECTIVE OBSERVATION:

✓ legs weak chest pain shortness of breath dizzy
weak Other

COMMENTS: Initially submaximal constant workrate tests of 200 and 400 kilogrammeters per minute were performed. Observations made during the 5th and 6th minutes of these tests, with an established physiologic steady state, revealed excessive ventilation, excessive respiratory frequency and abnormally low respiratory exchange ratio. Cardiac frequency was normal. The low respiratory exchange ratio was consistent with excessive ventilation and partial depletion of body CO₂ stores prior to initiation of exercise.

Just before incrementing the workrate to determine maximum O₂ consumption the patient was again asked to pedal 4 minutes at 200 kg-m/min followed by a 5th minute at 300 kg-m/min and a 6th minute at 400 kg-m/min. Of interest is that ventilation and breathing frequency fell significantly compared to that seen during the initial submaximal testing at 200 kg-m/min. This suggests psychogenic rather than physiologic mediation of the excessive ventilation.

Ventilation and respiratory frequency were increased relative to oxygen consumption and tidal volume was abnormally reduced. The heart rate was normally matched to oxygen consumption. Arterial blood oxygen saturation as estimated by ear oximetry was 96% resting and 96 to 97% throughout exercise. This was a normal response. He achieved an oxygen consumption of 2000 ml per minute (the predicted value is 2,568 with a normal range of 1,768 to 3,368 ml per minute). He expended only 53% of his breathing capacity (35 X 3.99 = 136.5 liters per minute) and 84% of cardiac chronotropic capacity. An arterial blood gas (drawn within 2 seconds of cessation of maximal exercise revealed no metabolic acidemia, PaO₂ 97, P₅₀ 37.4, pH 7.41, bicarbonate 21.

SIGNATURE AND TITLE

MEM
MCKENDRICK F. MCNABB, M.D.

DATE

6/24/81

IDENTIFICATION NO.

ORGANIZATION

PULMONARY FUNCTION LABORATORY

PATIENT IDENTIFICATION (if typed or written entries give Name, last first, middle, grade, date, hospital or medical facility)

REGISTER NO.

WARD NO.

CONSULTATION SHEET
Standard Form 513
513-10a

DUP 0947278

The patient indicated that tiredness of his legs kept him from continuing the incremental exercise load. This is consistent with lack of specific training and conditioning for the task of pedaling a cycle against increasing resistance.

- Impression: 1) Normal tolerance of heart and lungs to exercise i.e. normal aerobic capacity .
2) While neurogenic stimuli from a diseased lung may cause excessive ventilation the tendency demonstrated here is more consistent with psychogenic hyperventilation.

McKendree E. McNabb
MCKENDREE E. MCNABB, M.D.
Medical Director

DUP 0947279

DU 038573

	VENTILATION RATE	BREATHING RATE	TIDAL VOLUME	ml.	ml./min	ml./min	REDACTED	
200	18.3	25.1	730	570	400	86		0.70
"	22.1	26.0	850	700	470	88		0.68
"	23.1	31.2	740	670	480	88		0.71
"	24.7	31.9	770	680	500	89		0.73
300	25.0	29.5	850	710	520	92		0.73
400	27.8	29.9	930	850	620	98		0.72
500	35.7	32.4	1100	1080	860	104		0.79
600	43.7	37.5	1170	1220	1030	112		0.84
700	50.6	41.6	1220	1320	1170	120		0.89
800	61.2	40.3	1520	1670	1540	131		0.95
900	64.3	40.0	1610	1690	1630	138		0.97
1000	72.4	45.3	1600	1870	1810	145		0.97
1100	72.1	35.4	2040	2000	1890	148		0.95

	PATIENT	PREDICTED
VENTILATION @ 0.75 L.O ₂ UPTAKE	18.7 ± 1.0	
VENTILATION @ 1.00 L.O ₂ UPTAKE	25.0 ± 1.0	
VENTILATION @ 1.50 L.O ₂ UPTAKE	36.6 ± 1.5	
TOTAL VOLUME @ VENTILATION 30 Vmin	2017 ± 152	
HEART RATE @ 0.75 L.O ₂ UPTAKE	95.0 ± 3.1	
HEART RATE @ 1.00 L.O ₂ UPTAKE	105.6 ± 3.1	
HEART RATE @ 1.50 L.O ₂ UPTAKE	126.9 ± 3.4	

DUP 0947280

1927 21 APR DATE 6/91

Ear Oximetry Data Sheet

REDACTED

Date 6/19/81

All values are visual means of the DP 47201A recording for each minute.

at the termination of the test.

Submaximal Constant Workrate Tests

Incremental test

kg-m/min	kg-m/min	kg-m/min

Rest	Rest	Rest

Minute	kg-m/min	%
0	Rest	96
1	200	96
2	"	96
3	"	96
4	"	96
5	300	96
6	400	96
7	500	96
8	600	96.5
9	700	97
10	800	97
11	900	97
12	1000	97
13	1100	97
1	Rest	96.5
2	Rest	97
3	Rest	97

DUP 0947281

DU 038575

PULMONARY FUNCTIONS

WAFW

	6-19-81		PRED.	DATE		PREDICTED
(L)	3.90	(106)	3.67	RV (L)		
	5.46	(109)	5.02	FRC (L)		REDACTED
(L)	71			TLC (L)		
75%	3.03		3.60	RV/TLC (%)		
50%	0.90			D _L CO _{sb} * (ml/min/mmCO)	26	30

*Patient effort on the Dco test was not optimal
however the result is normal M.E. McNabb, M.D.*

*In patients with anemia or polycythemia D_LCO should be corrected, i.e.
uncorrected D_LCO = corrected D_LCO (Am. Rev. Resp. Dis. 1970, 102, 965)
.06865 X Hgb (gm%)

PATIENT EFFORT

Inconsistent

INTERPRETATION:

NORMAL

ABNORMAL

BORDERLINE

IMPAIRMENT:

OBSTRUCTIVE

RESTRICTIVE

BOTH

SEVERITY:

MINIMAL

SLIGHT

MODERATE

MARKED

SEVERE

COMPARISON - PREVIOUS STUDIES:

No previous tests.

DATE	TIME	PaO ₂ (mmHg)	PaCO ₂ (mmHg)	pH	H ⁺ (NM/L)	HCO ₃ (MeQ/L)	COMMENTS
1/9/81	2:45 P	97	34	7.41		21	Room Air; Immediately after maximal exercise

M.E. McNabb, M.D.

VAF 10-17(598)
Feb. 1980

DUP 0947282

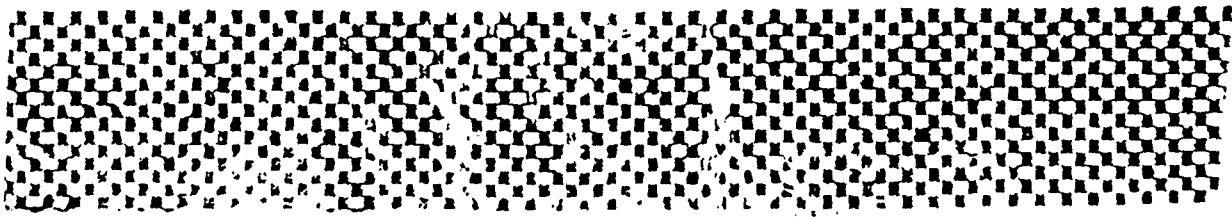
DU 038576

LOAD STATE EXERCISE: Average values representative

AND [REDACTED]

WARD 21A MC, DATE 6/19/81

VARIABLE	UNITS	POWER OUTPUT						Rep
REDACTED								
WORK RATE	kg-m/min.	200	200	400	400	600		200
O ₂ UPTAKE	ml/min.	800	735	1200	1000	1600		680
CO ₂ OUTPUT	ml/min.	700	540	1150	810	1600		500
RESPIRATORY EXCHANGE RATIO		0.90	0.73	0.95	0.81	1.00		0.73
CARDIAC FREQUENCY	beat /min.	95	94	110	100	125		89
VENTILATION	l/min.	20	27.3	32	37.25	42		24.7
RESPIRATORY FREQUENCY	per min.	18	35.8	20	39.7	20		31.9
TIDAL VOLUME	ml.	1100	760	1600	940	2100		770



DUP 0947283

DU 038577