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State Board of Workmen's Compensation
494 Labor Building
254 Washington Street, S.W.
Atlanta, Georgia

Re: Mr. Larkin Houston

This 56 year old utility man in the weaving division of the Asbestos Mill at UniRoyal was first hired in May of 1949. It has been seventeen years and three months since his original exposure.

He has a total dust exposure of 48.3 MPPCFY.

Mr. Houston was first seen in 1957 and stated at that time that he was entirely well. His past history was remarkable only in that he had sustained a fracture of his nose in 1935, and had been left with a deviation of the nasal septum which, he stated, created no difficulty.

Physical examination in 1957 revealed a blood pressure of 110/70 and completely normal heart and lungs. There was no clubbing.

He has been seen annually since that time; and in April of 1959, stated that he had had a good year but noted that his wind seemed a little shorter on heavy exertion and that he gave out a little more easily than in previous years. Physical examination was normal.

There have been no dramatic and sudden changes since that time. However, he has noted a steadily progressing shortness of breath on exertion and during the past year has observed that his breathing reserve has been steadily diminishing, and particularly when attempting to push heavy objects or exert himself.

During the past year, this sense of dyspnea has worsened and patient has found himself losing increasing amounts of work-time because of a sense of steadily diminishing strength, energy and stamina and increasing dyspnea.

Since July of 1967, he has found himself becoming short of breath quite easily and in the course of his daily job which has required considerable lifting and pushing of heavy objects. He notes that if walks on even a straight and level surface for any period of time, his breathing becomes progressively shorter.

He has been coughing in the morning for the past several years and has observed that the morning cough has become more severe in the past year or two and has become productive of small to moderate amounts of dark, sometimes yellowish, sputum which has never contained blood.

He has begun to cough more during the day but during this time has not produced sputum in significant degree. He has been unaware of wheezing or rattling within the chest.

There have been occasional parasternal aching pains which have been related to hard breathing but not specifically to exertion: and there has been no radiation of pain.

He has become aware of his heartbeat following exertion in the last few months and notes that it tends to pound in a regular fashion.

He has had no dependent swelling but observes that his legs tire more easily than they formerly did. They do not ache on exertion.

Since July of 1967, he has begun having bilateral upper parasternal aching discomfort which seems occasionally to radiate into the right subscapular region. This ache has been relieved by rest.

Because of increasing fear of coronary artery disease he received an Exercise Tolerance Test on 2 November 1967. At this time he walked on a treadmill for three minutes at a rate of two miles per hour. During the third minute he became moderately increasing dyspneic, but experienced no chest pain and was not observed to cough.

Immediate post-exercise, two and five minute electrocardiograms were obtained and showed no significant ST-T wave changes.

Exercise cardiogram was interpreted as negative and this information was given to the patient.

Annual physical examinations since 1957 have revealed the nasal septum to be deviated to the right and partial obstruction to breathing through the left nostril. There has been no evidence of sinus disease manifested by supra-sinus tenderness.

Respiratory excursion and descent of bases to percussion has been adequate and remains so. The lungs have been consistently clear except in August of 1967 when some diminution of breath sounds over the bases anteriorly and posteriorly was noted. There have been no rales.

The cardiac examination has been consistently normal and the second aortic sound has always been normally louder than the second pulmonic sound. However, again in 1967, A2 was noted to be only slightly greater than P2. There have been no murmurs or evidence of peripheral arteriosclerosis.

Urinalyses have been consistently normal over the past ten years.

So, also, have white counts and differential examinations of the stained blood.

Packed corpuscular volumes have varied from 49 in 1957 to 50 in 1967. The hemoglobin has generally stayed in the neighborhood of 14.7 grams per cent.

He has tended to have slightly more than usual number of red cells. His RBC was 6.8 million units in 1957 and has steadily but slowly decreased since then to 5.53 in 1962 and 4.98 million cells per cubic mm. in 1967.

Serial chest x-rays obtained during this decade have been consistently normal except perhaps for a very slight decrease in the vertical diameter of his chest in the past three years. Slight irregularity of the left cardiac outline has appeared as has a very small tenting adhesion in the mid portion of the left diaphragm.

The vascular markings remain distinct although a very minimal nodularity is making its appearance in the lower medial right lung field.

These x-ray changes are not yet diagnostic of pulmonary asbestosis as we have learned to recognize it in this mill, but are quite suspicious and combined with other findings (noted below) render such a diagnosis highly probable.

Vital capacities have changed very little since 1962 although he has gained ten pounds in weight and have decreased from 3.1 liters to 3 liters. Both values are equivalent to 75 per cent of predicted normal.

Forced vital capacity at 0.5 seconds has decreased from 2.57 to 1.92 liters or from 83 to 63 per cent of predicted normal.

The forced expiratory volume at 1.0 seconds has not changed as greatly and has decreased from 2.94 to 2.47 liters or from 92 to 82 per cent of predicted normal.

He continues to have satisfactory maximal expiratory flow rates and maximal mid expiratory flow rates, although there is again a decreased from 6.8 to 5.0 and from 3.6 to 3.3 liters per second.

His maximal ventilatory free has decreased from 1.70 to 1.15 liters or from 119 per cent of predicted normal to 85 per cent.

His alveolar ventilation indices has shown a steady decrease from 1.6 to 1.1, although the latter figure is not in and of itself pathognomonic by any means of obstructive disease.

The significant observation from these values is that he continues to have normal ventilatory ability without evidence of significant expiratory obstructive disease. There is, however, an increasing decrement in vital capacity and maximal voluntary ventilation values.

His residual volumes calculated as percent of total lung capacity (October 1967) are 25.1 and 25.8 per cent. For a man of his age and body surface area, the predicted value is 30.8 per cent. Again, it can be seen that there is no evidence at this time of significant emphysema or obstructive difficulty.

The defect is more that of a restrictive defect and this also demonstrated graphically on his spirograms.

In October of 1967, he received numerous other blood gas studies.

A seven minute pulmonary nitrogen emptying rate was performed and showed 1.2 per cent nitrogen at seven minutes. This test suggested normal mixing of the inspired gases.

His arterial partial pressure of oxygen following fifteen minutes breathing of 100% oxygen was 600 ml. of mercury.

Steady state-resting carbon monoxide diffusion studies were performed and yielded values of 11.98 and 11.7 ml. of carbon monoxide per minute per mm. of mercury.

Minute volumes of ventilation on these tests were 8650 and 8120; and minute alveolar ventilation was 5120 and 4800 ml. per minute.

The ratio of dead space volume to tital volume was 0.31 and 0.32 - values again suggesting an absence of significant obstructive ventilatory defect.

Physiological dead space on these two examinations was 136 and 148 ml.

There was some evidence of alveolar hyperventilation manifested by partial pressures of oxygen in the alveoli of114.2 and 115.8.

The Aa pressure differences were 46.9 and 48.5 mm. of mercury and his arterial oxygen saturation was 92.2 and 92.2 per cent on the two diffusion studies.

These studies are interpreted as indicating an alveolar-capillary diffusion defect resulting in less than normal arterial oxygen saturation.

With exercise at one mile per hour (1.5 ft. per sec.), he is able to increase his arterial oxygen saturation to 94.8, but as noted above, became increasingly fatigued and dyspneic - obviously being able to maintain this increase only at the expense of increasing work expenditure and fatigue.

Mr. Houston has shown a moderately accelerated downhill course during the last twenty-four months, as noted subjectively in his history.

Although there are a few physical findings and only early roentgenologi findings to validate his symptomatic complaints, the physiological function tests do provide a concrete basis for his complaint.

Clinical Impression:

1. Pulmonary asbestosis, due to occupational exposure to asbestos fibers
   a) restrictive ventilatory defect, secondary to one
   b) Alveolo-capillary diffusing defect, secondary to one, with moderate arterial oxygen unsaturation.

2. Deviated nasal septum
3. Right epididymal mass, benign

I would judge that he is approximately fifty percent incapacitated at this time as a result of his asbestos disease; and that he can never be expected to return to heavy physical work. The outlook for progression of this disease must be regarded as hopeful, but guarded.

John G. Wells, M.D.