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ARLAN CONN, M.D.
LEON LEWIS, M.D.
SHELDON MARGEN, M.D.
8438 WESTER STREET
BERKELEY 8, CALIFORNIA
THOMAS 8-7788

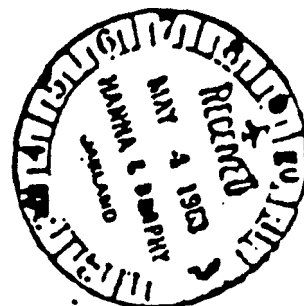
INTERNAL MEDICINE

April 23, 1963

Hanna and Brophy
1540 San Pablo Avenue
Oakland 12, California

Attention: Mr. Ivan A. Schwab

Re: MEYER, Mr. Edward
vs Plant Rubber and Asbestos Works, et al
IAC Claim Number: SF 199-141



Gentlemen:

Mr. Meyer was examined today at your request at the San Leandro Memorial Hospital. He is a 48 year old male, married, with two adult children.

EMPLOYMENT HISTORY:

Plant Rubber and Asbestos Works, asbestos insulation worker, five or six years to March 1962. During 21 years he worked for nine different employers, always performing the same type of work, covering pipes and boilers with asbestos molds. The material used was called 85% magnesium pipe covering.

PHYSICIANS:

R. M. Adams, M. D.; G. Stephens, M. D.; K. Hardy, M. D.; C. M. Christiansen, M. D. (radiology - therapy).

HOSPITALIZATION:

Doctors' Hospital, San Leandro, March 22, 1962, for 7 days; April 10 - 21, 1962. San Leandro Memorial Hospital, April 19, 1963, to the present.

FAMILY HISTORY:

Father died at 66 of a "stroke". Mother died at 60 of cancer of the liver. One brother died at 39 of a "stroke"; another living brother has back trouble. Two sisters are well. Mr. Meyer's wife has chronic pancreatic insufficiency. His two adult children are well. There are no other instances of cancer in the family other than his mother. There is no family history of diabetes or tuberculosis.

PERSONAL HISTORY:

Mr. Meyer was born April 8, 1915, in New Haven, Connecticut. His family moved to the Bay Area during his childhood, and he has lived here ever since. He

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completed the first year of high school. There was no military service. He limited the use of tobacco to about one package of cigarettes daily for many years. Alcohol was used in moderation.

Mr. Meyer has been certified for Social Security benefits on the basis of disability.

PAST MEDICAL HISTORY:

He recalls no significant past illnesses. In 1952 there was a head injury with skull fracture while he was installing insulation in the bottom of a ship and a ballast weight dropped on his head. In 1957 craniotomy was performed for removal of subdural hematoma due to the injury five years previously. (W. B. Allen, M. D.) During the interval between head injury and craniotomy he developed post-traumatic epilepsy, and Dilantin therapy was instituted. He has continued the use of this drug to date, although he has had no recurrence since operation. On occasions he sees Dr. L. Lawrence for follow-up of his cerebral injury. Appendectomy was performed (date not recalled). He developed a post-appendectomy fistula and required secondary repair. He was once thought to have ulcerative colitis. In 1958 or 1959 he was hospitalized at Providence Hospital with the diagnosis of pneumonia.

SYSTEMIC REVIEW:

Mr. Meyer is too distressed to interrogate in detail. He has suffered from constipation recently, possibly resulting from medication. His principal symptoms are indicated by the Chief Complaints.

CHIEF COMPLAINTS:

1. Persistent chronic cough, productive of whitish expectoration, of several years' duration.
2. Increasingly severe cough since March 1962.
3. Progressive shortness of breath.
4. Acute episode of shortness of breath four days ago.

HISTORY OF THE PRESENT ILLNESS:

On April 19, 1963, Mr. Meyer entered San Leandro Memorial Hospital on the advice of Dr. Robert Adams, because of acute dyspnea, cough and fever. On April 14 he was chilled when he was caught in the rain. He developed an acute respiratory infection, and became very dyspneic the following day. Dr. Hardy suggested that he use a steam tent at home, but his cough became worse, more productive, and the temperature rose to 103° F. On April 19 his chest became quite painful, and he was ordered to the hospital.

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For approximately three years prior to a thoracotomy in April 1962, Mr. Meyer had noted progressive shortness of breath. This became very much worse before his hospitalization a year ago.

In April 1962 Dr. K. Hardy performed a thoracotomy for biopsy of the lung. Mr. Meyer says that "they got a tumor on my chest".

Mr. Meyer ascribes the shortness of breath to a tumor and damage to the lung due to inhalation of asbestos. During the 21 years he has worked with asbestos insulation, he has worked without respiratory protective devices -- at times in situations where considerable dust was generated, and at times where ventilation prevented dust accumulation. He states that none of the workers with whom he has been associated have worn masks or other protective devices.

Since surgery in 1962 Mr. Meyer has been totally disabled. He has been unable to walk more than a very short distance without having very marked shortness of breath. He has spent the time at home. He received a series of radioactive cobalt treatments, under the supervision of Dr. Christiansen. This did not prove helpful. He has remained under the supervision of Drs. Hardy and Adams.

PHYSICAL EXAMINATION:

Estimated weight, 170 pounds (or less). Estimated height 68 inches. Blood pressure 140/90 (recorded blood pressure values on the hospital chart are all in normal range).

Mr. Meyer is a moderately uncomfortable, obviously dyspneic white male, of mesomorphic constitution, well muscled and without apparent deformity. There are bilateral scars of the frontal area and one of the left temporal area; there is a right thoracotomy scar, and a right abdominal incision.

Aside from the scars and slight depressions in the areas, the cranium is negative. Eyes, ears, nose and throat are clear. There are fissures in the angles of the mouth. The tongue is smooth and pink. The palate and pharynx are clear. The mouth is edentulous, well compensated.

The neck is free of palpable lymph nodes, and there are none in the axillae. The thyroid gland is not enlarged. Carotid artery pulses are normal.

The thorax is asymmetrical. The right hemithorax is smaller than the left. There is generalized impairment of resonance and dullness at the right base. Breath sounds are distant, almost absent at the right base, and are bronchovesicular to bronchial in character throughout. There are many loud inspiratory rales of both lung bases, more marked on the right than the left. The area of

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cardiac dullness cannot be made out. Heart rate and rhythm are normal. The aortic second sound is somewhat accentuated. There are no murmurs.

The abdomen is soft. There are no palpable masses. The liver is not enlarged. The genitalia appear normal.

The extremities show slight cyanosis of the fingernails. The toenails are all somewhat abnormal. Several are markedly horny and thick. There is no clubbing of the fingers or toes.

Neurological Examination:

Mr. Meyer is a well oriented, intelligent and cooperative man, who seems to bear his extreme disability with remarkable equanimity. His manner is friendly, and his mood is not remarkably depressed.

As tested in bed, there are no apparent abnormalities of the nervous system. Cranial nerve examination is negative. Sensory and motor function are intact. Reflexes are physiological.

REVIEW OF MEDICAL REPORTS:

An early radiograph of February 10, 1962, is reported by J. D. Coate, M. D., as showing: a soft tissue shadow protruding from the right side of the mediastinum at the level of the aortic arch and "bilateral thickening of the bronchovascular shadows in both lower lobes, as described on previous examination April 22, 1961". Dr. Coate thought the changes in the lower lobes suggested pulmonary occupational disease.

Blopsy specimens from thoracotomy on April 11, 1962, consisted of two pieces of lung, one of which consisted mostly of tumor showing the microscopic character of oat cell bronchogenic carcinoma. This section, as well as the one without tumor, showed a few asbestos bodies and variable fibrosis.

Although there are no reports on file directly from Dr. K. Hardy, the thoracic surgeon who performed thoracotomy, there is a report from Dr. R. M. Adams which indicates that he considered Mr. Meyer permanently disabled, "mainly because of his inoperable carcinoma of the lung . . ." which he thought "directly connected with the asbestosis which he developed as a result of his employment".

This view is supported by Dr. R. C. Dickerman, pathologist, in a report of November 27, 1962. He reviewed the blopsy material and listed four references dealing with pulmonary asbestosis and carcinoma. The articles referred to were published between 1939 and 1948.

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ANNOTATED BIBLIOGRAPHY:

Three of the articles referred to by Dr. Dickman were submitted for review by Mr. James J. Vonk of the State Compensation Insurance Fund.

1. Cureton, R. J. R., "Squamous Cell Carcinoma Occurring in Asbestosis of the Lung", British J. Cancer, 2:249, 1948.

The author reports a case of a 37 year old woman who worked in a factory making asbestos pipe covers for seven years between ages 15 and 22. She developed severe respiratory symptoms, terminally a pleural effusion, and clinical signs suggesting neoplasm. At autopsy a bronchogenic carcinoma was found in the left lower lobe. On histologic examination, this was considered to be a non-keratinizing squamous cell carcinoma. There was also sub-pleural fibrosis, thought due to asbestosis. Typical giant cells and asbestos bodies were observed. The author refers to prior observations in 19 cases of neoplasm associated with asbestosis up to 1943, and states that the question of relationship between the two disorders could not at that time be specified.

2. Homburger, F., "The Coincidence of Primary Carcinoma of the lungs and Pulmonary Asbestosis: Analysis of Literature and Report of Three Cases", American J. Path., 19:797, September 1943.

Homburger reports "at least 19 known cases", including the three he reported, of asbestosis associated with pulmonary carcinoma. In his laboratory there were four instances of carcinoma in eight cases of asbestosis examined.

In a tabular summary of the reported cases, it is interesting to note that the duration of exposure varied from 19 months to 25 years, that there were intervals of freedom from exposure before death for as long as 15 years, that almost all of the tumors were squamous or oat cell type, and that all but four of the carcinomas are indicated as having occurred in either the right or left lower lobe.

3. Hueper, W. C., "Environmental and Occupational Cancer", U. S. Public Health Service, Supp. 209, 1949.

Hueper reports 23 cases of "asbestos cancer of the lung" in the American, English and German literature since 1935. Among 92 autopsies on asbestos workers reported by a German observer (Wedler), there were 14 cases of lung cancer, 4 in females.

Hueper states that histological character of the cancers and histogenetic derivation "support a specific causation of these reactions by inhaled asbestos dust".

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The following additional summaries are submitted, as selected from a large number of articles reviewed:

4. Bonser, G. M., Faulds, J. S., and Stewart, M. J., "Occupational Cancer: of the Urinary Bladder in Dye-stuff Operatives and of the Lung in Asbestos Textile Workers and Iron-Ore Miners", A. M. J. Clin. Path., 25:126, February 1955.

This study from the University of Leeds, Carlisle, England, states that in 1928 one of the authors (MJS) began to collect data from workers in two factories and eventually had a series of 80 autopsies among workers exposed to asbestos. In the lungs of 8 persons autopsied, there were small numbers of asbestos bodies, but no pulmonary asbestosis. A total of 72 subjects -- 45 male and 26 female -- had asbestosis, and among these 12 males and 3 females had developed tuberculosis, and 12 males and 2 females had developed lung cancer. In other words, among the males 26.1% developed cancer, and among the females 8.7% developed cancer. The average age at death was 56.

The authors point out that "no adequate comparable post mortem material from persons working under factory conditions and not exposed to asbestos dust is available with which to compare the incidence of lung cancer in asbestos workers. The incidence here cited is very high, and in comparison with the incidence in iron-ore miners must be highly significant. The occurrence of occupational lung cancer in the exposed female is noteworthy." The authors state that, following introduction of regulations for the control of asbestos dust, the occurrence of both asbestosis and associated cancer showed a definite decline.

5. Doll, R., "Mortality from Lung Cancer in Asbestos Workers", Brit. J. of Indust. Med., 12:81, April 1955.

Doll reports the cause of death as determined at autopsies for 105 persons employed at one asbestos works. Lung cancer was found in 18 instances, 15 times in association with asbestosis.

The following interesting comment occurs in the summary: "113 men who had worked for at least 20 years in places where they were liable to be exposed to asbestos dust were followed up, and the mortality among them compared to that which would have been expected on the basis of the mortality experience of the whole male population. 39 deaths occurred in the group; 15.4 were expected. The excess was entirely due to excess deaths from lung cancer (11 against 0.8 expected) and from other respiratory and cardiovascular diseases (22 against 7.6 expected). All the cases of lung cancer were confirmed histologically, and all were associated with the presence of asbestosis." (Note: Underscoring by L.L.)

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6. Lynch, K. H., McIver, F. A., Cain, J. R., "Pulmonary Tumors in Mice Exposed to Asbestos Dust," A. M. A. Arch. Indust. Health., 15:207, March 1957.

These workers at the Harvard School of Public Health exposed mice to dusty atmosphere containing asbestos fibers. 127 animals, killed after 17 months' exposure, were compared to 222 controlled animals. Primary lung tumors were found in both groups: 40% of the dusted mice and 36% of the controls. Multiple pulmonary tumors were demonstrated in 10% of the dusted and 8% of the controls. The authors were unable to establish proof of malignancy. They state they were "unable to demonstrate any proof of the carcinogenicity of asbestos under the conditions of this experiment, but the increased incidence of multiple lung tumors in dusted animals must be regarded as a possible accentuation by the inhalation of asbestos dust of an existing tendency to develop lung tumors". They disclaim any significant contribution of this study to solution of the problem of relationship between asbestosis and cancer in man.

7. Braun, D. C., Truon, T. D., "An Epidemiological Study of Lung Cancer in Asbestos Miners", A. M. A. Arch. of Indust. Health., 17:634, June 1958.

This comprehensive article reviews the evidence for and against the association of lung cancer with asbestos exposure and asbestosis. The authors point out that there has been a serious defect in most of the studies -- namely, that they were concerned only with the dead and not with the living workers in the asbestos industries. In order to remedy this defect, they studied causes of death among a large segment of population in Quebec, and attempted to relate cancer of the lung to dust exposure, duration of employment, and smoking habits. In the end they compared the death rates from cancer of the lung for asbestos workers to that of other population groups in Quebec, according to accepted statistical methods.

They state: "The mortality rate for lung cancer as computed on the basis of 9 'proved' deaths among the cohort (i. e., asbestos exposed. L. L.) was 25.3 per 100,000. When the 3 small 'suspected' cases were added, the total 'rate' for the cohort was 33.8." According to their findings, these authors thought the mortality rate from lung cancer did not appear to increase with length or degree of exposure -- facts which they considered strong evidence against the carcinogenicity of asbestos.

Although there were small variations in rate among various population groups studied, the authors state: "On the basis of what are believed to be complete and reliable data, it seems fair to conclude that the asbestos miners in the Province of Quebec do not have a significantly higher death rate from lung cancer than do comparable segments of the general population."

This article does not give an account of the mineralogy of asbestos in Quebec. The study was made possible through a grant from the Quebec Asbestos Mining Association.

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Braun and Truan quote Goldblatt and Goldblatt, who contributed a section edited by E. R. A. Merewether, the head of official industrial hygiene for Great Britain, entitled: "Industrial Carcinogenesis and Toxicology". The Goldblatts said, with reference to asbestos, "there is no reliable criterion by which one can anticipate carcinogenicity and, as is well known, relatively minute changes in the structure of a chemical carcinogen are sufficient to diminish or eliminate carcinogenetic action. . . . If asbestos is indeed to be regarded as a carcinogen, the need is felt to demonstrate some property which can be regarded as something more than inertness". They thought that until evidence of direct carcinogenesis was established, asbestos might be considered as a "co-carcinogen".

This point is, in our opinion, very important, since it is possible that known chemical carcinogens, such as those in tobacco tars, may actually be concentrated by absorption on asbestos dust and fibers. The possibility that carcinogenesis may be abetted by asbestos seems to us to be a fruitful approach to further investigation.

8. Anderson, J., Campagna, F. A., "Asbestosis and Carcinoma of the Lung: Case Report and Review of the Literature", Arch. of Environmental Health, 1:27, July 1960.

This is another single case report of a 52 year old man who worked for 30 years as an asbestos insulator, both inside and outside, in new and old buildings. He wore no protective gear at any time. In addition to asbestos, he was exposed to fiberglass. He was a heavy smoker until five years prior to his death, having smoked "three packages of cigarettes daily for many years". The autopsy findings revealed diffuse asbestosis with typical asbestos bodies, alveolar septal thickening, and thickened pleura. There was a large tumor mass of the right hilar area which, on microscopic examination, revealed bronchogenic carcinoma.

This case report illustrates the difficulty of distinguishing the carcinogenic effect of other pulmonary irritants from that of tobacco.

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DISCUSSION:

In an early article on asbestos hazards published by the International Labor Office in 1938, criteria were proposed for the establishment of industrial origin of cancer. It was said the following two conditions must be satisfied: "(1) that the incidence-rate in the occupation under review should exceed that in the general population to a significant extent and (2) that in the occupation concerned there should be sufficient association of a worker with a substance

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proved experimentally to have carcinogenetic properties." At that time the author said, "These two postulates cannot yet be regarded as having been fulfilled in the case of the asbestos industry, but there is sufficient evidence to warrant careful observation in the future."

In 1963, 25 years after these remarks were written, it can still be said that absolute proof of causal relationship between asbestos and lung cancer has not been established. Detailed epidemiological work, such as that reported by Braun and Truan, suggests the possibility that autopsy data may be misleading. The increasing frequency of lung cancer among both sexes, and the apparent correlation with use of tobacco and inhalation of smog, undoubtedly confuse the issue.

Despite the absence of certain proof, however, there are several suggestive indications of relationship between asbestos inhalation and lung cancer. These are:

1. The unusually high percentage of cancer deaths among individuals with proved asbestosis.
2. The relatively high frequency of lung cancer in women asbestos workers -- data observed before the frequency of lung cancer in the female began to rise in the general population.
3. The high prevalence of carcinoma of the lower lobe, as contrasted with a more random distribution and relatively high incidence of upper lobe carcinoma in general.
4. The frequent occurrence of squamous and oat cell carcinoma in association with asbestosis.
5. The decline in the frequency of asbestosis and of lung cancer among British operatives after the institution of dust control measures.

On the basis of the established diagnosis in this case and the admittedly disputable but generally positive evidence of correlation between asbestosis and lung cancer, it is our opinion that Mr. Edward Meyer is suffering from a disease which has as part of its etiology occupational exposure to asbestos fibers.

DIAGNOSIS:

1. Pneumoconiosis: Asbestosis.
2. Oat cell carcinoma of the lung.

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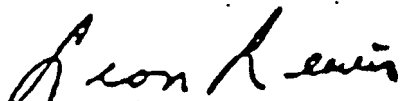
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3. History of post-traumatic epilepsy and late surgical treatment of
subdural hematoma.

Sincerely yours,


Leon Lewis, M. D.

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