FILE NAME: Brakes (BRK)

DATE: 1984 b2Ø

DOC#: BRK08y

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November 27, 1984

Gary Galiher & Associates
Attorneys at Law
Suite 88, Melim Building
333 Queen Street
Honolulu, Hawaii 96813

Re: [redacted]

Dear Mr. Galiher:

On 11/10/84, I reviewed the case of [redacted] at the request of Mr Galiher, after receiving medical reports on 11/9/84. Following are pertinent findings in the medical records:

SYMPTOMS

He first noticed dyspnea on exertion in 1975, when he was skin diving. Dyspnea and non-productive cough increased in 1980, when he could only climb one flight of stairs.

OCCUPATIONAL EXPOSURE TO ASBESTOS

He was a general mechanic at Schoffield motor pool and ordinance for over 30 years, where he worked on auto brakes and clutches. He would remove old brake lining, cut new lining to size, drill holes in lining, rivet the lining to the clutch and brake shoes and then would grind the asbestos on a sanding machine to correct size. He used compressed air to blow out dirty brake assemblies. He used asbestos cloths in welding. He would sweep the work place, daily thereby raising a cloud of dust.

SMOKING

He smoked one half pack of cigarettes daily for 34 years..He stopped in 1975,

XRays

First report, Feb. 15, 1979, was "Diffuse interstitial pulmonary disease of unknown etiology".

On 3/7/80, Dr. Sakuda reported his XRays to show "Moderate diffuse interstitial infiltration with a nodular pattern and left diaphragramtic calcification. These changes were compatible with previous asbestos exposure".

Dr. Childs reported on 1/20/81,"That the pattern of diffuse pulmonary fibrosis is compatible with the clinical diagnosis of asbestosis".

PULMONARY FUNCTION TESTS

A screening pulmonary function test in June 1978 was abnormal.

On 1/23/81, he was reported to have "moderate restrictive lung disease with deterioration of lung volume with increased restriction since 3/7/80. From March 7, 1980 to 1/30/81. total lung capacity decreased from 3.9 liters to 3.6..Vital capacity decreased from 2.8 to 2.47 liters. ". Findings were consistent with asbestosis.

PHYSICAL EXAMINATION

January 3, 1981. Dry interstitial rales were heard in both lung bases. There was questionable early clubbing of fingers.

Biopsy from bronchoscopy on 1/6/82, revealed no evidence of malignancy.

DIAGNOSIS

Dr. Massey - Markers of asbestos exposure and asbestosis present 5/21/80.

Dr. Morgan - Deterioration in pulmonary function tests, clinical symptoms, chest XRay and History are all related to progression of asbestosis.

CLINICAL COURSE

Dr. Schonfeld, Major M.C., filed a report to U.S. Army personnel Office on 12/8/78, reqesting full permanent disability for Mr. [redacted], due to asbestosis, effective 12/14/78.

He was admitted to Kuakini Medical Center on 1/28/83 for hemoptysis and influenza. His abnormal breath sounds increased with an increase of diffuse infiltrate on XRay. An XRay on 1/24/83 was reported to show an acute component besides chronic interstitial disease which could represent infection.
His pulmonary arterial oxygen level was low, despite use of oxygen. On 1/31/83, he had a respiratory code. On 2/9/83, he had a tracheostomy and died later the same day.

DIAGNOSES AT TIME OF DEATH

1. Influenza
2. Asbestosis ...parenchymal & pleural
3. Cor Pulmonale

AUTOPSY FINDINGS...2/10/83

LUNGS - MACROSCOPIC

Diffuse nodules, 0.5 cm. in diameter, were present under the thin visceral pleura of both lungs. The lungs were firmer than usual. The cut surface reveal numerous empty cystic bulbous spaces, 1 cm. in diameter, most pronounced in the sub-pleural aspects. Sub-pleural empty cystic spaces measuring up to 2.5 cm. are present in apices of both upper lobes.

LUNGS _ MICROSCOPIC

Sections of all lobes are examined microscopally and appear similar. The visceral pleura is thin. Many distal airways are moderately to markedly dilated and are lined focally by a stratified squamous epithelium. Some of these lumin are filled with collections of neutrophiles, necrotic cells and fibrin.

There is marked fibrous thickening not only of the alveolar septas but also of the alveolar ducts and respiratory bronchioles. These thickening walls are characterized by fibroblasts, wavy collgenous tissue, smooth muscle-like tissue, many small blood vessels, moderate numbers of lymphocytes and plasma cells, with lesser numbers of neutrophiles. Also present are scattered histocytes, containing both anthrocotic pigment and hemosiderin. Definite asbestos or ferruginous bodies are not identified in the interstitium.

Scattered alveolar spaces contai n collections of neutrophiles, degenerating cells, fibrin and hemosiderin-laden macrophages. Others reveal connective tissue proliferation within the alveolar spaces associated with small blood vessels and chronic inflammatory cells.

Examination of the lung tissue after digestion reveals the presence of two asbestos bodies. A quantitive count is 2 bodies per 5 gm. of lung tissue.

AUTOPSY DIAGNOSES

1. Acute and organizing bronchopneumonia bilateral, patchy. moderate
2. Chronic bronchiectasis and bronchitis diffuse, moderate, bilateral
3. Bronchial, squamous metaplasia, bronchi multifocal, moderate, bilateral
4. Medial hypertrophy, pulmonary arteries moderate, bilateral, compatible with clinical, pulmonary hypertension
5. Chronic, Interstitial fibrosis, diffuse, severe bilateral
6. Asbestos bodies: 2/5/gm. tissue

On or about 4/18/84, I received 6 paraffin blocks of [redacted] autopsy from Kuakina Medical Center. They were labelled 18-7 LUL, 18-8 LLL, 18-9 LLL, 18-10 RUL, 18-11 RML, and 18-12 RLL. These blocks were from Autopsy 83-A-18. The numbers 83-A were missing from the blocks. Hemotoxylin Eosin and iron stain slides were made from each block. On histological examination, the H&E slides were similar. There was extensive severe interstitial fibrosis, moderate amount of anthracosis,and peribronchiolar fibrosis. Numerous macrophages were in the alveolar spaces. Many bronchi were dilated - some contained inflammatory cells- evidence of bronchiectasis and honeycomb disease. Numerous chronic inflammatory cells were dispersed in the interstitium, suggestive of viral pneumonia. Thickened pleura with evidence of pleural plaque formation was found in the LUL. Asbestos bodies were not found on any of the H & E and iron stained slides.

On Sep. 25, 1984, I received 6 slides on Autopsy 83-A-18, [redacted]....two of lymph nodes, H & E , one of LLL,LUL,and RLL iron stain, and one large slide of lung digest. There were also 22 Kodak 2' x 2' transparencies.

The findings of severe interstitial and peribronchial fibrosis with honeycomb disease, plus early pleural plaque formation are consistent with and supportive for the pathological diagnoses of parenchymal and pleural asbestosis.

Other diagnoses were:

1. acute and chronic bronchopneumonia
2. chronic bronchiectasis
3. acute and chronic bronchitis

Interstitial or virai pneumonia can induce interstitial fibrosis. However, it does not produce honeycomb disease or pleural plaque formation, as present in this case.

INABILITY TO DEMONSTRATE ASBESTOS BODIES

As expected, asbestos bodies were not found in the brake worker. Chrysotile asbestos used almost exclusively in auto brakes (1) does not form asbestos bodies (2). Chrysotiie fibers in the lung, fragment into subunits and are leached of mineral constituents with the passage of time (3).

Warnock (4) indicates, because of preferential of chrysotile, that persons who inhale only chrysotile might be found to have asbestos related fibrosis or cancer and a small concentration of fibers.

In animals, after 18 months inhalation of anthrophyllite and chrysotile, only the chrysotile would disappear from the lung. Chrysotile seldom forms asbestos bodies in man (5).

An analogy can be made in the management of a clinical case of pulmonary tuberculosis, in which the tubercle bacilli, similar to our asbestos bodies, cannot be demonstrated. In this situation, the patient may have a history of contact with people with tuberculosis. clinical signs of the disease, such as a productive cough, fever, hemoptosis, fatigue, night sweats, weight loss and definite XRay findings of tuberculosis. One thing is missing, and that is the ability to find tubercle bacilli by culture or smear.

The overwhelming majority of physicians woud treat this patient

for tuberculosis because of the reasonable medical certainty that he has the disease, despite the inability to demonstrate tubercle bacilli.

IN SUMMARY, I can state with reasonable medical certainty that the severe pulmonary and pleural fibrosis in Mr. [redacted]'s lungs, that contributed to his death, was the result of asbestos exposure and the clinical diagnoses, supported by the histological findings are:

SEVERE PARENCHYMAL AND PLEURAL ASBESTOSIS
WITH HONEYCOMB DISEASE

References

1. Rohl,A.N. et al -Asbestos Exposure During Brake Lining Maintenance and repair ..Envir. Res 12-110 1976

2. Churg, A.M. & Warnock,M.L. Asbestos and Other ferruginous Bodies Am,J.Path 102:447 1981

3. Craighead, J.E.-Archives Path. & Lab. Med. Report of Pneumoconiosis Comm..106:543 -1982

4. Warnock, M.L. et al -The Relation of Asbestos & Lung Cancer Path. Annual 18 109 1983

5. Casey, F.R. et al -Asbestos Related Diseases -Clin. Chest Med. 2: 170-202 1981

Sincerely yours,

Albert Ehrlich

Albert Ehrlich, M.D.
Pathologist

AE:ee
cc. T. Hart 111
encl: Prints and transparencies
Statement