367 17

CCD 75

MEDICO-LEGAL OPINION

in
respect
of

OCCUPATIONAL CHEST DISEASE

by

G. W. H. Schepers, M.D., D.Sc., F.C.C.P.
Director
Saranac Laboratory
7 Church Street
Saranac Lake, N.Y.

November 5, 1956

Claimant: W.E. Taylor
Deceased - June 1953
Saranac Laboratory No. P-56-636

Referred by: Olson and Olson
111 North 5th Avenue
P.O. Box 830
Pasco, Wash.

M-367 83

CRMC-Motley1000-Subj-001731

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Re: W.E. Taylor
OCD 75 - P-56-106
November 5, 1956

I. MATERIAL STUDIED

A. Report on dust exposures at asbestos cutting operation: White Bluffs - H.B. Perry - July 7, 1956

B. Asbestos Identification - R.E. Brown - June 19, 1956

C. Record of Employment - Olson and Olson - Oct. 18, 1956

D. Autopsy Protocol - Robert D. Johnston, M.D. - June 19, 1953

E. Eight chest radiographs dated

Oct. 15, 1948	Oct. 3, 1952
June 6, 1949	Oct. 6, 1952
Sept. 19, 1950	Oct. 27, 1952
Dec. 17, 1951	Oct. 27, 1952

F. Histological preparations: 2 slides of lung tissue furnished by Doctor Johnston and marked A-33-6-53.

G. Letter by R.L. Olson - Oct. 18, 1956.

II. OCCUPATIONAL HISTORY

A. Non-dusty:	1918 - Oct. 1943 -	25 yrs.	
	May 1946 - Feb. 1948 -	1 yr.	10 mos.
Employed -	July 1948 - Oct. 1948		3 mos.
Unemployed -	(Jan. 1949 - June 1949 -		5 mos.
	(Oct. 1949 - Aug. 1950 -		10 mos.
	(Oct. 1952 - June 1953 -		6 mos.
	Total	29 yrs.	

(continued on page 2)

II. OCCUPATIONAL HISTORY (cont'd.)

B. Dusty (Asbestos Exposure)

* 1.	Asbestos mechanic - Oct. 1943 - Dec. 1945 - E.J. Bartells Insulating Co. Shipyards, Vancouver, Wash.	2 yrs.	2 mos.
2.	Asbestos mechanic - Dec. 1945 - May 1946 - Northwest Insulating Co. Shipyards, Vancouver, Wash.		5 mos.
3.	Asbestos mechanic - Feb. 1948 - Apr. 1948 - V.S. Jenkins Insulating Co. N. Richland, Wash.		2 mos.
4.	Asbestos mechanic E.J. Bartells Insulating Co. - Oct. 1948 - Jan. 1949 - N. Richland, Wash. - June 1949 - Oct. 1949 -		3 mos. 4 mos.
5.	Asbestos mechanic - Aug. 1950 - Sept. 1950 - Chas. R. Brower and Co. Seattle, Wash.		1 mo.
** 6.	Asbestos mechanic - Sept. 1950 - Oct. 1952 - V.S. Jenkins Co. N. Richland, Wash.	2 yrs.	1 mo.
	Total	5 yrs.	6 mos.

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* Stated to have worked in excessively dusty environment in confined spaces (ships' holds) and to have worked many hours overtime.

** As determined by H. E. Perry the potential exposure of the deceased may have been to a dust composed of 85% magnesia and 15% amosite with an average concentration in the work room (doors closed) of 4.2 million particles per cubic foot. At the power saw (which operated intermittently only) dust concentration could have been 21 mppcf and 10 ft. away concentrations of 8.2 mppcf were determined. Of these particles 99% may have been less than 10μ, 1% from 10μ to 20μ, and 1% greater than 20μ.

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V. RADIOGRAPHS (cont'd.)

#3 - dated June 6, 1949: Postero-anterior photofluorograph (72 mm). The appearances are similar to the preceding film but more clearly portrayed as this is a technically superior film. Some linear and reticular shadowing can now also be discerned over the basal lobes.

#7 - dated Sept. 19, 1950: Postero-anterior photofluorograph (72 mm). No material change noted.

#51382 - dated Dec. 17, 1951: Postero-anterior teleradiograph. This film confirms the presence of an extensive area of bullous emphysema or cystic degeneration involving the greater part of the right upper lobe which is considerably enlarged. Pulmonary markings are relatively defective in the upper lobe region of the left lung as well, indicating the presence either of impaired pulmonary blood vessels or some emphysema; both basal lobe regions are the seat of diffuse hazy or ground glass opacification with a superimposed fine micronodulation. There are horizontal linear shadows as well. The cardiac outline has become irregular towards the lung apex (so-called shaggy heart). The right pulmonary arterial branches appear to be distended and the conus pulmonalis is somewhat prominent.

#14945 - dated Oct. 3, 1952: Postero-anterior teleradiograph. There has been a marked quantitative advance in the disease process. The expanded right upper lobe has been virtually replaced by a honeycombed state and marked emphysema is also apparent in the left upper lobe, vascular markings being almost indistinguishable along the outer third of the lung. Both basal lobes are contracted and show diffuse ground glass opacification with superimposed punctate shadows and reticulation. There is some pleural thickening at both bases, apparent along the outline of the thoracic cage, over the diaphragm and between the lungs and the heart. The heart is enlarged and has assumed the classical configuration associated with cor pulmonale, the conus pulmonalis being markedly distended. The proximal portions of the pulmonary blood vessels are dilated on the right.

#14969 - dated Oct. 6, 1952: Postero-anterior teleradiograph. This film is of lesser quality and reveals no features additional to those demonstrable on October 3, 1952.

#10 - dated Oct. 27, 1952: Postero-anterior teleradiograph. This film is somewhat overexposed which enables better visualization of the parenchymal changes in the basal regions. These are now seen to consist of a complex coarse system of superimposed linear shadows which give a dense spongy impression. Basically the chest condition appears to be the same as on Oct. 3, 1952.

#10 - dated Oct. 27, 1952: Lordotic view. No new features are revealed by this view but the distension of the conus pulmonalis and the avascular emphysematous condition of both upper lobes is well brought to light.

Impression: This series of radiographs is compatible with a diagnosis of pulmonary fibrosis, probably asbestosis with associated emphysema and cor pulmonale. The fibrosis and emphysema were already detectable on the first miniature film dated Oct. 15, 1942, and the subsequent changes seen are semi-quantitative.

Dr. G. W. H. Schepers of Saranac Laboratory, Medico-Legal Opinion on W. E. Taylor (1956)
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VII. DISCUSSION (cont'd.)

From the fact that the deceased evidently died from congestive cardiac failure coupled with the fact that such a large proportion of the pulmonary blood vessels and alveolar capillaries have been stenosed or occluded by the asbestotic tissue reaction, together with the observation that loss of blood vessel markings were apparent in the x-ray films at an early stage, it may be inferred that the asbestosis was a material factor in the cause of disability and death. The finding of an anatomically patent foramen ovale may have been a factor in the cyanosis which was such a prominent feature at an early stage. This foramen was, however, physiologically patent. It is not stated whether this meant that it held in check the flow of blood from the left to the right auricle or vice versa. In the cor pulmonale associated with asbestosis the tension in the right auricle may exceed that in the left so that cyanosis may have been brought about by passage of venous blood from the right auricle to the left.

The asbestosis present in this case is rather patently a process of long standing. This inference is borne out by the fact that the disease process was already well established at the time of the first radiographic examination on Oct. 15, 1948. The discovery of two varieties of asbestos bodies also suggests that the deceased was subjected during a primary or earlier noxious exposure to a fine fiber dust and a secondary or later exposure to a relatively coarse variety of amosite dust. The long fibers inhaled during the later experience were too coarse to become embedded in the pulmonary parenchyma, but were detained instead within the alveolar spaces previously distended and distorted by the asbestotic fibrosis resulting from the initial exposure.

It should be noted that the deceased was employed in two predominant phases, namely, 2 years and 7 months (between the years 1943 to 1946) and a period of 2 years and 1 month (during the period 1950 to 1952) with intermittent employment totalling 10 months during the period 1948 to 1950. As the asbestosis was already radiologically apparent on Oct. 15, 1948, it is clear that the main disease must have resulted from the earlier exposure. It should further be pointed out that in asbestosis there almost invariably is a latent period of several years between the phase of effective exposure to the asbestos dust and the commencement of symptoms and radiological signs. In the present instance it is reasonably certain that the exertional dyspnea reported during 1950 was related to the earlier, adequate exposure and that the ultimate fatal outcome could have resulted from the earlier period of exposure whether or not the deceased was exposed further to asbestos dust during subsequent years.

GWS:mam

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Review of material in the case of one W. E. Taylor,

deceased, Saranac Laboratory Number P-56-586,

OCD 75. for opinion and report $75.00

