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: Olsen and Olsen
114 North 5th Avenue
P.O. Box 636
Pasco, Wash.

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 -		8 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. B. Perry the potential exposure of the deceased may have been to a dust composed of 85% magnesia and 15% ansbite 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 90% may have been less than 10μ, 1% from 10μ to 20μ, and 1% greater than 20μ.

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 visualisation 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

VI. HISTOPATHOLOGY

The two histological sections submitted by Doctor Johnston reveal the following features.

There is extensive thickening of alveolar walls, mainly due to collage deposition. This process has involved the capillaries which are obliterate in most areas. The majority of the alveolar spaces are distended and disto Others are atelectatic and filled with cells, cellular debris and asbestos

Fibrous tissue deposition around small blood vessels and bronchioles is present to a prominent degree. Some of the smaller blood vessels have becom occluded by thrombus formation. In others there is stenosis apparently as a result of the perivascular fibrosis. The muscular coats of most blood vesse are thickened. A few veins appear to be distended and distorted. The muscul of one medium-sized blood vessel included in one of the sections is irregular hypertrophied and there is local proliferation of the intima at a few sites.

Some of the terminal bronchioles are markedly distended and resemble bul emphysematous spaces. There is also some distension of one small bronchus. other areas the bronchioles are narrowed through external constriction. The mucosa is somewhat hypertrophic in the small bronchi with predominance of goblet cells.

In one area there is a mixture of acute and subacute pneumonia, a predominance of polymorphonuclear leucocytes and of macrophages with interspersed plas cells and multinucleated giant cells respectively characterizing the two proce ses. In some areas this pneumonic process has undergone partial organization with deposition of collagen strands and formation of capillary plexuses among the cells and fibrous bundles.

Asbestos bodies of two varieties are present in large numbers. The most conspicuous are relatively large, coarse, dumbbell-type bodies measuring up to 50 microns in length. Their shanks are highly crenated. All these bodies are stained a dark-brown color and are only seen within alveolar spaces, mostly those which are partially collapsed. At no point is one of these bodies seen to be embedded in the fibrous tissue in either the thickened alveolar walls or in the perivascular and peribronchial zones. These asbestos bodies are often accompanied by large koniophores, which are filled with dark-brown particles. The impression is gained that these large asbestos bodies have been secondarily trapped in distorted narrowed alveoli.

The second variety of asbestos body is very slender and elusively short although a few long fibers were also found. These bodies are much more numerous and are found embedded in the fibrous tissue and among the cells in the alveolar walls and around the blood vessels and bronchioles. They tend to be fusiform in shape and their sheaths stain a much lighter shade of brown. The indications appear to be that these are the asbestos fibers etiologically associated with the fibrous process in this case. The larger of these types of asbestos bodies resemble those seen in amosite asbestosis. The smaller bodies cannot be too conclusively identified, but in chrysotile asbestosis the component minute fibrils produce structures of this kind.

VII. DISCUSSION

There can be no question about the diagnosis of asbestosis as the primary disease process in this case. Not only is there an adequate history of exposure to asbestos-containing dust, but the radiographic
changes, the clinical course and the
typical of

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 th asbestotic tissue reaction, together with the observation that loss of bloo vessel markings were apparent in the x-ray films at an early stage, it may b inferred that the asbestosis was a material factor in the cause of disabilit and death. The finding of an anatomically patent foramen ovale may have bee a factor in the cyanosis which was such a prominent feature at an early stag 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 righ 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 examinatio 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

GWS, 11/5/56

The Saranac Laboratory
Attn: Dr. G. W. H. Schepers
Re: W. E. Taylor
Page 2

We represent the last employer who employed W. E. Taylor prior to his death. Subsequent to his death his widow has applied for the widow's pension through the Washington State Board of Industrial Insurance. The Department of Industrial Insurance has found that the decedent had asbestosis which was a material contributing factor of the cause of his death and have held that our client, the last employer, is liable for payment of the widow's pension.

Our concern is twofold, the first being, did the defendant contract asbestosis while working for our client, or at least was it increased to the point where his employment with our client could be held to have contributed to it sufficiently to be held liable, and secondly, the question as to whether our client would be entitled to contribution from previous employers of the decedent.

We have enclosed the entire work record of Mr. Taylor but basically the facts thus far discovered would indicate that he was not engaged in any "dusty" occupation prior to 1943 at which time he went to work in the shipyards at Vancouver, Washington as an asbestos worker. The record would show that from October of 1943 to May of 1946 Mr. Taylor worked in the asbestos industry at Vancouver, Washington. Testimony of co-workers at that time indicated that Mr. Taylor was working in the holds of ships and boiler rooms of ships that were being built at the shipyards there. Testimony is to the effect that the conditions were extremely dusty and there was poor ventilation, that the workers worked six to seven days a week and worked from 8 to 12 hours a day. This having been in the war time years, there was a considerable amount of overtime work available and performed by the decedent and others in his craft.

From May of 1946 to approximately February of 1948 Mr. Taylor was not engaged in a place where he had exposure to asbestos fibers. From February of 1948 to April of 1948 he was again employed in the asbestos field.

His first employment with our client occurred in October of 1948 at which time he worked approximately 3½ months for our employer. He subsequently was employed from June of 1949 to October of 1949 and subsequently again worked from September, 1950 to October 27, 1952 for our client, all of which work for our client was done in the asbestos field.

client, but should you need any further information, please feel free to either write me at the above address, or call me collect at the number stated on the letterhead.

Insofar as we have been given a limited time in which to contact you and secure a report, we would appreciate your prompt attention to this matter if at all possible..

We must, of course, have all of the items which we have enclosed herein returned to us as most of them have either been admitted into the court records or will be admitted in the immediate future.

Very truly yours,

OLSON & OLSON

By:

RLO:HD
Enc. a/s

Registered mail -
return receipt requested

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(continued on page 2)

Re: W.E. Taylor
COD 75 - P-56-686
November 5, 1956

III. CLINICAL HISTORY

(Olsen and Olsen)

A. October 1950 - Consulted doctor in Vancouver for intermittent shortness of breath.

B. Spring 1951 - Exertional dyspnea with cyanosis.

C. 1952-1953 - Progressive dyspnea and cyanosis.

D. Died June 1953.

IV. GROSS AUTOPSY FINDINGS

(Autopsy Protocol by Robert D. Johnston, M.D.)

A. Pulmonary fibrosis: bilateral, interstitial, asbestotic, anthracotic

B. Pneumonitis: acute, subacute, chronic, lower lobes

C. Pleuritis: bilateral, acute, fibrous, pleuropericardial adhesions

D. Bronchiectasis: early

E. Lymphadenitis: hyperplastic, mediastinal, anthracotic

F. Pulmonary emphysema: marked: upper lobes

G. Cor pulmonale: hypertrophy right ventricle 9-10 mm, ante mortem thrombosis of right auricular appendage

H. Passive congestion: acute and chronic (lungs and liver), acute (spleen and kidneys)

I. Emboli: pulmonary; left kidney

J. Anatomically patent, physiologically closed foramen ovale

K. Weight of right lung - approximately 1200 grams
" " left " - " 1600 grams

V. RADIOGRAPHS

#29 - dated Oct. 15, 1948: Postero-anterior photofluorograph (72 mm) - slightly out of focus. The following diagnostic features are evident in this film: marked transradiancy of the upper half of the right lung field with downward displacement of the interlobar fissure. The appearance suggests a large pulmonary cyst or emphysematous bulla. The left upper zone is also somewhat transradiant. There is partial "ground glass" opacification of both

CAVILLE B. OLSON ROGER L. OLSON

LAW OFFICES OF
OLSON AND OLSON
114 NORTH 4TH AVENUE
P. O. BOX 866
PASCO, WASHINGTON
PHONE LI-7-7597

October 16, 1956

The Saranac Laboratory
P. O. Box 551
Saranac Lake, New York

Attn: Dr. G. W. H. Schepers

Re: W. E. Taylor, deceased

Dear Sir:

Pursuant to your letter of July 13, 1956 to us and our telephone conversation with you of September 12, 1956, we have finally got together most of either the original artibles presented in evidence or copies thereof and would like to request your assistance in giving us your opinion pertaining to the problem of the claim for death on behalf of W. E. Taylor who allegedly died from asbestosis as having been a material contributing cause.

I am enclosing herein the following items for your perusal:

1. Asbestos Identification
2. Work record of decedent
3. Autopsy report on decedent
4. A study of the dust exposure as set up in a test conducted at Richland, Washington
5. Three X-Rays admitted in evidence being dated 10-3-52, 10-6-52 and 12-17-51.

In addition to which we are enclosing five X-Rays not yet admitted in evidence but which are dated 10-15-48, 6-6-49, 9-19-50, 10-27-52, 10-27-52.

We are also writing this date to Dr. Robert D. Johnston of 11520 S.E. Maxon Road, Vancouver, Washington, requesting that he immediately forward to you the slides prepared by him at the time of autopsy. He has, however, indicated that he would send the slides only upon request of a doctor so even though we have written to him, we would appreciate it if you would forthwith write to him and request the slides and tissue blocks from him.

It has been shown that as early as October, 1950 that Mr. Taylor had seen a doctor in Vancouver, Washington at which time he had advised his doctor that he had been short of breath off and on.

While working in the employ of our client as early as the spring of 1951 it was discovered that Mr. Taylor had difficulty while working in any place where he had any physical exertion because it would cause him such a shortness of breath and his condition would turn cyanotic. He was, therefore, placed in what is known as the cutting room and was engaged part of the time with power saws and hand saws in cutting and preparing the asbestos product for fitting. The asbestos used was commonly termed as 85% magnesia with a 15% asbestos binder. Much of the time that he was working in the cutting room, however, he was engaged solely in the cutting of a form of paper used for a covering for the asbestos and also cutting metal bands. We attempted to set up a test in a cutting room as comparable to the actual workshop as we could. In fact, even the doors and windows were kept closed at the time of the test in order to give a maximum concentration as far as possible. We have enclosed the results of that test in this letter.

Further testimony would indicate that some lay people felt that he was in fairly good health until as early as 1952 at which time it became progressively more difficult for him to breathe and his cyanotic worsened. He was actually discharged because of a force reduction in October of 1952 and died in June of 1953.

We have also had tested a sample of the asbestos that was being used and have enclosed a copy of the report indicating that anthophyllite was being used. However, we do not have an accurate history of whether the anthophyllite was the only type that was used or not at this time. However, in our telephone conversation you indicated that you could determine from the slides the type of asbestos that would have accumulated in the asbestosis bodies. We would like to have this information as well.

This, of course, has been a condensation of a great many witnesses and may not be sufficiently elaborate to aid you in your conclusions, particularly as to whether the decedent may have had asbestosis at the time that he came to work for our

SARANAC LABORATORY
TRUDEAU SCHOOL

November 6, 1956

Olson and Olson
114 North 5th Avenue
P.O. Box 888
Pasco, Washington

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

REGISTERED NO. 00000

Spec. del'y fee $

Fee $ 40 Ret. receipt fee $

Surcharge $ Rest. del'y fee $

Postage $ 7½ ☐ Airmail

NOV 6 1956 N. Y.

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From

To

POD Form 3800
May 1954