OWENS-ILLINOIS
LIBBEY GLASS PLANT
ASH STREET (11) TOLEDO, OHIO

July 17, 1956

Dr. G. W. H. Schepers, M.D., D.Sc., Director
The Saranac Laboratory
7 Church Street
Saranac Lake, New York

P-56-682

Dear Doctor:

In accordance with directions of Dr. Charles F. Shook, Medical Director of Owens-Illinois Glass Co., I am sending under separate cover the autopsy specimen, Lungs, of Lee Bryston an employee of the Libbey Glass Division of Owens-Illinois who died July 8, 1956 and whose death certificate states he had Silicosis and Tuberculosis. A form furnished by your Laboratory has been filled out and is enclosed. — Rec'd 7/20/56

The autopsy protocol has not yet been received from the pathologist, but will be forwarded as soon as received.

Should additional information be requested we will attempt to obtain it for you.

Respectfully,

K. H. Howard

K.H. Howard, M.D., Plant Phys.

PLAINTIFF'S EXHIBIT
Kirstein-NY
8-28-98

55911

OWENS-ILLINOIS

GENERAL OFFICES (1) TOLEDO 1, OHIO

September 11, 1956
Dict. 9/10/56

Dr. G. W. H. Schepers
The Saranac Laboratory
Saranac Lake, New York

Dear Dr. Schepers:

Since Dr. Shook is out of town for a few days, we are acknowledging the report you sent last week on Mr. Lee Bryston.

Certainly the finding of unusual amounts of trydimite and crystobolite on chemical analysis of the lungs is most unexpected. We have rechecked with the plant and thus far have no clue as to where he might have been exposed to such dusts.

Around 1927 he worked for a few years as a punch press operator for Willys Overland, then he was unemployed two years, following which he came to work at our Libbey plant--and never worked any place else. During the first year or two of his Libbey employment he was in the general labor group as a clean-up man. In this capacity he did spend a few hours off and on in the batch department, but he never was regularly employed there. For the last 20 or more years he had been a guard and watchman and, so far as we know, never visited the batch department at all.

His brother states that during his two years of unemployment before he came to Libbey, Mr. Bryston was really unemployed and, consequently, didn't have any kind of industrial dust exposure whatsoever.

As for X-rays, the plant has no record that any were ever taken. We have even checked with the local health department, which in recent years has used a mobile X-ray unit for TB case finding. They have no record of ever having X-rayed Mr. Bryston. It's unfortunate, but no X-rays can be found anywhere.

Naturally we are looking forward to seeing your final report. While there is no immediate urgency for this, we would like to see it as soon as convenient so we can reach some conclusion on this case.

Sincerely yours,

W. G. Hazard

W. G. Hazard - Industrial Relations Division
M

PLAINTIFF'S EXHIBIT
Kirstein-153
8-28-96

TMD

THE SARANAC LABORATORY
For the Study of Tuberculosis, Occupational Disease, and Industrial Hygiene
Founded by EDWARD LIVINGSTON TRUDEAU in 1885
7 CHURCH ST., SARANAC LAKE, NEW YORK

Charles F. Shook, M.D. September 4, 1956

P. O. Box 861 Re: Lee Bryston
Reference: Saranac Laboratory P-56-682
OCD 68

Telephone: Laboratory 613
Director 580

The amounts of tridymite (0.6 - 1.7%) and cristobalite (0.4 - 1.2%) found in the lung ash are very much in excess of what we have previously found. Perhaps these forms of free silica explain the pulmonary pathology. We know that cristobalite and tridymite are many times more potent than quartz as fibrogenic agents.

The occupational history you furnished us over the telephone does not help to explain this case. He probably worked in a ceramic products plant at one time or another in his life to account for the presence of the excessive quantities of high temperature free silica found in his lungs. Could this matter be looked into again, or may we assume that he could have been exposed to these substances while performing his duties as a plant guard?

I will set all this up in a formal report when I return shortly from an out-of-town trip. We do hope you will let us have a look at the X-rays of this most interesting case.

With kind regards,

Yours sincerely,

G. W. H. Schepers, M.D., D.Sc.
Director

GWHS:LB

55868

The objectives of The Saranac Laboratory, a non-profit organization incorporated under the Education Law of New York State, are: Research, Consultation, and Education

Wed. Jan 18, 1956 2:30 p.m.

Telephone call from Dr. Shook of Owens-Illinois, Toledo. (and Bill Hazard).

They are sending us (today) the lungs of one of their watchmen who died. This may be a medico-legal case for silicosis but Dr. Shook doesn't believe the man had silicosis - he had no exposure to silica dust.

The data about the occupational exposure, etc., will be forwarded to us from the Libby plant
Owens Illinois
Toledo

and our report should be sent there.

However, send copies of the report and all pertinent correspondence to Dr. Shook also.

PLAINTIFF'S EXHIBIT Kirstein-MB 8-28-98

Dr. Shook wished to tell Dr. Bristol that he (Dr. Shook) doesn't like the new forms.

Also, films of Lee Bryston will be coming along soon. Would like to get reading as soon as possible - please push it through. Question of silicosis? T.B.?

IV. DISCUSSION (cont'd.)

confirms the fact that the disease is dominantly of industrial origin. This does not yet dispose of the possibility that the deceased may have been one of those unusual individuals who retain inhaled foreign matter in unusual quantities and react excessively to them. The marked involvement of hilar root glands further suggests the possibility that early exaggerated reaction in these structures may have obstructed the egress of trapped siliceous dust both through blockage of lymphatics and stenosis of bronchi. Such an explanation accounts for the development of massive lesions in atelectatic lung lobes in rare cases of silicosis. There may also have been an infective factor which involved these hilar lymph glands at an early stage. No trace of such infection persisted.

V. DIAGNOSIS

The disease process found in the lungs of the deceased is an aberrant form of silicosis characterized by diffuse mural sclerosis with associated hypertrophic emphysema and focal conglomerate nodular fibrosis with associated regional pulmonary atelectasis, the etiological agents being tridymite, cristobalite, quartz, and mullite.

GWMS:mmm

Nov 5, 1956

55879