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MEDICINE AT ITS BEST

November 6, 2007

John P. Comerford, Esq.
Lipsitz & Ponterio
135 Delaware Avenue, 5th Floor
Buffalo, New York 14202

FAX (716) 849-0708

RE: James Girton
JA07-295

Dear Mr. Comerford:

I have reviewed the records and pathology materials you sent related to Mr. Girton. According to the information provided Mr. Girton had exposure to asbestos from automotive brake and clutch materials. He was exposed to dust when brake linings were either hand sanded or resurfaced with an electric bench grinder. From 1954 to 1972 he worked at several car dealerships in New York and continued to do work at home on his own vehicles and for others from 1972 to 1979. He was around other mechanics as well when they did brake maintenance and removal.

The pathology materials I received correspond to the pathology from Lourdes Hospital (S07-5671) from Mr. Girton's right pleural and lung biopsies on June 11, 2007. The biopsy shows an invasive malignant tumor of the pleura diagnostic for malignant mesothelioma based on the immunohistochemical stains reported and provided for my review. These show the tumor cells positive for calretinin and CK5/6 and negative for BerEP4 and CEA. There is lung parenchyma contained within the lung biopsy and iron stained section revealed no asbestos bodies. The mesothelioma in the available biopsy sampling appears to be biphasic but predominantly epithelial. The diagnosis of mesothelioma was also confirmed by review at the Brigham & Women's Hospital.

To ascertain the lung burden of asbestos bodies and/or fibers, portions of the lung tissue from the paraffin blocks were digested using our standard sodium hypochlorite digestion, followed by collection of the residue on polycarbonate membrane filters for counting asbestos bodies by light microscopy or examining fibers using electron microscopy. There was insufficient tissue for determining the dry weight of the lung tissue.

The first analysis, by light microscopy, searched for asbestos bodies on a filter with a detection limit of 35 asbestos bodies per gram of wet lung tissue. No asbestos bodies were found in this analysis by light microscopy.

The first electron microscopic analysis used tissue from block A1 and analyzed all fibers at least 3 micrometers in length at a viewing magnification of 8,000 times in the electron microscope. In this analysis the detection limit was 18,700 fibers per gram wet lung. The types of asbestos fibers found included one tremolite fiber and one probable chrysotile fiber [undetectable magnesium], each representing 18,700 fibers per gram wet lung. The tremolite fiber was 9.8 micrometers by 1.6 micrometers and the probable chrysotile fiber was 16.9 by 0.16 micrometers. In addition to these fibers there were 3 fibers of probable talc detected ranging in length from 7.3 to 25.7 micrometers. No commercial amphibole fibers were detected in this analysis.

The second electron microscopic analysis analyzed fibers from block B1 at a viewing magnification of 4,000 times in the electron microscope. This analysis had a detection limit of 1,900 fibers per gram wet lung. In this analysis the concentration of asbestos was determined to be 38,700 fibers per gram wet lung. The types of asbestos fibers included chrysotile with partial depletion of magnesium at 21,000 fibers per gram wet lung, tremolite at 1,900 fibers per gram wet lung and additional probable chrysotile with complete depletion of magnesium at 15,500 fibers per gram dry lung. The length of the chrysotile fibers ranged from 4.9 to 61 micrometers; 10 of the 11 chrysotile fibers found were greater than 5 micrometers in length. The one tremolite fiber was 12.4 micrometers in length. The additional probable asbestos fibers ranged from 5.1 to 30.4 micrometers. In addition to the chrysotile and tremolite fibers 4 fibers of talc were found ranging from 5.3 to 29.9 micrometers in length. No commercial amphibole fibers were detected in this analysis.

In summary these lung fiber burden analyses confirm the absence of detectable commercial amphibole fibers within the detection limits of these analysis. The background range for commercial amphibole (amosite and/or crocidolite) would be up to 1,000 fibers per gram wet lung tissue. The background range for chrysotile fibers greater than 5 micrometers in length would be up to approximately 5,000 fibers per gram wet lung, and the concentrations of fibers greater than 10 micrometers in length for chrysotile in the general background population would be near 0. Therefore these findings confirm an elevated burden of chrysotile and related amphibole fibers in Mr. Girton's lung. This is consistent with his occupational history and independently determined from the history.

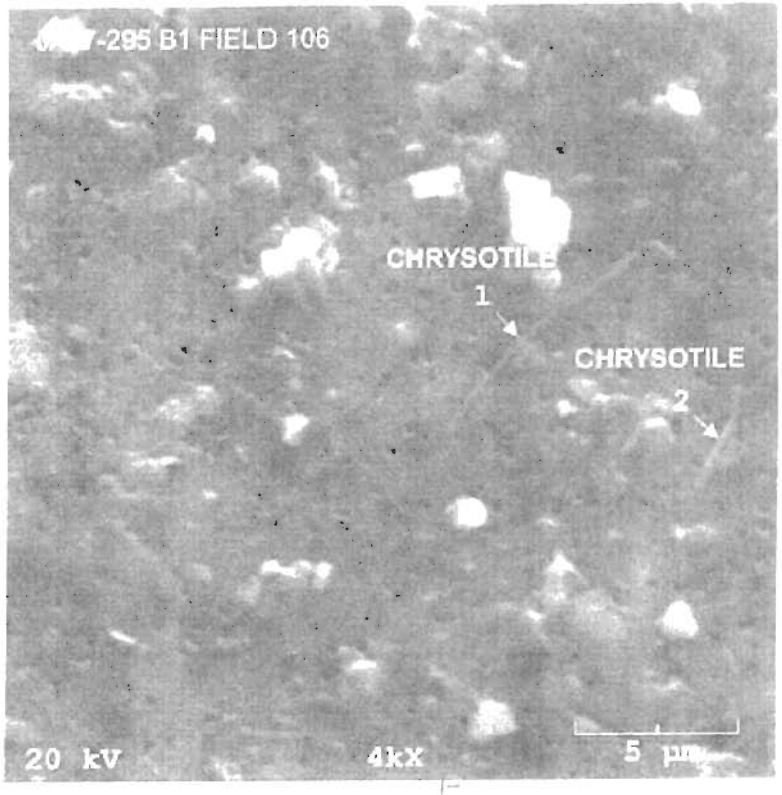
Asbestos exposure is well recognized to be the cause of nearly all malignant mesotheliomas. Mr. Girton had a history of asbestos exposure and developed a malignant mesothelioma. Therefore I can conclude to a reasonable degree of medical certainty that Mr. Girton's asbestos exposure was the cause of his malignant mesothelioma and will likely be the cause of his death.

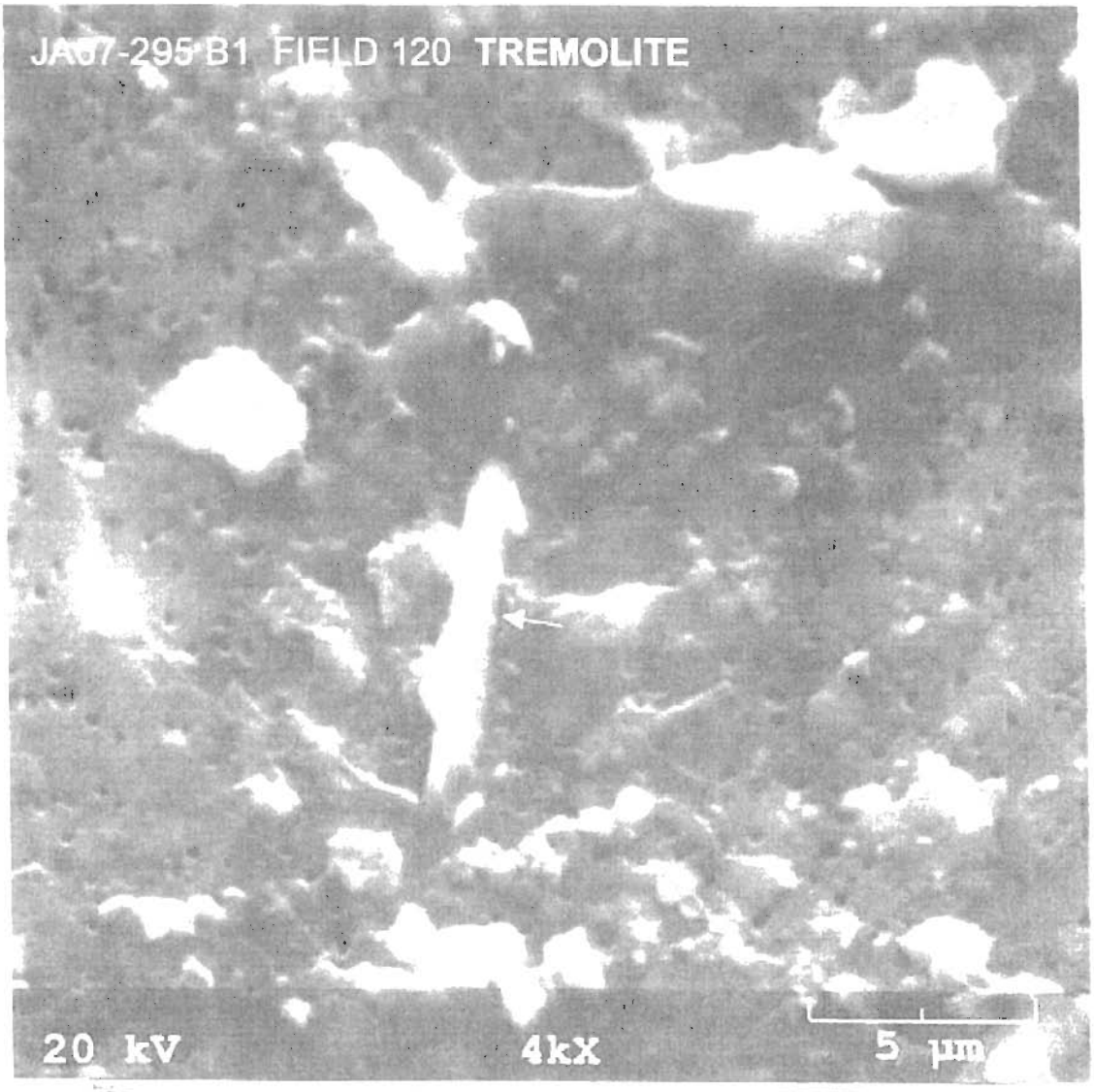
Please let me know if you need additional information

Sincerely,

Jerrold L. Abraham, M.D.
Professor of Pathology and
Director of Environmental and
Occupational Pathology

P.S. The pathology materials are being returned under separate cover.
JLA/hjg


-295 B1 FIELD 106
CHRYSOTILE
1
CHRYSOTILE
2
20 kV
4kX
5 µm


JA07-295 B1 FIELD 120 TREMOLITE
20 kV
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MEDICINE AT ITS BEST

June 22, 2006

John Guinan, Esq.
Levy, Phillips, and Konigsberg
800 Third Ave.
New York, NY 10022

Fax (212)605-6290

Re: Bennett Scott Hoser
JA06-151

Dear Mr. Guinan:

As requested I have reviewed the records and pathology I received related to Mr. Hoser. My understanding from the information provided is that Mr. Hoser was diagnosed with mesothelioma at age 45 in October 2005. He had a radical pneumonectomy at Sloan Kettering in March 2006. His work history provided indicates he grew up on a dairy farm in New Jersey where he worked on various tractors. From at least 1975-1979 Mr. Hoser would personally sand the brake discs for the tractors (International Farmall Super MTA Tractors). He was also present as a young man when his father took a bench grinder to the brake discs, which created considerable airborne dust. Mr. Hoser also had exposure at Warren County Vocational Technical School in Broadway, NJ, in an auto mechanics class from 1977-1979, in which he worked on brakes, clutches, and gaskets. In 1978 he worked at Louie's Garage in Bloomsbury, NJ, and was present when multiple brake jobs were performed. He also worked at a Ford tractor dealership in Washington, NJ from February-July 1979, and worked on brakes, clutches, and gaskets on Ford tractors. Occasionally Mr. Hoser also performed brake jobs at his home. During the 1980's Mr. Hoser worked as a correctional officer in New Jersey and serviced numerous international harvester tractors at the site, with further exposure to asbestos-containing brakes and clutches. His testimony recorded March 3, 2006 goes into more detail on his work history.

The pathology materials I received correspond to the surgical pathology report S06-8961 from Memorial Hospital in New York. The sections of lung show some evidence of talc pleurodesis with foreign body reaction in sections 12 and 18. Section 21 from the right lower lobe and 20 from the right middle lobe show lung and 19 from the right upper lobe shows lung with tumor. The tumor was confirmed to be an epithelial malignant mesothelioma of the pleura, and there was also involvement of the peritoneum.

Portions of block 21 and 19 were digested using our standard sodium hypochlorite technique, with collection of the residue on nuclepore filters for counting of asbestos bodies by light microscopy and analysis of fibers using electron microscopy.

The first analysis, by light microscopy, used tissue from block 21 and had a detection limit of 181 asbestos bodies per gram dry lung or 54 asbestos bodies per gram wet lung. No asbestos bodies were detected in this analysis.

The first electron microscopic analysis used tissue from block 19 and analyzed all fibers at least 3 micrometers in length at a viewing magnification of 8000x in the electron microscope. In this analysis the detection limit was 20,000 fibers per gram wet lung or 93,000 fibers per gram dry lung. In this analysis the total concentration of asbestos was determined to be 1,213,000 fibers per gram dry lung (f/g-d). The predominant type of asbestos found was chrysotile at 466,000 fibers per gram dry lung followed by actinolite at 187,000 f/g-d and additional probable chrysotile from which magnesium had been completely depleted at 560,000 f/g-d. All of these chrysotile and probable chrysotile fibers were quite long, ranging in length from 5.1 up to 55.5 um and in diameter from 0.09 up to 0.23 um. The actinolite fibers were 2.6 and 6.0 um in length. This is certainly documentation of unusual chrysotile exposure, since the background concentration for such long chrysotile fibers would be extremely low in the general population, as discussed below.

The second electron microscopic analysis used tissue from block 19 and analyzed all fibers at least 3 um in length at a lower magnification (4000x on the viewing screen of the electron microscope). In this analysis the detection limit was 52,000 f/g-d. A total concentration of asbestos fibers at 261,000 f/g-d was noted. In this analysis all of the fibers detected were either magnesium depleted chrysotile (104,000) or probable chrysotile with no detectable magnesium, ranging in length from 8.3 to 20.0 um and in diameter from 0.14 up to 0.28 um. No amphibole asbestos fibers were detected in this analysis.

The third electron microscopic analysis used tissue from block 21 and analyzed all fibers at least 3 um in length at a magnification of 8000x on the viewing screen of the electron microscope. In this analysis the detection limit was 108,000 f/g-d. No asbestos fibers were detected in this analysis.

The fourth electron microscopic analysis used tissue from block 21 and analyzed all fibers at least 3 um in length at a viewing magnification of 8000x on the viewing screen of the electron microscope. In this analysis the detection limit was 80,600 f/g-d. Asbestos fibers were detected at a concentration of 484,000 f/g-d. Chrysotile asbestos (partially magnesium depleted) and probable chrysotile asbestos (with no detectable magnesium) were detected at concentrations of 161,000 fibers per gram lung and 242,000 f/g-d, respectively. One actinolite fiber was detected representing a concentration of 80,600 f/g-d. The chrysotile fibers ranged in length from 6.9 to 22.6 um with diameters ranging from 0.14 to 0.18 um. The actinolite fiber was 12.3 um in length with a diameter of 0.8 um.

The last electron microscopic analysis used tissue from block 21 and analyzed fibers at least 3 um in length at a magnification of 4000x on the viewing screen of the electron microscope, searching specifically for fibers present at lower concentration than the detection limits of the other analyses. In this analysis the detection limit was 11,000 f/g-d. In this analysis chrysotile fibers were found at 45,000 f/g-d, ranging in length from 6.1 to 61.1 um and in diameter from 0.13 to 0.22 um. Actinolite fibers were found representing 22,000 f/g-d ranging in length from 4.0 to 10.8 um and in diameter from 0.47 to 0.63 um.

These findings are certainly consistent with Mr. Hoser's history of exposure predominantly to friction materials containing chrysotile asbestos fibers. There is no evidence of any commercial amphiboles in any of the analyses of his lung tissues. In the general background population, 95% of chrysotile fibers are shorter than 5 um, and calculations of the upper limits for chrysotile fibers longer than 5 um would result in a limit of approximately 50,000 f/g-d. For chrysotile fibers as long as most of those seen in Mr. Hoser's lung tissue, the upper limit of background would be much much lower than 50,000 f/g-d. Mr. Hoser's lung tissue contains greatly elevated concentrations of long chrysotile fibers.

These findings allow me to conclude to a reasonable degree of medical certainty that Mr. Hoser's asbestos exposure was the cause of his malignant mesothelioma and will likely be the cause of his death.

Please let me know if you need additional information.

Sincerely,

Jerold L. Abraham, M.D.
Professor of Pathology and
Director of Environmental and
Occupational Pathology

p.s. The pathology materials are being returned under separate cover.
JLA/lbp

JA06-151 HOSER
SAMPLE C2
4000X ANALYSIS

FIELD 77