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July 12, 1983

Mr. Don Gholeson
T.E.I.A.
P.O. Box 1109
Galveston, Texas 77550

RECEIVED

AUG 2 1983

MONSANTO CO.
LOSS PREVENTION DEPT.

Re: Donald Garretson
DOB: 4/16/22

Dear Sir:

Mr. Garretson was seen in my office for an examination on July 6, 1983. The examination was to determine whether or not in my opinion Mr. Garretson was suffering from medical illness attributed to his occupation. This is a report of this examination, my review of medical records submitted by your office, x-rays taken at other locations, and the results of laboratory information gathered since being seen by me. I will summarize his history and results of my physical examination, the results of laboratory evaluations, x-rays, and pulmonary functions, obtained both prior to this visit, and since, review of the compounds that he has been exposed to in the past, and then present my opinion concerning the question at hand.

Mr. Garretson is 61 years old, and states the following: Mr. Garretson complained of shortness of breath, and that this was his primary problem at the time. He first noticed problems approximately eight years ago, when he noticed tiring more easily than he had been accustomed to previously. Four or five years ago he noted the onset of shortness of breath, which he states has progressed gradually since. Approximately eight months ago he noted severe shortness of breath while doing some demolition work which consisted of swinging a 2 x 4. Since that time he has noted that his dyspnea occurs after walking approximately 50 paces on level ground. He has had a daily cough for approximately eight months which is usually not productive. Although he has a history of tobacco abuse, he does not give a history consistent with productive cough in the morning. He has noted both pain on the left anterior chest and some difficulty in taking a deep breath. His review of systems revealed that he had had some mild joint pains in the fingers of both hands, although he had not noticed any redness of these joints. A week prior to

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the visit to my office he had noted mild swelling of his fingers. He occasionally felt discomfort in both of his shoulders. His social history included smoking approximately a pack of cigarettes a day from early adulthood until approximately a year ago. His family history was negative for pulmonary disease or rheumatoid arthritis.

Mr. Garretson provided me with the following occupational history: He states that the first type of employment he recalled was digging ditches, and doing odd jobs during the summers as a youth. He worked in a fig factory canning figs until he joined the infantry and served as an infantryman during World War II. He denies ever being exposed to toxic gases during the war. Approximately thirty-six years ago, in January, he went to work for Monsanto, and worked from then until April in a polystyrene unit where he was responsible for handling paper sacks each which was filled with 50 pounds of polystyrene pellets from an hopper. Following this he began working in an ethylene unit, where he was apparently an operator working near ethylene gas which was a by-product of petroleum cracking. He stated that this in process involved the cracking of propane to ethylene, and that on occasion he did have mild exposures to the gases. He worked on this unit for sixteen years, and then had a job change to chief operator of a polyethylene unit. In this capacity, for the next twenty years, he worked in close proximity to the following compounds: Ethylene, Hexane liquid, Methanol liquid, Butane gas, Hydrogen gas, "Disobutylaluminum Hydride liquid", Titanium Tetrachloride, and ammonia. He stated that in that time he was exposed to several spills of the Titanium Tetrachloride, and ammonia. All the spills occurred outside and never in an enclosed area. He stated that none of the exposures ever caused enough symptoms to warrant treatment by a physician, emergency team, or hospitalization. He specifically denied doing any sandblasting, or working near an area where sandblasting was being done. He denied exposure to asbestos fiber or dust, although he stated that he had seen bound asbestos insulation in his environment. He was never involved in any demolition of old insulation equipment.

The patient had a complete physical examination done in my office. It was normal with the following exceptions: The patient was noted to have clubbing of his fingernails (he states the he felt that he had had this all his life, but had had this brought to his attention by a physician in 1979, and had not noticed any changes in his fingernails since that time), he was noted to have a mild conjunctivitis in both eyes, and auscultation of his chest revealed coarse inspiratory rales in all lung fields, and fine end inspiratory rales at the lateral bases. No wheezing nor rhonchi were heard.

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On July 7, 1983 Mr. Garretson reported to the pulmonary laboratory of the University of Texas Medical Branch Hospital where a fairly comprehensive battery of pulmonary functions were done. Although I do not have the official interpretation done by that department at this time, I have received a copy of the results of these tests. The pulmonary functions demonstrated a reduction in the diffusion of carbon monoxide to 52.3% of predicted. His FEV1, MMF, vital capacity, and total lung capacity are all within normal limits. Gas exchange was done in a step-wise fashion until the patient reached a workload of 600 KPM which is equivalent to the work necessary to do farming, mining, heavy industry, or the playing of tennis, dancing, or canoeing. Mr. Garretson had a marginal arterial oxygen tension of 79.7 at rest, however his alveolar arterial gradient was 24.4, which is high. With exercise, his arterial oxygen tension dropped to a low of 55 at 600 KPM, at which time his alveolar arterial gradient was 64.3, his blood PH was 7.344, and his arterial carbon dioxide tension was 33. The gas exchange was done in a step-wise fashion, and the development of significant difficulty in his gas exchange was noted at 300 KPM, when his arterial O₂ had dropped to 62, his alveolar arterial gradient was 50, and his base excess, which is representative of the formation of lactic acid, began a significant drop. This would be when I would expect the man to begin feeling significant discomfort. This workload would be equivalent to housework, or clerical duties, the playing of golf, or bowling.

I have reviewed the pulmonary functions done over the years on this gentleman. With the exception of the pulmonary functions done on 3/21/83, the lung mechanics (represented by the vital capacity, FEV1), have been within comfortably normal limits, as they are now. There is no evidence in these previous records that more revealing studies, such as gas exchange or diffusion capacities, were done.

Antinuclear antibody, rheumatoid factor, VDRL, were all done at my office and were found to be negative, as they have been found to be by other examiners. A review of previous studies by this patient, particularly when he was hospitalized recently, revealed no significant abnormalities other than the serum protein electrophoresis which was interpreted as having a high gammaglobulin showing a polyclonal configuration (consistent with a number of disorders).

I have reviewed x-rays on this patient dating back to July 28, 1977. They show a progressive process involving the lung fields, but sparing the pleura, which is consistent with a fibrotic process. X-rays done in 1977 revealed prominent hilar nodes or vascular structures, which had the appearance of calcified nodes, with the suggestion of eggshell like calcification.

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Although this was not as apparent in 1980, I felt there was a suggestion of eggshell calcification in those x-rays as well. The process appeared to be progressive over time. Tommograms done at St. Mary's Hospital, ordered by me to clarify the issue of eggshell calcification (suggestive of silicosis) did not substantiate my suspicion.

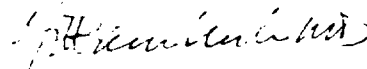
In summary, he has been exposed through his occupation to a number of compounds which he could identify, and probably a number of others that he could not. With the exception of the compound which he called "Disobutylaluminum Hydride", the effect of acute and chronic exposures to the rest of the compounds he named have been defined. Some are suspected carcinogens (polystyrene, polyethylene), and some can cause reversible irritation of the upper respiratory mucosa if inhaled in high enough concentrations. The Titanium tetrachloride, and ammonia, can both cause severe pulmonary injury if inhaled in high enough concentrations. Patients that have survived exposures of this degree have shown over the passage of years the development of scarring (fibrosis) of the lungs. The types of exposures that result in this usually result in death. The patient stated that none of his exposures even required medical attention, so they must be considered relatively mild, and not of the degree necessary to cause the type of illness he now presents with.

I believe Mr. Garretson is suffering from a debilitating, progressive, but not occupationally acquired pulmonary disorder. His occupational history fails to include exposure to compounds which are known to cause diffuse pulmonary fibrosis. Should information become available to me which suggests that there have been other compounds which he has been exposed to, such as free silica, or asbestos dust, I would have to reconsider my impression.

At this time, the true nature of the illness that Mr. Garretson is suffering from remains obscure. A lung biopsy, preferably an open lung biopsy, remains desirable. There is a chance, although slim, that he has an illness which is treatable.

I have included copies of relevant data generated by this evaluation. If I can provide you with any further information please don't hesitate to contact me.

Sincerely,


F.M. Zaunbrecher, M.D.

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