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SECTION OF PULMONARY DISEASES

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August 15, 1986

CONSULTATION SUMMARY
Record Review

FLANDERS DOBSON.
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RE: Record review in the case Flanders R. Dobson vs, Tylo Roofing /Reynolds Aluminum
Record review requested by Attorney Robert Montstream

The following is a summary of medical charts and record analysis, as well as review of available laboratory data including chest x-rays, after a preliminary discussion with Attorney Robert Montstream. This is carried out with complete medical records and a copy of the Workmen's Compensation Deposition of Flanders Robert Dobson on August 1, 1985.

OCCUPATIONAL EXPOSURES. Date of birth: 2/20/13 - Sylvania, Georgia

1925 - 1930 -	He did part time work with cleaning of offices and special delivery for the local post office, also helping his father in a funeral business.
1930 -	He moved to Florida to seek work. He was employed as a caddy for approximately 1.5 years.
1931 - 1933 -	He worked for the Ringling Brothers/Barnham and Bailey Circus in their Florida headquarters. In approximately 1933, he moved to Passaic and worked for the Federal Creosote Company approximately 2-3 years.
1935 or 1936-	He worked for the Modern Dairy and Grocery in Patterson, New Jersey, working in the warehouse and driving a truck until approximately 1943.
1943 -	The patient moved to Bridgeport and worked for the Alcoa Aluminum Co. for approximately three years, where he worked with molders, skimming molds containing magnesium and sustaining a burn during his employment.
1945, or 1946	He worked for six months at Cilco, a local lumber company, unloading lumber.
1945, or 1946	He first began work for Tylo Roofing Company, in Stratford, Conn., working in the asbestos building for approximately six months, where he placed prepared asbestos shingles on skids, stacking them and packing

them up to dry for bundling. The shingles had been freshly cut and pressed by machine with an appropriate design placed on them before drying and packing.

1946 - After six months the patient was transerred "upstairs", where he was involved in operating a machine that mixed material for the production of asbestos containing shingles, which involved mixing eight bags of marble dust, 1200 pounds of cement, and three bags of asbestos fiber into the mixer, preparing between 22-23 mixtures a day and working between 5-6 days a week. The asbestos fibers were apparently the product of two separate suppliers, one being Johnson and Johnson, that produced a long asbestos fiber with two bags used to one bag of a powdered asbestos produced by the Johns Manville Company. The patient claimed in testimony during deposition on August 1, 1985, that to the best of his recollection he worked at the mixing operation for approximately 20 years. AT that point the company ceased the operation with asbestos approximately 9-10 years before his retirement in 1978. At that point the patient was transferred to the felt mill involved in the production of asphalt shingles. During his employment in the mixing process, the patient testified that at times he wore a paper or cloth mask, and at times he did not, based on how dusty the area was at the time.

Material produced by the Tylo Company for employees is available for review but does not carry a date. It indicates that the company produces asbestos cement shingles used exclusively as siding (we do not manufacture asbestos cement roof shingles) and this is described as involving asbestos fiber and portland cement. The document goes on to describe wages, insurance, union representation, vacation policies, and other information pertinent to the employees.

Observations made by Mr. Edward Largent at the Tylo Company, Inc., Stratford, Connecticut, on January 11, 1968, are reported to the company in a report dated February 7, 1968. It is noted that the air was sampled for particulate in the raw materials feeding station on the second floor directly above the asbestos slurry tank. No other fiber measurements were made. The dust found in samples in the impinger flask liquid used to sample during the operator's cycle of 20 minutes was 1.4 million particles per cubic foot in one sample, and 5.9 million particles per cubic foot in the second sample. Air sampling was not carried out because the equipment was not in operation due to repairs at the time of the study. Noise levels and potential dust was evaluated. The report notes that it is probably fortunate that only "long fiber" chrysolite asbestos was being used at the Stratford plant. It was concluded that the dust found in samples #1 and #2 were so minimal as to seem non-hazardous as far as asbestosis was concerned. Under the microscope no fibers were seen that had the characteristic of asbestos fiber, suggesting that the dust was probably largely, in not entirely, cement or limestone, or that asbestos fibers were present but so slender that they could not be seen by standard light microscopy. The collection of additional samples was suggested to confirm the possibility of a few asbestos fibers in the work area. The higher level of dust noted in the second sample was ascribed to an activity before sampling, when a high section of scrap fell to the floor generating a heavy cloud of dust, which was considered a temporary situation. It was concluded that there should be no great concern about dustiness near the slurry tank, but additional samples were required to appraise the potential dustiness of the process.

An industrial hygiene report by Ronald E. Benton, dated September 9, 1976, identifies findings in an industrial hygiene survey at the Tylo plant on August 24 and August 25, 1976. Two areas were noted where several bags of asbestos was being stored. It was concluded that the housekeeping in the areas was poor with loose asbestos accummulating on table tops, benches, and floors. It was noted the asbestos was mixed by hand with water to form a paste, which was applied to pipes as an insulation material. The paste was not made daily but on a demand basis for patch work on existing insultation. Recommendations were made for proper precautions including clothing and respirator use during the cleanup of asbestos in the storage area, and it was recommended that the storage area be cleaned. It was also recommended that the use of asbestos for repair of pipe insultation be discontinued, and it was recommended that monitoring be done where there was residual asbestos from previous operations. Some recommendations for maintaining the plant from the standpoint of housekeeping, record keeping and medical examination was also made. In addition, it was noted that OSHA was considering lowering the asbestos fibers standard from 2.0 fibers/cc to 0.5 fibers/cc, and was also considering labelling asbestos as a carcinogen. The need for record keeping in view of that position was underscored.

In a letter dated June 24, 1983, Attorney John Barry Donohue, Jr., in a letter to Clifford J. Sheehan, Esq., regarding litigation on Rabpco, various employees vs. Johns Manville vs. Tylo Roofing Company, the following information is provided. He notes that the from 1961 through 1969 Tylo Company, Inc., manufactured mineral fiber siding, using long fiber asbestos as a binder. Manufacture of this product was discontinued in 1969 and sales terminated in 1970, or 1971, when the last inventory was sold. The entire factory site was sut down in 1981, the site sold and razed and it was further noted that the primary operation at the site had been the manufacture of asphalt roofing shingles, which did not contain asbestos.

The sum and substance of the above information is that Mr. Flanders R. Dobson did have significant exposure to asbestos fibers for approximately 20 years, largely in the manufacturing of an asbestos containing material to produce roofing and siding tiles, with a total exposure probably not exceeding 20-21 years, with evidencethat the company discontinued the manufacture of asbestos products in 1969 with most inventory sold off and no longer at the site by 1970, or 1971. Therefore, it is likely that the patient did not have significant asbestos exposure for the last seven or eight years of employment, with the patient retiring in 1978 at age 65.

HISTORY OF MEDICAL DISEASE, DIAGNOSIS AND MANAGEMENT:

11/82 - The first medical record available for review documenting patient's disease and treatment was an initial admission to the Park City Hospital on 11/19/82 with a history of epistaxis for six months and probable hemoptysis. The patient was known to have had hypertension for approximately ten years and known to have not taken medication on a regular basis. In addition, it was noted that in approximately 1940, or 1945, he had cauterization of the left nostril because of bleeding. He had surgery on the right neck at the Park City Hospital in approximately 1965, and at the time of discharge it was found that a lymph node had been resected revealing granulomas but without bacteria.

He was either given no treatment or may have received PAS alone for a period of one year, but that information is not substantiated. He had been seen in the emergency room at the Park City Hospital after a burn on the dorsum of the right hand in approximately 1979, and also had been seen in the emergency room after an accident in approximately 1977. The record documents the patient's work involving the mixing of asbestos including opening the bags of asbestos and producing the mixture, sometimes wearing a mask but frequently not wearing a mask, however arroneously summarizing his exposure to 27 years, rather than a probable 20-21 years.

The medical summary also indicates the patient had a 40-50 pack year history of smoking over a period of 45 years, beginning smoking at age 20 and discontinuing smoking at age 65, for a variable consumption level which at times reaches 1½ packs per day and for some period of years up to 3 packs per day.

At that time the patient had some shortness of breath on exertion when woaking 5-6 blocks and when climbing two flights of stairs. The admission blood pressure was 140-150/90-100 mmHg., and the patient was described as a well developed, well nourished black male who looked relatively well for his stated age.

Chest x-rays revealed a generalized increase in interstitial markings with subsegmental atelectasis and scar noted at both bases. In addition there was bilateral pleural thickening, which is greater on the right but no calcification was identified. Congestive heart failure could not be ruled out as a cause for the increased interstitial markings. Chest tomograms revealed generalized interstitial change with scarring at the right base but no calcification. Some pleural thickening was verified in both apices.

Pulmonary function tests on 11/22/82 revealed significant obstructive airway disease with only a partial response to bronchodilator and many parameters that did not show a significant change. The maximum voluntary ventilation was 49% of predicted but increased to 53% of predicted after bronchodilator. The diffusion capacity was 83% of predicted but total lung capacity was only 84% of predicted with an increased residual volume and RV/TLC ratio, consistent with obstructive airway disease. The study was interpreted as showing moderate obstructive disease and the diffusion capacity was considered within limits of normal, though only 83% of predicted. The total lung capacity was 6.04 liters with a predicted of 7.17, or 84% of predicted, but this was not commented upon in the report.

A PPD revealed a strong positive reaction and his sputum was evaluated by acid fast smear and culture, which was negative but with specimens sent to the State for further study. The report of culture subsequently revealed mycobacterium avium complex, and in December, 1982, he was placed on INH, 300 mg, and Rifampin, 300 mg. b.i.d. for nine months, which was completed.

His electrocardiograms reveal non-specific T-wave changes and premature ventricular contractions. His final diagnosis was hypertension, possible pulmonary tuberculosis, and epistaxis. COPD was not referred to in the discharge summary, though it was clearly noted on the record. It was also noted that several sputa for pap smear revealed atypical squamous cells with some squamous metaplasia. No definite malignant cells were identified. A second specimen on 11/29/82 suggests a few atypical cells suspicious for carcinoma with squamous metaplasia and a variety of other cells. A specimen of 11/26/82

revealed a few atypical squamous cells suspicious for carcinoma but again no definitive diagnosis could be made and the cells were placed in Class III.

A biopsy of the left main stem bronchus on 11/24/82 did not reveal evidence of neoplasm. The biopsy was obtained from a mildly abnormal area above the oriface of the left upper lobe bronchus.

On 8/11/84, the patient presented to the Park City Hospital as a 71 year old black male with previous treatment for tuberculosis and mild hypertension, who had a dizzy episode associated with a fall and a period of unconsciousness for approximately 10 minutes on arising from bed. He had a history of two previous falls in the last year. On further questioning the patient indicated that he had gone to the bathroom and after urinating had "blacked out." There were no GI symptoms. The patient did sustain a laceration over the forehead requiring sutures. He also admitted to two previous episodes similar to this one that were not evaluated or treated, His electrocardiogram revealed a sinus bradycardia and syncope was ascribed to bradyarrhythmia. His electrocardiogram revealed non-specific T-wave changes and a Holter monitor disclosed 50 ectopic beats per hours, but with no coupling or prolonged arrhythmia. An EEG was within normal limits. His hypertension appeared controlled during hospitalization. He was noted to have a sickle cell trait and on discharge was also given the diagnosis of chronic obstructive pulmonary disease. The chest x-rays on this admission revealed bilateral interstitial infiltrate, which appeared chronic and unchanged from previous films. The heart size was normal, and therefore there seems little evidence this could be congestive heart failure. An echocardiogram was within normal limits. Pulmonary function tests were not performed on that hospitalization.

4/11/85 - The patient was hospitalized at the Park City Hospital after being seen in the emergency room with a complaint of chest pain that was substernal in character without radiation. The patient denied nausea, vomiting, sweats, or dyspnea and pain radiated to the right shoulder rather than to the left. The records notes that he was receiving medication for COPD and hypertension, and he was also on Quinidine for arrhythmia. Examination of the lungs revealed no wheeze, rhonchi or rales. An echocardiogram was not remarkable and the patient's studies did not reveal evidence for myocardial infarction but his chest pain was ascribed to angina pectoris. He had a diagnosis of COPD, ASHD, compensated. He was placed on Quinidine, 200 mg. b.i.d., and Isordil, 20 mg. before meals and bedtime. An outpatient stress test was planned. A chest film on this admission revealed the lungs to be clear according to the interpreter but cardiac size was marginally increased. It is of some interest that no comment is made on interstitial markings and the diagnosis of pulmonary asbestosis was not made at that time.

4/27/85 - The patient was re-admitted to the Park City Hospital because of chest pain and shortness of breath that occurred while he was playing golf. It was noted that he was not a clear historian but there was no evidence for sweats, nausea or vomiting to suggest a myocardial infarction, though this needed to be ruled out. He gave a history at that time of sleeping with three pillows but had not had ankle edema. He did complain of paroxysmal nocturnal dyspnea. A chest x-ray was initially considered consistent with congestive heart failure. The record again notes a variable smoking history but the summary reveals a numerical error in that it indicates he was a heavy smoker for 58 years. The patient was noted to smoke between ½ pack and 3½ packs per day. In the hospital the patient was treated with diuretics, Quinidine, theophylline, and Isordil. Cardiac enzymes were within normal limits.

Pulmonary function tests were interpreted as revealing moderate airway obstruction with air trapping and the diffusion capacity was considered severely reduced, now being 24.9% of predicted. A review of airflows is certainly consistent with airway obstruction in that small airway parameters and peak flow are quite abnormal, though the FEV1/FVC ratio was 69% with a predicted of 66%, possibly reflecting a restrictive component. The FVC was 56% of predicted and total lung capacity was 92% of predicted with an RV/TLC ratio of 52%, suggestive of combined obstructive and restrictive disease. The chest x-ray revealed increased interstitial markings unchanged from 1982 and 1983, suggesting that interstitial pulmonary fibrosis was the mechanism. No pleural fluid was noted and cardiomegaly was again identified.

On the chest film of 4/28/85 a rounded area of increased density was noted in the right upper lung field, which was not present on previous studies and further evaluation was suggested. It was considered that this might represent a "pseudo tumor" and that congestive heart failure should be aggressively treated to rule this out. Further films revealed a resolving congestive heart failure but with incomplete clearing of the density and a question of right hilar adenopathy.

Final diagnosis on discharge on 5/2/85 was acute respiratory insufficiency due to chronic obstructive lung disease, asbestosis, and arteriosclerotic heart disease with ventricular arrhythmia and an undiagnosed density in the right upper lobe. A CT scan had revealed multiple pleural masses as well.

On 5/28/85, the patient was re-admitted to undergo percutaneous lung biopsy by Dr. Bader. The biopsy material revealed cells compatible with small cell carcinoma, especially compatible with the oat cell variety.

The patient was seen by Dr. Seyed Aleali, an oncologist, who initiated chemotherapy with plans to aradiate the right hilum at a later date after several courses of chemotherapy. The patient received Adriamycin, Vincristin, Cytoxan, and Decadron intravenously.

In a letter dated July 15, 1985, Dr. Simkovitz addresses the issue of etiology of the patient's disease in a letter to Attorney Donald Cousins, indicating that the patient has lung cancer of the small cell type, and that based on reasonable medical probability this is causally related to his asbestos exposure. In a letter dated June 20, 1985, Dr. Simkovitz indicates that "probably there is some factor in smoking which made him a little bit more susceptible to acquiring the lung changes." He concluded that the patient had both asbestosis due to asbestos exposure and that it was a co-factor in generating the carcinoma of the lung, indicating that any type of cell cancer could be associated with asbestos exposure.

REVIEW OF X RAYS: The following list of x-rays and CT scans have been available for review and returned to their primary facility: Chest x-rays were reviewed for 11/19/82, 11/22/82, 4/11/84, 4/11/85, 4/27/85, 4/28/85, 4/30/85, 5/2/85, 5/28/85, 7/15/85, and 4/1/86. In addition, scull x-rays on 8/11/84 and 9/11/84 are available for review. CT scans on 5/15/85, 5/28/85, 6/3/85, 6/6/85, 9/4/85, and 4/29/86 have been reviewed and returned to their original files. The interpretation of the above films and CT scans is not greatly different from that noted in the medical record, with the exception that the patient has manifested some increased interstitial markings, consistent with asbestosis for some years that were initially ascribed to possible congestive heart failure, but which were entirely too fixed to be explained on that basis. Since the pattern is consistent with the pulmonary fibrosis that may be seen with asbestos exposure, it is certainly reasonable to assume that this represents mild pulmonary asbestosis. There is evidence for bilateral pleural plaques, consistent with asbestos exposure as well. The tumor

mass and right hilar adenopathy is quite clear by x-ray and CT scans beginning in 1985, with some response to chemotherapy.

IMPRESSION: The review of records including report of laboratory studies and biopsy specimens, as well as the review of x-rays themselves and CT scans would lead me to conclude the patient had the following diagnoses:

COPD secondary to heavy cigarette smoking.
Mild pulmonary fibrosis consistent with asbestosis secondary to asbestos exposure.
Pleural plaques consistent with those porduced by asbestos exposure, not functionally significant.
Essential hypertension over 14 years with mild cardiomegaly.
ASHD with angina pectoris and arrhythmias, treated and controlled.
Probable atypical mycobacterium infection, treated and stable without evidence of recurrence.
Small cell carcinoma of the lung, treated by chemotherapy with partial response.

COMMENTS: The patient's pulmonary function tests clearly document the presence of some airway obstruction and his cigarette smoking history is certainly sufficient to anticipate at least mild COPD. The significant reduction in diffusion capacity in the last study suggests that significant pulmonary emphysema must be present since it would be difficult to ascribe this loss in diffusion capacity to pulmonary fibrosis alone, given the evidence for fibrosis by both x-ray and the measurement of pulmonary function testing, including total lung capacity. There is simply not enough restrictive disease to explain the loss in diffusion capacity to asbestosis alone. I therefore regard the asbestosis as definite and present as a reasonable explanation for the diffuse pulmonary fibrosis, which is chronic and relatively stable. I believe that pleural plaques are secondary to significant asbestos exposure in the past, but do not have functional significance from the standpoint of symptoms or impairment in function. The patient clearly had hypertension for at least 14 years that generally did not get consistent treatment, though in recent years it appears to be controlled. It certainly produced evidence of cardiomegaly and was no doubt a contributor to ASHD with arrhythmias and angina pectoris.

The patient's small cell lung cancer is the most serious complication of combined smoking and asbestos exposure. Generally, squamous cell carcinoma or adenocarcinoma are most common in patients who have lung cancer associated with moderate or heavy asbestos exposure. However, as noted by Dr. Simkovitz, it is possible to get any cell type associated with heavy smoking and an asbestos exposure of significance. Therefore, although we might ascribe an adenocarcinoma to asbestos induction as a most characteristic and reasonable cell type to ascribe to asbestos, nevertheless, we do in reality see all cell types.

In the literature the increased incidence fo lung cancer among patients who are significant cigarette smokers and have moderate or heavy asbestos exposure varies from 50-82 times the baseline population, including individuals of matched background and age who have neither a cigarette smoking history nor known asbestos exposure. In general, the current literature tends to favor an increased incidence closer to 50 times the baseline

when there are the combined hazards of smoking and asbestos exposure. In the patients studied with asbestos exposure but no smoking history, the increased incidence of lung cancer varies from three to five times that of the non-asbestos exposed, non-smoking individual. In addition, although there is this increased incidence of lung cancer among asbestos workers with moderate to heavy exposure, who have never smoked cigarettes, it is clear that the increased incidence is not higher than five times the baseline, and in many series closer to three times the baseline.

This data makes it quite clear that the major etiologic factor in inducing lung cancer is cigarette smoking and that the asbestos exposure is clearly a factor, but a minor factor. That is, one does not see very many lung cancers in individuals working in asbestos industries in the absence of a smoking history, though the numbers are clearly increased from a bseline population. Therefore, one must place the majority of blame for cancer induction on the cigarette smoking. The patient's cigarette consumption began at ½ pack per day, at age 20, and in more recent years had reached up to 3-3½ packs per day, in my experience an exposure intensity that tends to correlate with the development of more undifferentiated or highly malignant types of tumors. The lighter smokers have a tendency to acquire squamous cell carcinoma at a later age as a better differentiated and more slowly moving tumor. Those with very intense cigarette smoking, such as 2-3 packs per day, have a higher risk for a more malignant and rapidly growing tumor such as the small cell carcinoma. It is therefore my best medical judgement that the major factor in inducing lung cancer here was the patient's very heavy cigarette consumption history, and that the asbestos exposure was very definite and a definite but minor factor in cancer induction.

Since the latent period between first exposure to a cancer inducing agent and the development of the tumor may be as long as 30-40 years, we may ascribe the tumor formation to his 20 year period working in the mixing of an asbestos material for the production of shingles or siding, and we may consider his very heavy cigarette smoking in more recent decades as a major causative factor. Given the rate of growth of small cell tumors and their relatively rapid growth, it is likely that the tumor was initiated at a microscopic level well after the patient had terminated work at the Tylo Company, at which time the only on-going cancer inducing factor was his continued heavy smoking.

It is quite likely that the patient's continued smoking was a contributor to his arteriosclerotic heart disease and angina pectoris, as well as playing a role in the induction of lung cancer.

I believe the mild dyspnea on exertion experienced by the patient in recent years and first alluded to in 1982 was the product of COPD combined with mild pulmonary fibrosis consistent with asbestosis superimposed on arteriosclerotic heart disease and poorly controlled hypertension. I believe that all of these conditions combined produced an impairment materially and substantially greater than that that which could be ascribed to his asbestosis alone. I believe the pleural plaques are simply markers of asbestos exposure but do not represent a significant mechanism of impairment.

I trust this report responds to the questions raised with regard to the etiology of his disease. If further clarification is required, please do not hesitate to contact my office.

Sincerely,

Thomas J. Godar, M.D.
Director, Section of Pul[illegible]

TJG:ll
c: Hon. Frank J. Verrilli
Donald Cousins, Esq.