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METROPOLITAN LIFE INSURANCE COMPANY
FREDERICK H. ECKER, PRESIDENT
NEW YORK CITY

ANTHONY J. LANZA, M.D.
Associate Medical Director

March 20, 1931

Mr. Allan Kardwell,
15 Broad Street,
New York City.

Dear Mr. Kardwell:

Herewith is the report of the asbestos study. This is the summary report of the American plants studied. The Canadian summary report will be forthcoming shortly. In this report the plant of the Johns-Manville Company at Manville, N.J. is designated as Plant A. I am also sending a copy to Mr. Sayer and if you wish additional copies, please let me know. Needless to say, I will be very glad to discuss this report or amplify any portions of it if you so desire.

It is, of course, understood that this report is confidential and it will be given no publicity by us except with the consent of the firms concerned. After you have had a chance to go over it, I should like the opportunity of discussing with you and your associates the question of publication.

With best regards and assuring you of my sincere appreciation of your unfailing assistance which made this report possible, I am,

Sincerely yours,

A. J. Lanza, M.D.
Assistant Medical Director.

Enc.

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ALL-STATE LEGAL SUPPLY CO.

EFFECTS OF THE INHALATION OF ASBESTOS DUST

UPON THE LUNGS OF ASBESTOS WORKERS

Industrial Health Service
Policyholders Service Bureau
Metropolitan Life Insurance Company
New York

FOREWORD

The authors wish to express their sincere thanks to all those who aided in making this study possible, especially the officials and employees of the asbestos companies who cooperated to the fullest extent and gave every facility for securing data. Also to Dr. Pancoast of the University of Pennsylvania for his interest and valuable advice in interpreting X-ray films and to Dr. F. V. Meriwether, United States Public Health Service, Surgeon in Charge of the United States Bureau of Mines Cooperative Clinic in Picher, Oklahoma, whose interpretation of all the X-ray films listed in this report is followed. Also to Dr. W. Atmar Smith, and Dr. Kenneth Lynch of Charleston, S.C. for information and advice, particularly as to the pathology of asbestosis;* to Dr. W. W. Wild of Charleston, S.C.; to Dr. Joseph E. Wyatt of Newark, N.J., and Dr. E. E. Fellows of New York, for assisting in interpreting X-ray films; to Dr Paul O. Snoke of Lancaster, Pa.; to Dr. R. H. Stevenson of Danville, Quebec, Canada; to Dr. L. U. Gardner and Mr. Donald E. Cummings of Saranac Laboratory, who have undertaken the study of the pathological effects of the inhalation of asbestos dust through animal experimentation, with the aid of a grant from the Metropolitan Life Insurance Company.

* "Asbestosis Bodies in Sputum and Lung, Kenneth M. Lynch, M.D. and W. Atmar Smith, M.D., Charleston, S.C., Journal of American Medical Association, August 30, 1930, Vol. 95, No. 9, pp. 659-661.

INTRODUCTION

In 1929 the Metropolitan Life Insurance Company was approached by firms representing the asbestos industry in the United States with the request that a hygienic study be made of that industry. The officials representing these firms were desirous of ascertaining if asbestos dust was an occupational hazard in their establishments and, if so, what was the nature of this hazard and what should be done to prevent or control it.

About this time several articles had appeared in English medical journals describing a pneumoconiosis due to asbestos dust. While in one or two isolated instances the occurrence of this type of pneumoconiosis had been described in American journals, the industry itself appeared to be quite uninformed of the existence of any such occupational disease.

The hazard of silica dust with special reference to the lungs has long been appreciated and a great deal of study and research has been applied to the silica dust problem, primarily in the metal mining industry, in Great Britian, the United States, the British Dominions, as well as in other countries. The nature of the effects of silica dust expressed in the term "silicosis", with the resultant extraordinary predisposition to pulmonary tuberculosis, is well known. These effects have been associated with the inhalation of dust containing free silica in varying amounts. The effects upon the pulmonary tissue of dusts containing combined silica - silicates - are still a fertile field for investigation, but evidence is accumulating that certain of these dusts produce pathological results quite distinct from true silicosis.

The name asbestosis has been applied to the pneumoconiosis caused by asbestos dust and it will be so used in this report. Chemically, the asbestos of commerce is a hydrated magnesium silicate consisting primarily of

Silica 44.1%
Magnesia 43.0%
Water 12.9%

while ferrous iron and nickel are present in small quantities. This commercial variety of asbestos most commonly encountered is designated as chrysotile and is one of the four varieties of the mineral serpentine, in which it usually occurs in seams.

It should be borne in mind that silicosis (and presumably asbestosis) develops very slowly, taking from five to fifteen and even twenty or twenty-five years to become established. This rate of progress is influenced mainly by the dosage of silica which the lungs receive and this dosage, in turn, depends upon three variables: the amount of silica in the dust, the quantity of dust in the air, and the extent of exposure. Individual idiosyncrasy might be included as a fourth variable; however, little is known of the variations in susceptibility in those exposed to dust.

There does not appear to be present, in places where asbestos is mined or fabricated in North America, the clear-cut clinical picture which is so unescapable in communities with a true silicosis hazard, such as hard rock mining communities. It might be thought that some of the asbestos plants are of too recent origin for the typical effects of a silicate dust to become manifest, but this theory would not apply to the older mines or to all of the fabricating plants.

The Industrial Health Service of the Metropolitan Life Insurance

Company undertook the following investigation during the period from October, 1929 to January, 1931. This investigation included:

A. A study of dust conditions in asbestos mines, mills, and fabricating plants.

B. Physical examinations of asbestos workers, including X-ray films.

C. A study of dust exhaust systems designed to eliminate asbestos dust.

Asbestos fabricating plants located along the Atlantic Seaboard were included in this study and are designated in the report as plants A, B, C, D, and E. Asbestos mines and mills in the Province of Quebec are designated as F, G, H, and I.

The Canadian portion of the investigation was conducted jointly with the Industrial Hygiene Division of the Department of Public Health of the Medical School of McGill University. The results will be summarized in a report which will follow the present one dealing with the investigation in the United States.

DUST STUDIES

Apparatus and Methods of Sampling:

Both the impinger (1) and the electric precipitator (2) were used for the collection of air samples, for dusts, at the breathing levels and in close proximity to the workmen. Both of these forms of apparatus are adapted for this work, as they are transportable, easily set up and adjusted for the taking of the air samples at the proper level, and are extremely efficient.

The impinger collects the dust by aspirating the air and then impinging it onto a flat surface covered by a liquid in a container. The liquid used was distilled water, containing 50 percent alcohol to prevent the solution of some of the dusts, particularly any silica which might be present. The impinger was actuated by an electrically driven rotary pump and the rate of air sampled was measured by means of a resistance type of flow meter with an inclined manometer for measuring the pressure difference. Each sample represents the dust collected from a volume of 100 cubic feet of air.

The electric precipitator is designed upon the Cottrell precipitator principle used for recovering dusts and fumes commercially. This principle depends on electrifying the dust particles by making them pass through an electrostatic field and thus causing them to settle out upon a sheet of celluloid. The electrostatic field is set up by means of a transformer operating from the lighting lines on 120 volts, alternating current. Air is drawn through the apparatus by means of a small rotary fan, run by a motor, the quantity being

(1) "Comparative Tests of Instruments for Determining Atmospheric Dusts", Public Health Bulletin No. 144, Treasury Department, Washington, D.C.

(2) "Determination of Suspensoids by Alternating-Current Precipitators", Philip Drinker and Robert M. Thomson, Journal of Industrial Hygiene, Vol. VII, No. 8, June, 1925.

measured by means of a flow meter.

Samples of dust brushed from beams and pipelines near the ceiling also were secured for chemical analysis. All samples were shipped to the Industrial Hygiene Laboratory of the Metropolitan and the dust counts made according to the methods accepted by the U.S. Bureau of Mines and the U.S. Public Health Service.(1) Particles in size up to 360 microns in the greatest diameter were counted.

Chemical analyses were made for total (free and combined) silica according to the accepted standard method of Hillebrand.(3)

Percentage Frequency of Particle Size:

In the first plant studied (A) only particles 10 microns and under in size were counted. It has been demonstrated repeatedly that no silica particles exceeding 10 microns - a micron equals one-millionth part of a meter or one-thousandth part of a millimeter - in greatest diameter, enter the lung tissue, and for this reason the larger particles usually are not considered when determining dust counts. Later, as it appeared from some of the published articles(4) (English) that asbestos particles much larger in size were found in lung tissue, it was decided to count all particles and make differential counts of all those 10 microns and under.

Both samples collected by the impinger and the electric precipitator were counted. Microphotographs of these dust particles were further enlarged by being projected upon a screen by means of a lantern slide projector, the enlarged particles being measured with a ruler. Knowing the entire enlargement (by actual

(1) "Comparative Tests of Instruments for Determining Atmospheric Dusts", Public Health Bulletin No. 144, Treasury Department, Washington, D.C.
(3) "The Analysis of Silicate and Carbonate Rocks", W.F. Hillebrand, Bulletin 422, U.S. Geological Survey, Washington, D.C.
(4) "Pulmonary Asbestosis", W. E. Cooke, British Medical Journal, December 3, 1927.

measurement), it was easy to calculate the original particle size.(5) By the use of Hazen's (6) logarithmic probability paper, the logarithms of the function to be measured - in this case microns - are plotted as ordinates against the probability of occurrence as abscissae. Since the line is straight, extrapolation and interpolation of any desired region is facilitated.

The analyses are reported in total particles per cubic foot and also in the corresponding weight of the dust in milligrams. There is no indicated relationship between the dust counts and the corresponding weights. Consequently, the weights are not included in the tabulations in this report.

Table No. 1 shows by plants and by departments the dust counts in million particles per cubic foot of air. The plants included are all in the United States and are mainly of a textile nature. It will be noted that the counts in plant A are of particles 10 microns and under, while in the other plants all the particles were counted. Nevertheless, direct comparison is possible, as in no sample taken were the number of particles 10 microns and under less than 94 percent of the whole, which makes it evident also that in regard to dust control we are concerned only with the smaller sizes.

It is also important to note that whereas the dust in the preparation room is practically all due to asbestos, the contrary is the case in the other departments.

In the carding, spinning and weaving rooms asbestos is about 25 per cent. of the material used. On special jobs the percentage of asbestos may be

(5) Green, H. "A Photomicrographic Method for the Determination of Particle Size of Paint and Rubber Pigments", Journal Franklin Institute, 1921, 192, 637.
(6) Whipple, G.C. - Vital Statistics, John Wiley & Sons, N.Y., 1925, p. 451.

higher, but in general the figure quoted is approximately correct, the other material consisting principally of cotton. In the insulating departments of plant A, asbestos is but 5 per cent. of the total material used.

In the molded brake lining and clutch division of plant D, asbestos is about 25 per cent. of the total material.

The actual exposure to asbestos dust, therefore, is considerably less than is indicated in the dust counts in Table I.

TABLE I

Dept.	PLANT A		PLANT B		PLANT C		PLANT D		PLANT E	
	No. Samples Taken	Million Particles per cu.ft. of air	No. Samples Taken	Million Particles per cu.ft. of air	No. Samples Taken	Million Particles per cu.ft. of air	No. Samples Taken	Million Particles per cu.ft. of air	No. Samples Taken	Million Particles per cu.ft. of air
Preparation	5	Under 10 to 1½	9	½ to 8	8	2 to 43	—	—	5	30 to 82
Card room	5	½ to 1 2/5	5	¾ to 4¾	6	5½ to 16½	—	—	5	26 to 78
Ring frame spinning room	2	½ to ¾	—	—	—	—	—	—	—	—
Mule spinning	2	2/3 to 2½	5	1/5 to 1	5	1¼ to 4½	—	—	5	5½ to 10
Twisting	10	1/5 to 1¾	—	Included in spinning	9	1 to 5¾ (Includes broad loom weaving)	—	—	5	6 1/3 to 7 (Includes broad loom weaving)
Weaving	4	1/20 to 2	4	1/5 to 4/5	6	5¾ to 10	15	1 to 7	5	10½ to 19¾
Felt Dept.	6	1/3 to 44½	—	—	—	—	—	—	—	—
Sheet insulating	3	1/3 to 4	—	—	—	—	—	—	—	—
Molded brake band and clutch	—	—	—	—	—	—	2	½ to 3/5	—	—

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CONDITIONS IN THE PLANTS

The processes in these plants were very similar to cotton mills in general. For illustration, the process in the preparation room of plant B is detailed at length:

Asbestos is received in bags and these are emptied upon the floor and the asbestos is shoveled into pug mills and run for about five minutes in order to crush and open the fibres. From there the asbestos is shoveled into trucks and wheeled over to hoppers with either horizontal or vertical openers, similar to those used in the textile mills. After passing through the opener, the material is discharged into trucks. Waste manufacturing material is first run through a garnet machine and discharged into trucks which are wheeled over to the openers and the material is fed into these in the same manner as the new material. The material, after being discharged from the opener is put onto a vibrating or shaking screen where the long fibres are picked off by a suction hood and blown into a bin to be used again in textiles. The short fibres falling through the screen are either sold as such or used in making an asbestos cement.

The cotton is received in bales, opened, and also run through vertical openers. It is discharged into trucks. The filled trucks from the vertical and horizontal openers containing either the asbestos fibre, the cotton or the waste material are transported to separate storage bins. As needed, these materials are weighed and dumped onto the floor in proportion to the mixture desired and the resulting mixed materials are then passed through

a mixing picker. As this mixture is discharged from the picker, it is sprayed with a light mineral oil. The material is then passed onto a mechanical conveyor and taken to the storage bin in the card room. Two of these mixing pickers discharge onto this conveyor system, while a third machine is equipped with a suction device which conveys the material to a storage bin in the card room. The object of the two systems is to facilitate the handling of two grades of material at the same time. This third machine is not equipped with an oil spray. The object of the oil spray is primarily to entrap the small asbestos fibres and hold them enmeshed in the cotton fibre throughout the processes of carding, spinning, and weaving. Incidentally, it is apparent that it also diminishes the amount of dust. The material is saturated with about 4 per cent. of oil at this point, but by the time it reaches the looms the oil has diminished to less than 1 per cent.

While mineral oil was used in the preparation room of plant C, it apparently was not as efficacious as in plant B. In plant E oil was not used, nor was there any attempt at humidification or any system of dust exhausting. Carding machines were fed by hand. In plant A there was a humidifying, ventilating and heating system, while in plant B natural humidity sufficed for the process. In plant E the carding machines were equipped with a dust exhaust system, but it was not efficiently used. In plant C carding was done in two buildings, one of which was equipped with an air conditioning system, and in both buildings the machines had exhaust equipment.

Two plants, B and C, had artificial humidity installations in their spinning rooms. In the former the temperature was 76°F., relative humidity 78 per cent.

One plant, C, had a humidification system in the twisting department

and also in the weaving department where the relative humidity was 76 per cent. with a dry bulb temperature of 69°F., as compared with a relative humidity of 44 per cent. in the weaving room of plant A.

Aside from plant E, which was unnecessarily dusty, it would appear that the dust hazard was not excessive except in the preparation rooms of plants B and C. The dust counts are interesting too, when considered in the light of the permissible standard for siliceous dust, established by the United States Public Health Service,* namely 10 million particles (10 microns and under) per cubic foot. However, we are not justified in assuming that because asbestosis is a milder disease, clinically, than silicosis, the threshold of permissible dust counts is higher. Asbestosis is pathologically different from silicosis, and the experience so far does not warrant an attempt to define an arbitrary standard of dustiness.

DUST CONTROL

Various measures, such as oiling, humidification, and local exhausts, tended to reduce the dust. Nevertheless, it was evident that they were only partially successful. It also is apparent that, if it is expected to control dustiness in these plants, final reliance must rest upon properly constructed exhaust equipment. In plant C in one department an experimental installation was set up. In spite of some obvious faults, this equipment, on the basis of comparative dust counts, reduced the dust by 50 per cent. and with further alterations will probably be 75 per cent. effective. For all practical purposes, this is quite satisfactory. It is neither practicable nor economically desirable to install such equipment as will make the air entirely dust free, even if this

* "Health of Workers in Dusty Trades (Granite Industry", Public Health Bulletin No. 187, U.S. Public Health Service, Washington, D.C., 1929.

were possible. The normal defensive mechanism of the body takes care of a fair amount of atmospheric pollution. It is when the body is exposed to an excessive amount of dust over a long period of time that this defense mechanism breaks down.

The application of exhaust equipment to textile machinery involves considerable difficulty, especially where the construction of the plant is such that it is not possible to apply down draft suction to the looms.

It is desirable to install exhaust apparatus in any plant on an experimental basis first, and then check its efficiency by dust counts. This practice will result in saving the needless expenditure of much money.

PHYSICAL EXAMINATIONS

It was soon obvious that the diagnosis of asbestosis must rest mainly on X-ray films of the chest. As in the early stages of silicosis, the symptoms of the asbestos workers were usually indefinite and inconclusive. The following tables are based on physical examination, together with X-ray films of 126 persons actually working in asbestos plants in the United States. No Canadian results are included here. These cases were selected more or less at random from among those having more than three years of employment in the industry. The interpretations of these films all are based on the readings of one competent roentgenologist, but they have been studied by several others experienced in this field. The differences of opinion were of a minor nature, and there was general agreement as to interpretation. The films were read conservatively, the X-ray findings took into account the physical examination and the age of the individual, and only those were classed as positive about which there was

no major disagreement. All concerned were guided by their experience in silicosis and other types of pneumoconiosis, hence the films classed as negative for asbestosis were further subdivided into "doubtful" and "negative". Only time can tell whether the individuals classed as doubtful are progressing toward a definite asbestosis. Particular attention was focussed on the presence or absence of indications of tuberculosis and, in this respect, all the reviewers of these films were in accord. With the unhappy experience of silicosis in mind, it was felt that a great deal of care should be devoted to ascertaining, if possible, whether or not asbestosis predisposes to tuberculous infection.

Table II exhibits the diagnostic classification of these 126 individuals, with their symptoms and specific occupations. Five of these persons were not physically examined.

The cases of asbestosis are divided into two classes, first stage and second stage. The first stage embraces those who show by X-ray examination definite lung pathology sufficient in extent to warrant a diagnosis of pneumoconiosis, but who have no definite symptoms. The second stage cases exhibit more extensive lung pathology and also definite symptoms.

All these individuals* were actively engaged in factory work, and it was not practicable to make any distinction between them on the basis of working ability or disability.

Had any individuals been found who exhibited extensive pulmonary involvement and marked physical disability or total disability, they would have been classified as third stage, but no such were found.

*One asbestos worker (2nd degree stage) retired of his own accord and because of old age ten years previous to this study, but is still active about his garden.

It will be seen from Table II that of a total of 126 X-ray examinations four were diagnosed as second degree asbestosis; sixty-three as first degree; thirty-nine as doubtful; and twenty as negative.

Table II also shows that only eight individuals out of sixty-four with positive asbestosis were entirely free of symptoms, while ten out of thirty-seven with doubtful and seven out of twenty with negative diagnoses were free of symptoms.

Dyspnoea and cough were the symptoms most complained of, but none of these cases exhibited the urgent or evident type of "short wind" seen in true silicosis. Several of those classed as negative stated that they were "short-winded" and were so recorded, but too much emphasis should not be placed on these statements of subjective symptoms. Table III shows the occurrence of dyspnoea and cough.*

TABLE III

	Positive	Doubtful	Negative
Dyspnoea	40	16	8
Cough	39	20	10
Not examined	3	2	0
Total	67	39	20

The incidence of tuberculosis (based upon X-ray films) as revealed in Table IV is both interesting and important.

TABLE IV

	Positive	Doubtful	Negative
Tuberculosis (healed)	7	1	2
Tuberculosis (active)	1**	0	1
Total Examined	67	39	20

*In Tables III to XI, inclusive, the division into positive, doubtful, and negative refers to the cases as classified in Table II.

**This case was diagnosed as probably active on the X-ray findings.

During the progress of the study, physicians who were practicing in the communities where asbestos workers lived were questioned, and stated that they did not find an unusual amount of tuberculosis among these workers. The contrast between this state of affairs and that found in communities with a silicosis hazard, is noteworthy.

The following tables classify results of examinations by occupation in industry and by years of exposure. It is to be remembered, however, that these individuals are classified according to the department in which they were working at the time of examination or where they had spent most of their time. Many had changed from one department to another and from one plant to another during their years of employment in the asbestos industry. Since only the amounts of dust collected at the time of this survey in the various departments are known, there is no way of knowing the average amount of dust in the atmosphere inhaled by these people over the years of their employment. Consequently, it is not possible to correlate individual cases with definite amounts of dust exposure.

TABLE V

By Departments

	Positive	Doubtful	Negative
Weaving	22	13	5
Carding	13	4	1
Spinning	9	3	3
Warping	1	0	2
Spooling	3	5	2
Trucking	1	1	0
Loom fixing	3	1	0
Packing	1	0	0
Winding	3	4	1
Picker	2	0	2
Twisting	3	5	0
Other	6	3	4
Total examined	67	39	20

29	Downing	4	M	45	Peribronchial thickening and mottling, particularly bases.	Dry rales posteriorly at bases.	2½ inches	Dyspnoea; swelling of skin around nails.	---
30	Downing	4	M	53	Considerable early mottling in both lungs.	Scattered rales; diminished breath sounds.	1 inch	Dyspnoea; cough (dry); history of chest trouble.	Mottled like right shou...
31	Downing	4	M	54	Peribronchial thickening and early mottling both lungs.	Not examined.	Not taken	Not questioned.	---
32	Downing	4	M	56	Peribronchial thickening and mottling both lungs.	--	2 inches	None	---
33	Downing	11	M	61	Dullness in both lungs.	Heart enlarged; heart murmurs; increased valve sounds	1½ inches	Dyspnoea (slight)	Heart enlarged
34	Harding	15	M	62	Peribronchial thickening both lungs; mottling.	Scattered rales.	1 inch	Dyspnoea; cough on inhalation.	---
35	Harding	18	M	59	Peribronchial thickening and mottling both bases.	Dullness left apex.	2½ inches	Dyspnoea (slight); cough (with expectoration).	Mottled... left apex; thickening peribronchial (right shoulder)
36	Electric and steel work	14	M	59	General mottling both lungs, density right apex.	Dullness rt. apex; diminished breath sounds.	1 inch	Dyspnoea; cough on inhalation.	Rt. right apex. Probably active.
37	Moseley	11	M	59	Fine mottling especially on left side.	Dullness right upper lobe.	2 inches	Dyspnoea; cough (dry)	---
38	Spooling	6	F	63	Heavy and dense shadows both bases.	--	1½ inches	Dyspnoea (slight); cough (with expectoration); swelling of skin around nails.	Heart disease (mitral regurgitation).
39	Wooding	4	M	56	Dense peribronchial thickening and mottling both lungs.	Not noted.	--	--	---
40	Dooling	5	M	50	Peribronchial thickening and some mottling.	Diminished breath sounds.	1½ inches	Cough (dry); poor appetite morning.	---
41	Eglesing	6	F	51	Fine mottling in both bases, particularly right.	Slight dullness at bases.	2 inches	Has lost weight; swelling of skin around nails.	---
42	Carling	3	M	21	Considerable thickening and mottling both lungs.	--	2 inches	None	---
43	Fisher	5	M	46	Some thickening and mottling in both lungs, particularly right.	--	1½ inches	Dyspnoea; cough (dry).	---
44	Carling	3	M	49	Peribronchial thickening and mottling both lungs.	--	3 inches	Slight cough (with expectoration).	---
45	Harding	7	M	51	Shadows enlarged; peribronchial thickening both lungs.	--	2 inches	Slight cough (dry); swelling of skin around nails; subacute nurse.	---
46	Eglesing	6	M	53	Considerable peribronchial thickening and mottling both bases.	Heart enlarged; dullness left base.	1½ inches	Cough (with expectoration).	---
47	Dooling	3	M	53	Hilum shadows show several calcified spots in both lungs; some mottling.	--	1½ inches	Dyspnoea (slight); cough dry.	---
48	Carling	8	M	56	Shadows enlarged; fine mottling both lungs.	Heart enlarged.	2½ inches	Dyspnoea; cough (dry); history of colds; poor appetite.	---
49	Carling	3	M	59	Shadows enlarged; fine mottling both lungs.	--	2 inches	Dyspnoea; cough (dry); swelling of skin around nails; subacute nurse.	---
50	Eglesing	7½	M	53	Shadows enlarged; rather general mottling both lungs.	--	2 inches	Dyspnoea (slight); subacute nurse.	---
51	Carling	3	M	53	Calcified spots in both hilum shadows; fine mottling both lungs.	--	1 inches	Dyspnoea (slight); cough (dry).	---
52	Eglesing	7½	M	54	Hilum shadows enlarged; small amount thickening and mottling both lungs.	--	3 inches	Dyspnoea (slight); subacute nurse.	---
53	Accarling	3	M	57	Fine mottling in both lungs.	Rales at bases; diminished breath sounds.	1 inch	None	Heart disease (mitral regurgitation).
54	Dooling	8	M	60	Hilum shadows enlarged; dense mottling throughout.	Increased valve sounds.	2 inches	Dyspnoea; cough (with expectoration);	---

46	Carding	1	M	33	[illegible]			1½ inches	Dyspnoea (slight).		
57	Weaving	18	M	31	Some thickening and mottling, particularly at bases.	—		1½ inches	None		
58	Twisting	12	F	33	Peribronchial thickening and mottling both lungs.	—		1 inch	Dyspnoeal cough (dry).		
61	Spinning	8	M	29	Peribronchial thickening and mottling both lungs.	Scattered rales; diminished breath sounds.					
62	Several different depts.	15	M	61	Hilus shadows enlarged; generalized mottling both lungs.	Dullness at bases.		2½ inches	Cough (with expectoration); history of chest trouble.		
63	Spinning	6	M	48	Peribronchial thickening in both lungs; generalized mottling	—		2 inches	Cough (dry); swelling of skin around nails.		
56	Carding	7	M	31	Hilus shadows enlarged; considerable peribronchial thickening throughout.	—		1½ inches	Cough (dry); history of chest trouble.	Tumor on neck.	
51	Carding	8	M	24	Some thickening and mottling both lungs.	Rales at bases; some dullness.		2 inches	Dyspnoea (slight); poor appetite; sebaceous cornu.		
52	Picker	5	M	41	Peribronchial thickening and mottling both bases.	—		3 inches	Cough (with expectoration).		
53	Carding and dressing	9	M	38	Considerable peribronchial thickening and mottling both lungs.	Dullness at bases; diminished breath sounds.		1½ inches	Convulsions some dyspnoea		
54	Elevator operator	9	M	49	Mottling throughout both lungs; some calcified spots.	Rales and dullness at right base.		2 inch	Dyspnoea; cough (dry); poor appetite (morning); swelling of skin around nailbe.	Aortic murmur.	
55	Weaving	11	M	35	Right hilus enlarged; considerable mottling both lungs.	Diminished breath sounds;		2 inches	None		
57	Low run (doubling)	18	M	44	Considerable mottling both bases.	Increased voice sounds at bases.		2 inch	Losing weight; loudon hoar cough (dry).		
58	Weaving	8		41	Considerable peribronchial thickening and mottling both lungs.	Rales right apex.		2 inches	Cough (dry); swelling of skin around nails; sebaceous cornu.	Healed tbc.	
59	Weaving	9	M	38	Some mottling and peribronchial thickening both lungs.	Slight dullness at bases.		3 inches	Dyspnoeal cough (dry); swelling of skin around nails.	Healed tbc. apex.	
60	Weaving	11	M	37	Peribronchial thickening and mottling, particularly at bases	Diminished breath sounds.		3½ inches	Dyspnoea cough (with expectoration); lost weight; history of chest trouble.		
61	Weaving	10	F	+38	Marked hilus involvement; slight peripheral.	—		1½ inches	Dyspnoea (slight); cough (with expectoration); history of chest trouble.	Poor disease.	
62	Twisting	15	F	37	Rather marked mottling at bases.	Rales posteriorly, some dullness.		2½ inches	Dyspnoeal cough (dry); poor appetite (morning); history of chest trouble.	Healed tbc.	
63	Carding	5	M	44	Generalized mottling both lungs.	—		1 inch	Dyspnoeal cough (dry); slight clubbing of fingers.		
64	Weaving	11	M	36	Hilus shadows enlarged; mottling at bases.						

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26	Trimling	6	M	37	Some generalized mottling both lungs.	--	3 inches	Dyspnoeal cough (with expectoration); slight loss of appetite; lost weight; some moderate edema.	---
27	Gosling	6	M	47	Some peribronchial thickening both lungs near bases.	--	1½ inches	Dyspnoeal cough (dry)	---
28	Chorling	1½	M	21	Fine mottling scattered in both lungs.	--	3 inches	Some. Edematous edema.	---
29	Keeling	9	M	27	Considerable thickening in both lungs.	--	2 inches	Swelling of skin around nails.	---
30	Keeling	9	M	45	Several business both lungs; fibrotic bands at bases.	Diminished breath sounds.	2 inches	Dyspnoeal cough (dry); slight emaciation; edematous edema.	---
31	Keeling	6	M	37	Small amount mottling at bases; right hilus enlarged.	Dullness at bases; diminished breath sounds.	1½ inches	Dyspnoeal cough (dry); poor morning appetite; lost weight.	---
32	Keeling	10	M	45	Small amount thickening and mottling throughout both lungs.	--	1½ inches	Dyspnoeal poor morning appetite; swelling of skin around nails.	---
33	Keeling	6	M	30	Some peribronchial thickening both lungs; very small amount mottling.	Diminished breath sounds at bases.	2 inches	Dyspnoeal cough (dry); poor morning appetite.	---
34	Keeling	6	M	37	Some peribronchial thickening; mottling in both bases.	--	2 inches	Dyspnoea slight cough (with expectoration); slight swelling of skin around nails.	---
35	Spoolling	6	M	53	Some peribronchial thickening and fine mottling both lungs.	Some dullness at bases.	2 inches	Dyspnoeal cough (with expectoration); clubbing of fingers and swelling of skin around nails.	---
36	Keeling	9	M	37	Hilus shadows enlarged fine mottling throughout.	Slight dullness at bases; diminished breath sounds.	1½ inches	Dyspnoeal cough (dry); emaciation.	Death of Mr.
37	Spoolling	5	M	50	Considerable peribronchial thickening and fine mottling.	Diminished breath sounds.	2 inches	Dyspnoeal cough (with expectoration); leaden hue.	---
38	Twisting	5	F	19	Small amount peribronchial thickening fine mottling.	--	1½ inches	None.	---
39	Cording	4½	M	21	Some mottling both lungs, hilus shadows enlarged.	--	2 inches	Slight cough (with expectoration).	---

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TABLE II

Pathological Asbestosis (Classified as Negative)

Case No.	Dept. of Plant	Years of Exposure	Sex	Age	X-ray	Chest Findings	Chest Expansion	Symptoms	Complications
1	Winding	5	M	33	Small amount mottling; more fibrosis than normal.	--	2 inches	--	---
2	Trucking	3	M	38	Small amount thickening both lungs.	--	2½ inches	Slight cough (dry).	---
3	Winding	7	M	31	Some fine mottling; more fibrosis than normal.	--	2 inches	Cough (dry); swelling of skin around ankles; asbestos corns.	---
4	Twisting	5½	M	33	Hilus shadows enlarged; more fibrosis than normal.	--	2½ inches	Dyspnoea (slight).	---
5	Spooling	8	F	30	Beginning mottling at bases; some peribronchial thickening.	--	1½ inches	Dyspnoea; cough; (4th expectoration); poor evening appetite, constipation.	---
6	Spooling	5	M	31	Some peribronchial thickening; small amount mottling.	--	3½ inches	--	---
7	Winding	4	M	32	Some early mottling and thickening both lungs.	--	2 inches	--	---
8	Tape fla- ing	5	F	31	Hilus shadows enlarged; some mottling both lungs.	--	2 inches	Dyspnoea (slight); cough (dry); uniform weight; clubbing of fingers.	---
9	Jeering	1½	M	23	Hilus shadows enlarged; some peribronchial thickening.	Not examined.	--	--	---
10	Jeering	8	F	60	Calcified apex left hilus; some mottling both lungs.	Not examined.	--	--	---
11	Jeering	4	M	23	Hilus shadows enlarged; small amount mottling.	--	3 inches	Swelling of skin around ankle.	---
12	Jeering	6	M	24	Some thickening both lungs.	--	1½ inches	None.	---
13	Twisting	8	F	37	Hilus shadows enlarged; some mottling	--	1½ inches	None.	---
14	Spooling	2½ part time	F	31	Hilus enlarged (right); small amount mottling both lungs.	Heart enlarged, mitral murmur.	1 inch	Dyspnoea.	Aortic Aneurism.
15	Spooling	6	M	22	Some thickening and mottling both bases.	--	1 inches	Cough (dry).	---
16	Carding	3	M	39	Some peribronchial thickening; early mottling right lung.	--	1½ inches	Cough (with expectoration).	---
17	Twisting	9	F	36	Hilus shadows enlarged; peribronchial thickening both lungs.	--	2 inches	Slight cough (with expectoration); constipation.	---
18	Twisting	5	F	37	Some peribronchial thickening and mottling both lungs.	--	1½ inches	None.	---
19	Carding	8	M	41	Some peribronchial thickening and early mottling both lungs.	Suggestion of dullness at bases.	2 inches	Cough (dry); constipation.	---
20	General	10	M	47	Considerable peribronchial thickening and early mottling at bases.	--	2½ inches	None. Asbestos corns.	---
21	Jeering	7	M	31	Hilus shadows enlarged; some mottling both lungs.	--	2½ inches	Dyspnoea; swelling of skin around ankle.	---
22	Spinning	13	F	33	Some fine mottling both lungs; hilus shadows enlarged.	--	1½ inches	Dyspnoea (slight); cough (dry).	---
23	Spinning	8	M	28	Fine mottling both bases; right hilus enlarged.	--	2 inches	None	---
24	Spinning	6	M	30	Fine mottling both lungs; hilus shadows enlarged.	Heart enlarged.	2 inches	Cough (dry); occasional swelling of skin around ankles; dental caries.	---

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TABLE II

Receipts for Asbestosis

Case No.	Occupation of Parent	Years of Exposure	Sex	Age	X-ray	Chest Findings	Chest Expansion	Symptoms	Complications
1	Spinning	3	F	41	---	---	3½ in.	Cough (dry)	---
2	Carding	12	F	60	---	Diminished breath sounds.	1½ in.	Cough (dry)	---
3	Carding	17	M	47	---	---	2½ in.	Slight cough (with expectoration); loosening of skin around nails.	---
4	Carding	4½	M	46	Pleuritic adhesions both bases.	Friction rub at base.	2 in.	Dyspnoea (slight)	---
5	Receiving	8	M	26	---	---	3 in.	None	---
6	Spinning	5	M	21	---	---	1½ in.	None	---
7	Spinning	6	M	20	---	---	3 in.	None	---
8	Carding	4	F	29	---	---	1½ in.	None	---
9	Piecer	3	M	27	---	---	2 in.	Dyspnoea (slight); cough (with expectoration; asbestos corn.	---
10	Jiggling	3½	M	21	---	---	4 in.	Losing weight.	---
11	Piecer	3	F	33	---	---	¾ in.	Dyspnoea (slight); cough (with expectoration;	---
12	Waste	4	M	28	---	---	2½ in.	Slight cough (with expectoration).	---
13	Feeding	13	M	31	---	---	2 in.	None. Asbestos corns.	---
14	Grinding	3½	M	24	---	---	2½ in.	None.	---
15	Carding oiling	1 mos.	M	31	---	---	1½ in.	None.	---
16	Weaving	4	F	36	---	Slight dullness left side.	1 in.	Dyspnoea (slight); cough (with expectoration.	Fibroid pulmonitis
17	Carding	12	F	6	---	Rles (general) dullness; diminished breath sounds.	1½ in.	Dyspnoea; cough (with expectoration); emaciation; poor morning appetite.	Fibroid tbc. (left apex) chronic bronchitis
18	Weaving	18 (left apex impaired)	M	45	---	Dullness at apices.	2½ in.	Dyspnoea; cough (dry); poor morning appetite.	Fibroid tbc. (apices)
19	Weaving	3	M	37	---	Rles, dullness left apex.	¾ in.	Dyspnoea; cough (dry); emaciation.	Tbc. left apex (possibly active)
20	Weaving	3	M	31	Cavity mid-section both lungs, calcified spots.	Rles (scattered)	¾ in.	Dyspnoea (slight)	Tbc (?)

TABLE VI

Years of Exposure

	Positive	Doubtful	Negative
Over 15 years	13	1(part time)	3 (1 not con-
10 to 15 years	7	3	2 tin-
5 to 10 years	30	27	3 uous)
Under 5 years	17	11	12
Total examined	67	39	20

The following tables are also of interest, but, like the two previous, are of limited value and little in the way of definite conclusions can be based upon them.

TABLE VII

Age Groups

	Positive	Doubtful	Negative
50 and over	12	3	2
40 to 49	13	6	4
30 to 39	17	12	6
20 to 29	24	16	8
Under 20 years	1	2	0
Total examined	67	39	20

TABLE VIII

Sex

	Positive	Doubtful	Negative
Male (Total examined 108)	59	32	17
Female (" " 18)	8	7	3

TABLE IX

Chest Expansion

	Positive	Doubtful	Negative
1 to 2 inches	28	13	7
2 to 3 inches	31	21	12
3 inches plus	5	3	1
Not examined	3	2	0
Total	67	39	20

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It is possible that not sufficient attention has been paid to the effects of the pneumoconioses upon the heart.

INSURANCE CLAIMS

In the effort to throw any possible light on the relationship between asbestosis and pulmonary tuberculosis, an analysis of death claims and total and permanent disability claims was made in regard to those companies carrying Group insurance and where the figures were available.

There were 2099 lives involved for a total exposure of 7019 life years. The death claims are so small in number that reliable conclusions can not be arrived at from any subdivision of the figures. The same is true of the sickness claims under health insurance. The number of claims for respiratory diseases is high in two of the plants studied but low for the others, based upon the Metropolitan experience of 1927. However, during the latter part of 1928 and the first part of 1929 there was an epidemic of influenza.

The records of one establishment (plant B) showed that six of thirty-six death claims and eight of ten permanent and total disability claims were listed as due to pulmonary tuberculosis. This appeared to be an inordinate amount of tuberculosis from this plant. However, many of the employees were Negroes and also tuberculosis claims are generally high in this section of the country. Realizing the difficulty in diagnosing pneumoconiosis and the tendency to confuse it with tuberculosis, these claims were studied individually. The physicians who had treated the individuals were interviewed, and hospital and sanatorium records, including available X-ray films, were investigated with the following results.

Death claims:

1. Proved to be a typist, not exposed to dust; died of pulmonary and intestinal tuberculosis.

2. Colored male age 27; worked in asbestos one year and eight months; died of pulmonary tuberculosis following a hemorrhage; was in sanatorium ten months. Also had a four-plus Wassermann. No evidence of asbestosis.

3. Colored male age 32; worked in asbestos plant one year and seven months; died of pulmonary tuberculosis after one month in hospital. Cavitation both lungs; no evidence of asbestosis.

4. Colored male age 34; worked in asbestos plant two years, and six months. His physician believes this was a case of uncomplicated tuberculosis. Was not inmate of hospital or sanatorium.

No information could be obtained of the other two cases except from the death certificates which gave pulmonary tuberculosis as the cause of death.

Total and permanent disability claims:

1. Male, employed in asbestos plant three years. His physician states he first came under his observation with an old established case of tuberculosis about two years after asbestos employment started. Also had tuberculosis of the kidney and cervical glands.

2. Male, employed in asbestos plant eight months. His physician states old fibroid tuberculosis with tubercle bacilli in sputum. No X-ray available, but according to physician asbestos bodies were found in sputum.

3. Male, ten years employment. Two physicians who treated him at different times are now inclined to believe this man is not tuberculous, but has asbestosis.

4. White male, was re-examined at time of investigation. His physician reports well nourished, husky looking, good color, no cyanosis; no clubbing of fingers, diminished expansion, incessant cough; X-ray shows fine mottling disseminated through both lungs. No evidence of tuberculosis. Probably a second stage asbestosis.

5. White male, thirteen years in asbestos plant. Is now in sanatorium. An interesting case. His physician states he has extensive asbestosis and pulmonary tuberculosis; cavity in right lung; sputum loaded with tubercle bacilli and asbestos bodies; believes tuberculosis long antedated asbestosis. This patient is progressing in a satisfactory manner.

It was not possible to locate the other three cases of total and permanent disability who had been diagnosed as having pulmonary tuberculosis. On the basis of the information obtained the death claims appear to be due to uncomplicated tuberculosis; three of them were Negroes. It was more than probable that these men were tuberculous at the time their employment in the asbestos plant commenced.

Of the eight disability claim cases, one was uncomplicated tuberculosis and two were uncomplicated asbestosis who were put on disability because of a mistaken diagnosis of tuberculosis. In this same community we know of one death due to uncomplicated asbestosis in an individual with many years employment in the industry.*

CONCLUSIONS

1. Prolonged exposure to asbestos dust causes a pulmonary fibrosis of a definite type distinct from silicosis demonstrable on X-ray films. Clinically, it is of a type milder than silicosis.

2. There is associated with asbestosis in a large percentage of cases definite cardiac enlargement.

3. Asbestosis does not involve an unusual tendency to pulmonary tuberculosis.

*Personal communication. Details not yet published.

4. Asbestosis as observed in this series of cases does not cause any marked degree of disability. However, it is possible for uncomplicated asbestosis to result fatally.

5. It is not known how much asbestosis may add to the mortality of pneumonia and acute pulmonary infections other than tuberculosis.

6. It is not practicable as yet to establish standards for the dust content of air and in view of the many low dust counts in this report, compared with the U.S. Public Health Service standard for silica dust, it is possible that asbestos may cause pneumoconiosis more readily than free silica (SiO_2).

7. The amount of dust in the air in the asbestos plants studied can be substantially reduced.

RECOMMENDATIONS

1. That the industry seriously face the problem of dust control in asbestos plants.

2. That new employees be examined physically and rejected for employment if they show tuberculosis or pneumoconiosis.

3. That employees be examined physically every year or certainly every two years, this examination to include an X-ray examination of the chest.

4. That the industry sponsor studies on known cases of asbestosis, as well as studies on effects of asbestosis on the heart and circulation.

University of Toronto Library Services and

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December 10, 1934.

Dr. A. J. Lanza,
Metropolitan Life Insurance Co.,
1 Madison Avenue,
New York City.

Re: Preliminary Study of
Asbestos Dust Effects

Dear Dr. Lanza:

I have examined the galley proof of yours and Dr. McConnell's preliminary study entitled "Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers", which you were good enough to send me with your letter of December 7th, and have compared it with the mimeographed pamphlet of which it purports to be a condensation.

Confirming my telephone conversation of this afternoon, I am planning to transmit the proof sheets to Mr. Hobart so that he will receive them tomorrow and shall request that he return them to me not later than Thursday with his comments, after which I shall see that they are promptly transmitted to you. I shall also inquire of the Raybestos Company whether or not they desire to examine the proof of the article before its final printing and shall endeavor to persuade them not to insist upon such an examination.

I have the following comments to make:

(1) Conclusion No. 1 (galley 14) has omitted a sentence appearing in the original which, as modified by you, read as follows: "Clinically, from this study it appeared to be of a type milder than silicosis".

(2) There is also omitted the first paragraph on page 15 of the original report which reads as follows:

"During the progress of the study, physicians who were practicing in the communities where asbestos workers lived, were questioned, and stated that they did not find an unusual amount of tuberculosis among these workers. The contrast between this state of affairs and that found in communities with

a silicosis hazard, is noteworthy".

Both the foregoing observations included in your original report presented an aspect of your survey that was favorable to the industry and we should like to see them retained.

(3) On galley 14 the word "death" has been omitted after the phrase "6 of 36".

(4) Conclusion No. 3 (galley 14). The word "preliminary" is inserted — to my mind unnecessarily — since the entire article is definitely labelled at the commencement as a "preliminary" study.

(5) Referring to the last sentence of the third paragraph on page 13 (which is not changed in the galley proof), it seems to me that some sort of additional phrase or clause is required in order to relate it properly to what has gone before. As it stands now, it is not entirely clear just what one is expected to deduce therefrom.

Very truly yours

Vandiver Brown
Attorney

VB:T

CC: Messrs. George S. Hobart
E.L. Voorhees
S.L. Williams

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HOBART & MINARD

GEORGE S. HOBART
DUANE E. MINARD
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AUGUSTINE J. KELLY
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G. ADDISON HOBART

LEFCOURT BUILDING
1180 RAYMOND BOULEVARD
NEWARK, NEW JERSEY

TELEPHONE
MARKET
2-8184
2-8185
2-8186

Dec. 15, 1934.

Re preliminary study of Metropolitan Life Insurance Company

- - - - - - - - - - - - - - - - - - -

Mr. Vandiver Brown,
Attorney, Johns-Manville Corporation,
22 E. 40th Street,
New York City.

Dear Mr. Brown:

I received your letter of December 10th with enclosures, including proof of the preliminary study entitled, "Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers". I have carefully examined the proof and return same.

In reading this proof I have compared it with the paper of the same title which was prepared by the Industrial Health Service of the Metropolitan about three years ago. I submit the following suggestions (For convenience of reference I have numbered my comments in red pencil in the margin of the several pages of the galley proofs):

(1) Gal. 11. At the end of the paragraph which discusses the "variables" upon which the progress of the disease is influenced, the following sentence appears: "It might well be assumed that similar variables would influence the occurrence of asbestosis." You will recall that in dealing with the New Jersey Legislative Commission which has had charge of the proposed new legislation in New Jersey whereby silicosis is to be added to the list of occupational diseases for which compensation is required under the terms of the compensation statute, we have consistently urged that there is a substantial difference between silicosis and asbestosis — both as to the clinical nature of the disease and as to the reasonable probability of its incidence; and you will also recall that in particular, we have urged that asbestosis should not at the present time be included in the list of compensation diseases, for the reason that it is only within a comparatively recent time that asbestosis has been recognized by the medical and scientific professions as a disease — in fact one of our principal defenses in actions against the company on the common law theory of negligence has been that the scientific and medical knowledge has been insufficient until a very recent period to place upon the owners of plants or factories the burden or duty of taking special precautions against the possible onset of the disease to their employees.

I, therefore, dislike to have this report suggest that asbestosis might be assumed to be 'similar' to silicosis, either as to the period

of time within which it might develop, or as to the dosage received by the lungs, or as to the quantity of dust, etc.

(2) Gal. 11. At the bottom of this galley I observe that there is set forth in a note (instead of in the body of the article) the statement that appears in the Report of 1931 relative to the enlargement of the particles and the use of logarithms. (See p. 7 of Report of 1931) As a matter of curiosity I wonder why this statement (which seems to be quite important) is put in a note instead of in the body of the article. If the authors prefer to have it in the form of a note, of course, their judgment should control, as it has no particular bearing in any event upon the points in which the company is interested.

(3) Gal. 12. The last paragraph on this galley has a summary of the dust counts with respect to the several plants. I notice that it is stated that plant 3 "was unnecessarily dusty". I understand this does not in any way refer to any plant of Johns-Manville, but I am wondering whether the proprietor of plant 3 has had an opportunity to review the comment. It strikes me that the report would serve every purpose if we leave out in the first line of the paragraph on that galley the words "was unnecessarily dusty". However, that is a matter with which we are indirectly concerned and I merely suggest it for consideration It is the same expression which appears in the Report of 1931 at page 11.

Galley 13. No comment.

(4) Gal. 14. The word "death" should be inserted after the words "3 of 36" in the third paragraph of subtitle "Insurance Claims". You have already called attention to this in your letter of December 10 to Dr. Lanza.

(5) Gal. 14 under subtitle "Conclusions".

No. 1. I agree with your suggestion in letter to Dr. Lanza of December 10th that there should be added to this conclusion a statement similar to that which appeared (at p. 20) in the Report of 1931, namely, "clinically, it is of a type milder than silicosis" — or, if the doctors do not want to make the statement in that positive form, it might be as follows (as suggested in your letter): "clinically, from this study it appeared to be of a type milder than silicosis"; or, if that is too strong, it might be set forth as follows: "so far as indicated by the study, asbestosis is of a type (or appears to be of a type) milder than silicosis". The exact wording is not important, except that I think it would be desirable (if the doctors are satisfied that such is the fact), to have some expression comparing asbestosis with silicosis and indicating that there is a real difference between the two diseases, so far as scientific knowledge now indicates. Of course, here again I have in mind the probability that the New Jersey Commission will introduce a bill in the legislature which opens in January of 1935; and if it is the policy of Johns-Manville to oppose any bill that attempted to include asbestosis, as compensable, it would be very helpful to have an official report to show that there is a substantial difference between asbestosis and silicosis; and by the same token it would be troublesome if an

I have the honour to be, Sir,

Your most obedient humble servant,

(9)

No. 7. This is the answer to letter No. 6.

Sir,

I have the honour to inform you that I have received your letter of 15th instant, and I do not understand what the statement/conclusion is which you say I have arrived at.

As

No. 8. This letter is a request for information.

No. 9. This is the answer to letter No. 8.

No. 10. This is a letter of complaint.

Hobart & Minard

Lefcourt Building
1180 Raymond Boulevard
Newark, New Jersey

Dec. 15, 1934.

Re preliminary study of Metropolitan
Life Insurance Company

Mr. Vandiver Brown,
Attorney, Johns-Manville Corporation,
22 E. 40th Street,
New York City.

Dear Mr. Brown:

I received your letter of December 10th with enclosures, including proof of the preliminary study entited, "Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers". I have carefully examined the proof and return same.

In reading this proof I have compared it with the paper of the same title which was prepared by the Industrial Health Service of the Metropolitan about three years ago. I submit the following suggestion (for convenience of reference I have numbered my comments in red pencil in the margin of the several pages of the galley proofs):

(1) Gal. II. At the end of the paragraph which discusses the "variables" upon which the progress of the disease is influenced, the following sentence appears: "It might well be assumed that similar variables would influence the occurrence of asbestosis." You will recall that in dealing with the New Jersey Legislative Commission which has had charge of the proposed new legislation in New Jersey whereby silicosis is to be added to the list of occupational diseases for which compensation is required under the terms of the compensation statute, we have consistently agreed that there is a substantial difference between silicosis and asbestosis -- both as to the clinical nature of the disease and as to the reasonable probability of its incidence, and you will also recall that in particular we have urged that asbestosis should not at the present time be included in the list of compensation diseases, for the reason that it is only within a comparatively recent time that asbestosis has been recognized by the medical and scientific professions as a disease -- in fact one of our principal defenses in actions against the company on the common law theory of negligence has been that the scientific and medical knowledge has been insufficient until a very recent period to place upon the owners of plants or factories the burden or duty of taking special precautions against the possible onset of the disease to their employees.

I, therefore, dislike to have this report suggest that asbestosis

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might be assumed to be similar to silicosis, either as to the period of time within which it might develop, or as to the dosage received by the lungs, or as to the quantity of dust, etc.

(2) Gal. 11. At the bottom of this galley I observe that there is set forth in a note (instead of in the body of the article) the statement that appears in the Report of 1931 relating to the enlargement of the particles and the use of logarithum (See p. 7 of Report of 1931) As a matter of curiosity I wonder why this statement (which seems to be quite important) is put in a note instead of in the body of the article. If the authors prepfer to have it in the form of a note, of course, their judgment should control, as it has no particular bearing in any event upon the points in which the company is interested.

(3) Gal. 12. The last paragraph on this galley has a summary of the dust counts with respect to the several plants. I notice that it is stated that plant E "was unnecessarily dusty". I understand this does not in any way refer to any plant of Johns-Manville, but I am wondering whether the proprietor of plant E has had an opportunity to review the comment. It strikes me that the report would serve every purpose if we leave out in the first line of the paragraph on that galley the words "was unnecessarily dusty".

Galley 13. No comment.

(4) Gal. 14. The word "death" should be inserted after the words "6 of 36" in the third paragraph of subtitle "Insurance Claims". You have already called attention to this in your letter of December 10 to Dr. Lanza.

(5) Gal. 14 under subtitle "Conclusions".

No. 1. I agree with your suggestions in letter to Dr. Lanza of December 10th that there should be added to this conclusion a statement similar to that which appeared (at p.30) in the Report of 1931, namely, "Clinically, it is of a type milder than silicosis" -- or, if the doctors do not want to make the statement in that positive form, it might be as follows (an suggested in your letter): "Clinically, from this study it appeared to be of a type milder than silicosis"; or, if that is too strong, it might be set forth as follows: "So far as indicated by the study, asbestosis is of a type (or appears to be of a type) milder than silicosis". The exact wording is not important, except that I think it would be desirable (if the doctors are satisfied that such is the fact) except that I think it would be very helpful to have an official report to show that there is a substantial difference between asbestosis and silicosis; and by the same token it would be troublesome if an official report should appear from which the conclusion might be drawn that there is very little if any difference between the two diseases.

official report should appear from which the conclusion might be drawn that there is very little if any difference between the two diseases.

No. 2. I understand that the statement as to the cardiac enlargement is correct from a scientific standpoint, and I do not see how we can consistently object to it.

No. 3. I agree that the word "preliminary" is superfluous.

No. 4. O. I.

No. 5. This reads the same as on page 21 of the Report of 1931 and, hence, it may be a little late to object. However, I point out that as No. 3 now reads the inference might be drawn that asbestosis does add to the mortality of tuberculosis cases. But that inference does not seem to be justified from this study. I am wondering if it would be a more accurate scientific statement if there were omitted from No. 5 the last three words, to-wit "other than tuberculosis".

No. 6. This is the same as set forth on page 21 of Report of 1931, with the exception that the Report of 1931 at the end of the sentence has the words "than free silica (3102)" and the galley proof has the words "than granite dust". Hear again it may be a little late to criticize the statement, but I do not like the suggestion that there is a possiblility that asbestosis may cause pneumoconiosis more readily than granite dust. The reason I object is that many cases of silicosis are supposed to originate in granite dust and this statement as it now reads suggests that a possibility that a pneumoconiosis may arise more readily from asbhestos, than granite dust. I think we should, if possible, eliminate all that part of corelosion No. 6 following the semi-colon after the words "of air" in line 3 of the conclusion.

No. 7. This is the same as in the Report of 1931. To comment.

(6) Gal. 14 and 15 subtitle "Recommendations".

In my copy of the Report of 1931 I have a pencil memo. as to recommendation No. 4 to insert the word "possible" before the word "effects" so as to have it read "as well as studies on the possible effects of asbestosis on the heart and circulation". I do not think the point is of much importance, but I call attention to it so that it is not overlooked.

I agree with the other suggestions in your letter to Dr. Lanza of December 10th.

Very truly yours,

GMH/HF
Re-typed 2/14/95

Bridgeport, Conn.

Jan. 2, 1935.

Mr. Vandiver Brown, Attorney,
Johns-Manville Corporation,
New York City.

Dear Mr. Brown:

Thank you for your letter of Dec. 24.

Both Mr. Simpson and myself feel quite safe in leaving the approval of the report in your hands. I do think that Mr. Hobart made some excellent suggestions in his letter of Dec. 15, and hope that Dr. Lanza will give a lot of consideration to the comments.

I take it for granted that when this article comes off the press, the Metropolitan Insurance Company will see that you get copies of it, in which event, we certainly would like one for our files.

Yours very truly,

Secretary.

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PUBLIC HEALTH REPORTS

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EFFECTS OF THE INHALATION OF ASBESTOS DUST ON THE LUNGS OF ASBESTOS WORKERS

A Preliminary Study

By A. J. Lanza, *Assistant Medical Director*, William J. McConnell, *Assistant Medical Director*, and J. William Fehnel, *Chemist, Metropolitan Life Insurance Co.*

INTRODUCTION

In 1929 the Metropolitan Life Insurance Co. was approached by officials representing the asbestos industry in the United States, who were desirous of ascertaining whether asbestos dust was an occupational hazard in their establishments and, if so, what was the nature of this hazard and what should be done to prevent or control it.

About this time several articles had appeared in English medical journals describing a pneumoconiosis due to asbestos dust. While in one or two isolated instances the occurrence of this type of pneumoconiosis had been described in American journals, the industry itself appeared to be quite uninformed of the existence of any such occupational disease.

The hazard of silica dust, with special reference to the lungs, has long been appreciated, and a great deal of study and research has been applied to the problem (in the metal mining and certain other industries) in Great Britain, the United States, the British Dominions, and in other countries. The nature of the effects of silica dust expressed in the term "silicosis", with the resultant extraordinary predisposition to pulmonary tuberculosis, is well known. These effects have been associated with the inhalation of dust containing free silica in varying amounts. The effects upon the pulmonary tissue of dusts containing combined silica—silicates—are still a fertile field for investigation, but evidence is accumulating that certain of these dusts produce pathological results quite distinct from true silicosis.

The name "asbestosis" has been applied to the pneumoconiosis caused by asbestos dust and it will be so used in this report. Chemically, the asbestos of commerce is a hydrated magnesium silicate consisting primarily of silica (combined silica) 44.1 percent, magnesia 43 percent, and water 12.9 percent, while ferrous iron and nickel are present in small quantities. This commercial variety of

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prevent the solution of some of the dusts, particularly any silica which might be present. The impinger was actuated by an electrically driven rotary pump and the rate of air sampled was measured by means of a resistance type of flow meter with an inclined manometer for measuring the pressure difference. Each sample represents the dust collected from a volume of 100 cubic feet of air.

The electric precipitator is designed upon the Cottrell precipitator principle used for recovering dusts and fumes commercially. This principle depends on electrifying the dust particles by making them pass through an electrostatic field and thus causing them to settle out upon a sheet of celluloid. The electrostatic field is set up by means of a transformer operating from the lighting lines on 120 volts, alternating current. Air is drawn through the apparatus by a small rotary fan, run by a motor, the quantity being measured by a flow meter.

Samples of dust brushed from beams and pipe lines near the ceiling also were secured for chemical analysis. All samples were shipped to the industrial hygiene laboratory of the Metropolitan Co., and the dust counts were made according to the methods accepted by the United States Bureau of Mines and the United States Public Health Service (1). Particles in size up to 360 microns in the greatest diameter were counted.

Chemical analyses were made for total (free and combined) silica according to the accepted standard method of Hillebrand (3).

In the first plant studied (A) only particles 10 microns and under in size were counted. It has been demonstrated repeatedly that no silica particles exceeding 10 microns (a micron equals one-millionth part of a meter or one-thousandth part of a millimeter) in greatest diameter enter the lung tissu ; for this reason, the larger particles usually are not considered when determining dust counts. Later, as it appeared from some of the published articles (4) (English) that asbestos particles much larger in size were found in lung tissue, it was decided to count all particles and make differential counts of all those 10 microns and under.[1]

Total particles per cubic foot and the corresponding weight of the dust in milligrams were obtained. Since no relationship between the dust counts and the corresponding weights was found, the weights are not included in the tabulations in this report.

Table 1 shows by plants and by departments the maximum and minimum dust counts in million particles per cubic foot of air. Although the counts in plant A are of particles 10 microns and under,

[1] Samples collected by both the impinger and the electric precipitator were counted. In addition, microphotographs of these dust particles were enlarged by lenses projected upon a screen with a projectile projector, the enlarged particles being measured with a ruler. Knowing the latter enlargement (by actual measurement), it was easy to calculate the original particle size (D). By the use of Hazen's (5) logarithmic probability paper, the logarithms of the function to be measured (in this case microns) are plotted as ordinates against the probability of occurrence as abscissas.

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and in the other plants are for all particles, direct comparison is possible, as in no sample taken were the number of particles 10 microns and under less than 94 percent of the whole.

TABLE 1.—*Maximum and minimum dust counts in million particles per cubic foot of air, by plant and department*

Department	Plant A		Plant B		Plant C		Plant D		Plant E	
	Number of samples taken	Million particles under 10μ per cubic foot of air	Number of samples taken	Million particles per cubic foot of air	Number of samples taken	Million particles per cubic foot of air	Number of samples taken	Million particles per cubic foot of air	Number of samples per cubic foot of air	Million particles per cubic foot of air
Preparation	3	½–11½	9	½–6	6	2 –23			3	23 –42
Card room	3	½–14½	8	½–9½	6	3½–13½			3	11 –26
Ring frame spinning room	15	½– 1½								
Mule spinning		½–2½	3	½–1	3	1½– 4½			3	3½–10
Twisting		½–1½		(1)	6	1 – 3½			3	7½– 7
Weaving		½–2	4	½– 1½	6	3½–10	13	1 –7	3	10½–10½
Felt department	2	½–1½								
Sheet insulating		½–1								
Molded brake lining and clutch							2	½– 1½		

[1] Included in spinning. [2] Including loomed lower weaving.

Whereas the dust in the preparation room is practically all due to asbestos, the contrary is the case in the other departments. In the carding, spinning, and weaving rooms asbestos comprises about 25 percent of the material used. On special jobs the percentage of asbestos may be higher, but in general the figure quoted is approximately correct, the other material consisting principally of cotton. In the insulating departments of plant A, asbestos is but 5 percent of the total material used. In the molded brake-lining and clutch division of plant D, asbestos comprises about 25 percent of the total material. The actual exposure to asbestos dust, therefore, is considerably less than is indicated in the dust counts in table 1.

CONDITIONS IN THE PLANTS

The processes in these plants were very similar to those in cotton mills in general. For illustration, the process in the preparation room of plant B is here detailed at length:

Asbestos is received in bags. These are emptied upon the floor and the asbestos is shoveled into pug mills and run for about 3 minutes in order to crush and open the fibers. From there the asbestos is shoveled into trucks and wheeled to hoppers with either horizontal or vertical openers, similar to those used in textile mills. After passing through the opener, the material is discharged into trucks. Waste manufacturing material is first run through a garnet machine and

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discharged into trucks, which are wheeled to the openers, and the material is fed into these in the same manner as is the new material. After being discharged from the opener, the material is put onto a vibrating or shaking screen, where the long fibers are picked off by a suction hood and blown into a bin to be used again in textiles. The short fibers falling through the screen are either sold as such or used in making an asbestos cement.

The cotton is received in bales, opened, and also run through vertical openers. It is discharged into trucks. The filled trucks from the vertical and horizontal openers containing either the asbestos fiber, the cotton, or the waste material are taken to separate storage bins. As needed, these materials are weighed and dumped onto the floor in proportion to the mixture desired, and the resulting mixed materials are then passed through a mixing picker. As this mixture is discharged from the picker, it is sprayed with a light mineral oil. The material is then taken by a mechanical conveyor to the storage bin in the card room. Two of these mixing pickers discharge onto this conveyor system, while a third machine is equipped with a suction device which conveys the material to a storage bin in the card room. The object of the two systems is to facilitate the handling of two grades of material at the same time. The third machine is not equipped with an oil spray. The object of the oil spray is primarily to entrap the small asbestos fibers and hold them enmeshed in the cotton fiber throughout the processes of carding, spinning, and weaving. Incidentally, it is apparent that it also diminishes the amount of dust. The material is saturated with about 4 percent of oil at this point; but by the time it reaches the looms, the oil had diminished to less than 1 percent.

While mineral oil was used in the preparation room of plant C, it apparently was not as efficacious as in plant B. In plant E oil was not used, nor was there any attempt at humidification or any system of dust exhausting. Carding machines were fed by hand. In plant A there was a humidifying, ventilating, and heating system, while plant B depended on natural humidity. In plant E the carding machines were equipped with a dust-exhaust system, but it was not efficiently used. In plant C carding was done in 2 buildings, 1 of which was equipped with an air-conditioning system, and in both buildings the machines had exhaust equipment.

Two plants, B and C, had artificial humidity installations in their spinning rooms. In the former the temperature was 76° F., relative humidity 75 percent. One plant, C, had a humidification system in the twisting department and also in the weaving department, where the relative humidity was 75 percent with a dry-bulb temperature of 69° F., as compared with a relative humidity of 44 percent in the weaving room of plant A.

Aside from plant E, it would appear that the dust hazard was not excessive except in the preparation rooms of plants B and C. The dust counts are interesting, too, when considered in the light of the permissible standard for granite dust, established by the United States Public Health Service (7), namely, about 10 million particles (10 microns and under) per cubic foot. However, we are not justified in assuming that because available information suggests that asbestosis is a milder disease clinically than silicosis, the threshold of permissible dust counts is higher. Asbestosis appears to be pathologically different from silicosis, and the experience so far does not warrant an attempt to define a standard of dustiness for asbestos dust.

DUST CONTROL

Various measures, such as oiling, humidification, and local exhausts, tended to reduce the dust. Nevertheless, it was evident that they were only partly successful. If it is expected to control dustiness in these plants, final reliance must rest upon properly constructed exhaust equipment. In plant C in one department an experimental installation was set up. In spite of some obvious faults, this equipment, on the basis of comparative dust counts, reduced the dust by 30 percent and with further alterations will probably be 75 percent effective. In this case such a reduction seemed quite satisfactory. It is neither practicable nor economically desirable to install such equipment as will make the air entirely dust free. The normal defensive mechanism of the body takes care of a fair amount of atmospheric pollution. It is when the body is exposed to an excessive amount of dust that this defense mechanism breaks down.

The application of exhaust equipment to textile machinery involves considerable difficulty, especially where the construction of the plant is such that it is not possible to apply down-draft suction to the looms.

It is desirable to install exhaust apparatus in any plant on an experimental basis first, and then check its efficiency by dust counts. This practice will result in saving a needless expenditure of money.

PHYSICAL EXAMINATIONS

X-ray films were made of 128 persons (103 men, 18 women) working in asbestos plants in the United States. All but five of these were given physical examination. The cases were selected more or less at random from among those having more than 3 years of employment in the industry. It was soon obvious that the early diagnosis of asbestosis must rest to a large extent on X-ray pictures of the chest. As in the early stages of silicosis, the clinical symptoms of the asbestos workers were usually indefinite and inconclusive. The interpretations of these films are based on the readings of one competent roentgenologist, but they have been reviewed by several

others experienced in this field. The differences of opinion were of a minor nature, and there was general agreement as to interpretation. The films were read conservatively, taking into account the physical examination and the age of the individual, and were classed as positive only when there was no major disagreement. All examiners were guided by their experience in silicosis and other types of pneumoconioses. The films classed as negative for asbestosis were further subdivided into doubtful and negative. Only time can tell whether the individuals classed as doubtful are progressing toward a definite asbestosis. Particular attention was focussed on the presence or absence of indications of tuberculosis, and in this respect all the reviewers of the films were in accord. With the unhappy experience of silicosis in mind, it was felt that a great deal of care should be devoted to ascertaining, if possible, whether or not asbestosis predisposes to tuberculous infection.

The cases of asbestosis were divided into two classes, first stage and second stage. The first stage embraces those who show by X-ray examination definite lung pathology sufficient in extent to warrant a diagnosis of pneumoconiosis, but who have no definite symptoms. The second-stage cases exhibit more extensive lung pathology and also definite symptoms.

All these individuals [2] were actively engaged in factory work, and it was not practicable to make any distinction on the basis of working ability or disability. Had any individuals been found who exhibited extensive pulmonary involvement and marked physical disability or total disability, they would have been classified as third stage, but no such cases were found.

Of the total of 126 X-ray examinations, 4 were diagnosed as second-degree asbestosis, 63 as first degree, 39 as doubtful, and 20 as negative.

There is a definite increase in the percentage diagnosed as positive in relation to the years of exposure, as shown in table 2. Small numbers make it impossible to determine how far the factor of age enters into this increase.

TABLE 2.—*Classification of cases by years of exposure*

Years of exposure	Percentage positive	Number		
		Positive	Doubtful	Negative
Over 15 years	[illegible]	[illegible]	[illegible]	[illegible]
10 to 15 years	[illegible]	[illegible]	[illegible]	[illegible]
5 to 10 years	[illegible]	[illegible]	[illegible]	[illegible]
Under 5 years	[illegible]	[illegible]	[illegible]	[illegible]
Total examined	[illegible]	[illegible]	[illegible]	[illegible]

[1] Part time. [2] 1 not continuous.

[1] One asbestos worker (second stage) retired of his own accord, because of old age, 13 years previous to this study. But is still active about the factory.

Of the 64 persons given physical examination and diagnosed as having positive asbestosis, only 8 were entirely free from symptoms, while 10 out of 37 with doubtful and 7 out of 20 with negative diagnoses were free from symptoms. Dyspnoea and cough were the symptoms most complained of, but none of these cases exhibited the urgent or evident type of "short wind" seen in true silicosis. Several of those classed as negative stated that they were "short winded" and were so recorded, but too much emphasis should not be placed on statements of subjective symptoms. During the progress of the study, physicians who were practicing in the communities where asbestos workers lived were questioned and stated that they did not find an unusual amount of tuberculosis among these workers. The contrast between this state of affairs and that found in a community with a silicosis hazard is noteworthy.

The incidence of tuberculosis (based upon X-ray films) is given in table 3; 40 of the 67 examined had less than 15 years of employment in the industry.

TABLE 3.—*Incidence of tuberculosis*

	Positive	Doubtful	Negative
Tuberculosis (arrested)	[illegible]	[illegible]	[illegible]
Tuberculosis (active)	[illegible]	[illegible]	[illegible]
Total examined	[illegible]	[illegible]	[illegible]

[illegible] This case was diagnosed as probably active on the X-ray findings.

Table 4 gives information as to physical signs of chest trouble.

TABLE 4.—*Chest findings*

	Positive	Doubtful	Negative
Physical signs of chest trouble	[illegible]	[illegible]	[illegible]
No physical signs	[illegible]	[illegible]	[illegible]
Not examined	[illegible]	[illegible]	[illegible]
Total	[illegible]	[illegible]	[illegible]

[illegible] At least 4 of these showed an evidence associated with tuberculosis.
[illegible] 2 of these not associated with signs found with tuberculosis.
[illegible] 6 of these not associated with signs of tuberculosis.

What significance, if any, can be attached to the presence of "asbestos corns" on the hands of workers appears doubtful, as 18 percent of the positives (12 out of 64) and 15 percent of the others (doubtful, 7 out of 37, negative, 2 out of 20) showed such corns.

Each roentgenologist who reviewed the X-ray films called attention to the fact that these films indicated a very unusual incidence of enlargement of the heart. It is probable that this is a compensatory enlargement due to the additional work put upon the heart in efforts

to pump blood through the fibrosed lungs. It is possible that not sufficient attention has been paid to the effects of the pneumoconioses upon the heart.

Many workers had changed from one department to another and from one plant to another during their years of employment in the asbestos industry. Since only the amounts of dust collected at the time of this survey in the various departments are known, there is no way of knowing the average amount of dust in the atmosphere inhaled by these people over the years of their employment. Consequently, it is not possible to correlate individual cases with definite amounts of dust exposure.

INSURANCE CLAIMS

To throw light on the relationship between asbestosis and pulmonary tuberculosis, an analysis of death claims and total and permanent disability claims was made in regard to companies carrying group insurance and having available figures.

There were 2,039 lives involved, with a total exposure of 7,019 life-years. The death claims are so small in number that reliable conclusions cannot be reached from any subdivision of the figures. The same is true of the sickness claims under health insurance. The number of claims for respiratory diseases is high in two of the plants studied, but low for the others, as compared with the Metropolitan experience of 1927. However, during the latter part of 1928 and the first part of 1929 there was an epidemic of influenza.

The records of one establishment (plant B) showed that 6 of 36 death claims and 3 of 10 permanent and total disability claims were listed as due to pulmonary tuberculosis. This appeared to be an inordinate amount of tuberculosis from this plant. However, many of the employees were Negroes, and also tuberculosis claims are generally high in this section of the country. Realizing the difficulty in diagnosing pneumoconiosis and the tendency to confuse it with tuberculosis, these claims were studied individually. The physicians who had treated the individuals were interviewed, and the hospital and sanatorium records, including available X-ray films, were investigated, with the following results:

Death claims:

1. A typist, not exposed to dust; died of pulmonary and intestinal tuberculosis.
2. Colored male, age 27; worked in asbestos 1 year and 8 months; died of pulmonary tuberculosis following a hemorrhage; was in sanatorium 10 months. Also had a four plus Wassermann. No evidence of asbestosis.
3. Colored male, age 32; worked in asbestos plant 1 year and 7 months; died of pulmonary tuberculosis after 1 month in hospital. Cavitation both lungs; no evidence of asbestosis.

4. Colored male, age 34; worked in asbestos plant 2 years and 6 months. His physician believes this was a case of uncomplicated tuberculosis. Was not inmate of hospital or sanatorium. No information could be obtained on the other two cases.

Total and permanent disability claims:

1. Male, employed in asbestos plant 3 years. His physician states that he first came under his observation with an old established case of tuberculosis about 2 years after asbestos employment started. Also had tuberculosis of the kidney and cervical glands.

2. Male, employed in asbestos plant 8 months. His physician states finding of old fibroid tuberculosis with tubercle bacilli in sputum. No X-ray available; but according to physician, asbestos bodies were found in sputum.

3. Male, 10 years' employment. Two physicians who treated him at different times are now inclined to believe this man is not tuberculous, but has asbestosis.

4. White male, was reexamined at time of investigation. His physician reports well nourished, husky looking, good color, no cyanosis; no clubbing of fingers; diminished expansion; incessant cough; X-ray shows fine mottling disseminated through both lungs. No evidence of tuberculosis. Probably a second stage asbestosis.

5. White male, 13 years in asbestos plant. Is now in sanatorium. An interesting case. His physician states that he has extensive asbestosis and pulmonary tuberculosis; cavity in right lung; sputum loaded with tubercle bacilli and asbestos bodies; believes tuberculosis long antedated asbestosis. This patient is progressing in a satisfactory manner.

It was not possible to locate the other three cases of total and permanent disability who had been diagnosed as having pulmonary tuberculosis. On the basis of the information obtained, the deaths in death-claims cases appear to be due to uncomplicated tuberculosis; three of them were Negroes, who were probably tuberculous at the time their employment in the asbestos plant commenced.

Of the 8 disability claim cases, 1 was uncomplicated tuberculosis and 2 were uncomplicated asbestosis who were put on disability because of a mistaken diagnosis of tuberculosis. In this same community we know of one death due to uncomplicated asbestosis in an individual with many years' employment in the industry.[1]

CONCLUSIONS

1. Prolonged exposure to asbestos dust caused a pulmonary fibrosis of a type different from silicosis and demonstrable on X-ray films. Clinically, from this study, it appears to be of a type milder than silicosis.

2. Cases of definite cardiac enlargement were frequently found to be associated with asbestosis.

[1] Personal communication.

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3. A predisposition to tuberculosis due to asbestos dust was not indicated in this study.

4. Asbestosis as observed in this series of cases had not resulted in marked disability in any case.

5. It is not known how much asbestosis may add to the mortality of pneumonia and acute nontuberculous pulmonary infections.

6. It is not practicable as yet to establish standards for the asbestos dust content of air.

7. The amount of dust in the air in the asbestos plants studied can be substantially reduced.

RECOMMENDATIONS

It is recommended—

1. That the industry seriously face the problem of dust control in asbestos plants.

2. That new employees be examined physically, including X-ray examination of the chest, and rejected for employment if they show tuberculosis or pneumoconiosis.

3. That employees be examined physically, preferably every year, but at least every 2 years, this examination to include an X-ray examination of the chest.

4. That the industry sponsor studies on known cases of asbestosis, as well as studies on effects of asbestosis on the heart and circulation.

ACKNOWLEDGMENTS

The authors wish to express their sincere thanks to all those who aided in making this study possible, especially the officials and employees of the asbestos companies who cooperated to the fullest extent and gave every facility for securing data; also to Dr. Pancoast, of the University of Pennsylvania, for his interest and valuable advice in interpreting X-ray films, and to Dr. F. F. Meriwether, of the United States Public Health Service, surgeon in charge of the United States Bureau of Mines Cooperative Clinic in Picher, Okla., whose interpretation of all the X-ray films listed in this report is followed. Appreciation is also expressed to Dr. W. Atmar Smith, and Dr. Kenneth Lynch, of Charleston, S. C., for information and advice, particularly as to the pathology of asbestosis;[1] to Dr. W. W. Wild, of Charleston, S. C.; to Dr. Joseph H. Wyatt, of Newark, N. J., and Dr. H. H. Fellows, of New York, for assisting in interpreting X-ray films; to Dr. Paul O. Snoke, of Lancaster, Pa.; to Dr. R. H. Stevenson, of Danville, Quebec, Canada; and to Dr. L. U. Gardner and Mr. Donald E. Cummings, of the Saranac Laboratory, who have undertaken the study of the pathological effects of the inhalation of asbestos dust

[1] Asbestosis bodies in sputum and lung. By Kenneth M. Lynch, M. D., and W. Atmar Smith, M. D. Jour. Am. Med. Assoc., Aug. 31, 1929, vol. 93, no. 9, pp. 635-641.

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through animal experimentation, with the aid of a grant from the Metropolitan Life Insurance Co.

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ENDEMIC TYPHUS IN ALABAMA[1]

By J. N. Baker, M. D., James G. McAllister, Ph. D., and D. G. Gill, M. D., D. P. H., *Alabama State Department of Health, Montgomery, Ala.*

I. INTRODUCTION

In a preliminary report made in June of this year before the Conference of State and Provincial Health Authorities, held in Washington, the authors discussed the epidemiological aspects of endemic typhus as it occurs in southern United States. In that report it was noted that there had been a rapid increase in this disease in certain Southern States, as is shown in table 1. That other countries have experienced a similar rise in incidence is evidenced by the figures in table 2.

The distinction between epidemic typhus and the endemic typhus of this country was first recognized in 1898 by Brill. He (1) found in the United States a type of fever which, resembling typhoid, gave a negative Widal reaction. In further studies he (2, 3) demonstrated its similarity to typhus, but showed that it was milder in character and less contagious, only one case as a rule being found in a household. Also he reported that it was most prevalent during the fall of the year instead of late winter or spring. In 1912 Anderson and Goldberger (4) proved that Brill's disease was immunologically identical with Mexican typhus, or tabardillo. Naturally this led to the belief that it was louse borne.

[1] Read before the laboratory section of the American Public Health Association, Pasadena, Calif., Sept. 2, 1932.

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tomy is still in its infancy. Postoperative observations may be changed as the survival period increases, which, if it does, will alter the surgical indications and the selection of cases. Close cooperation between laboratory investigators, clinicians and thoracic surgeons is imperative in order to make the best selection of cases for surgery and to evaluate accurately the results obtained, whether they are failures or successes.

ASBESTOSIS

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Asbestosis is a pneumoconiosis caused by the inhalation of asbestos dust. It is distinct from silicosis in its pathology and clinically. Whether asbestosis will remain a distinct form of pulmonary dust disease or will prove to be of a type common to a number of dusts remains to be seen. So far, it is the only pneumoconiosis, other than silicosis, that has received any considerable amount of study from pathologists, clinicians and industrial hygienists.

Whereas silicosis has been recognized for many centuries, asbestosis is a newcomer. Asbestos (Canadian) is a hydrated magnesium silicate containing no free silica but about 44 per cent of combined silica, 43 per cent magnesium, nearly 13 per cent of water, and traces of iron and nickel. While asbestos was known to the ancients, the fabricating of asbestos on a large scale is comparatively new; it received a tremendous impetus from its use in connection with automobiles and as an insulating material and a heat resistant for a great variety of mechanical purposes.

While Hoffman[1] called attention to the possible harmfulness of asbestos dust in 1918, it was not until February 1927 that asbestosis was, so to speak, officially recognized in this country by the filing of a disability claim for workmen's compensation in Massachusetts. The claimant was a foreman in the weaving department of an asbestos plant, and the claim was upheld by the Massachusetts Industrial Accident Board. This was twenty-seven years after the first fatal case was reported in England by Dr. Montague Murray. In 1910 and again in 1934 a fatal case was reported in England, and in 1928 the British Factory Department conducted an investigation and enacted laws for the protection and compensation of the worker against this hazard. The whole subject in England has been summarized by Merewether.[2] At the present time, as nearly as can be estimated, there are about 12,000 individuals employed in the chief asbestos plants in the United States, of whom 10,000 might be exposed to asbestos dust.

In 1927 a fatal case of uncomplicated asbestosis was reported to the Medical Society of South Carolina,[3] and since then, including this case, there have been eleven fatal cases reported in the United States, eight uncomplicated and three complicated by tuberculosis. These reports, together with the fact that asbestosis figured in the extraordinary occupational disease litigation that has spread over this country, resulted in both laboratory and field studies of this new hazard. Gardner and Cummings[4] at Saranac Lake, N. Y., commenced animal experimentation with asbestos in 1928 and reported their observations in 1931. These two authorities in the United States and Gloyne[5] in England have described the pathology. While silicosis is predominantly parenchymatous, asbestosis is mainly interstitial; nor is asbestosis characterized by the nodular formation so distinctive of silicosis.

A search of all the death records on file in the Metropolitan Life Insurance Company revealed that asbestosis had been given as a cause or contributing cause of death in only nineteen cases. The first case noted was in 1924, there were two in 1927, one occurred in 1931, and the rest have occurred since 1933. The diagnosis was supported by autopsy in only six. In one of the six the primary cause of death was carcinoma, in another glioma of the brain, and in another pulmonary tuberculosis. In the other three asbestosis was given as the primary cause, with cardiac failure as the contributing cause. Some of these cases were reported in the literature.

Asbestosis complicated by heart disease was given as the cause of death in seven cases. There were no autopsies in these seven and pulmonary tuberculosis was diagnosed in several by the attending physician, so perhaps too much weight cannot be given to these certificates.

Diabetes and pulmonary tuberculosis were also mentioned in the remaining six cases, in which there were no autopsies. All nineteen patients were males and with one exception all were white.

In our studies of asbestos mines and fabricating plants,[6] the clinical picture of asbestosis was milder than that of silicosis. To be sure, the individual patient with marked asbestosis will greatly resemble the individual with silicosis. There is the same dyspnea on exertion, the same dry cough, and the more or less indefinite physical signs elicited by the stethoscope. The patient with asbestosis is apt to have clubbed fingers not usually seen in silicosis—and he is apt to be pale faced and even show a slight cyanosis, while the silicotic patient is apt to be fairly robust looking. Of course in each instance I refer to patients whose disease is not complicated by infection.

We did not find in communities in which asbestos was mined or fabricated the familiar picture of disability and tuberculous infection so characteristic of hard rock mining communities. Our observations were supported by the statements of physicians practicing in these communities. All the patients with asbestosis that we detected were, with one exception, working steadily at their trades. In only one case did we find evidence of active tuberculosis and that diagnosis was based only on the roentgen appearance. Several showed healed tuberculosis. Gardner and Cummings in their reports called attention to the difference between the action of asbestos dust and silica dust in relation to tuberculous infection in experimental animals, and their observations tend to bear out our clinical study.

In all, our clinical data are based on 126 physical examinations of asbestos workers, all of whom had more than three years' exposure and who were selected at random. Sixty-three of these presented a roentgen appearance which we thought indicated a pneumoconi-

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Read before the Section on Preventive and Industrial Medicine and Public Health at the Eighty-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 13, 1935.

1. Hoffman, F. L.: Mortality from Respiratory Diseases in Dusty Trades, Bull. 231, U. S. Department of Labor, June 1918.
2. Merewether, E. R. A.: A Memorandum on Asbestosis, Tubercle 15: 69 (Nov.), 109 (Dec.) 1933; 16: 152 (Jan.) 1934.
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4. Gardner, L. U., and Cummings, D. E.: Studies on Experimental Pneumonokoniosis: VI. Inhalation of Asbestos Dust: Its Effect upon Primary Tuberculous Infection, J. Indust. Hyg. 13: 65 (Feb.) 1931.
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7. Dublin, Kaltreider, Disability, 1934.

osis, but the symptoms were indefinite and inconclusive. We called these cases first stage. Four presenting evident pulmonary symptoms and corroborative roentgenograms, we termed second stage. Of these sixty-seven patients, twenty had been exposed more than ten years and thirteen more than fifteen years. We are still continuing our asbestos studies and hope to secure additional information on the time element in development and the tendency and rate of progress.

One feature that has impressed us is that the British investigators found asbestosis more severe and more menacing than we did. This difference may be more apparent than real, but it is possible that the English factories may be more dusty than ours. There were not available any comparative dust counts, but this impression is based on their reports and on statements made to myself and my colleagues by persons familiar with the English conditions. One process, described in the British reports as "mattress making" and stated to be extremely dusty, does not appear to have a counterpart in this country. In both countries, energetic steps have been taken to control the dust hazard in asbestos plants, so that it is probable that further cases of disabling asbestosis will be rare.

As in silicosis, the diagnosis centers on the roentgenogram. However, the whole matter of attempting to interpret these films and correlate them with the clinical evidence, if any, is difficult and elusive. If, in our studies, we had found a more clear-cut and severe type of pneumoconiosis with marked symptoms and disability as well as a distinctive roentgen appearance, such as my colleagues and myself had been accustomed to read in our previous investigations of silicosis, our task in attempting to make a positive diagnosis and estimate the extent of the disease would have been easier. There is no doubt that, especially in the beginning, we were handicapped by endeavoring to evaluate asbestosis with a silicosis foot rule.

The x-ray appearances are not clear cut or distinctive as in silicosis and do not lend themselves to ready grouping into progressive stages. There are less evident pathologic changes in these films and the shadows are finer, more granular and softer than in silicosis. The asbestosis film gives the impression of ground glass, and there is no nodulation with the consequent tendency of the nodules to coalesce and give dense opaque areas in the films. The distribution of the shadows is somewhat different, occupying the lower third of the lung, except in far advanced cases, when the shadows may occupy the major portion of the lung. We noticed frequently the well marked outline of the interlobar septum on the right side. We also noticed that a number of films in cases of asbestosis showed enlarged hearts, and this might be expected when one considers that the pathologic process tends to constrict the pulmonary blood vessels as they ramify along with the bronchioles.[7]

It is possible that the x-ray appearance of asbestosis may not be distinctive of this disease alone but uniform in appearance with pneumoconiosis due to other silicate dusts. Much more investigation and study of roentgenograms of industrial workers exposed to all sorts of silicate and other dusts are needed before it will be possible to speak definitely on this, the most important phase of the diagnosis of pneumoconiosis.

One thing is certain. The utmost care and patience are needed to elicit the occupational history of the patient and to correlate this and the clinical picture with the roentgenogram before a diagnosis of asbestosis is justified. As noted in relation to other occupational health hazards, there is an all too frequent tendency to make a diagnosis of a specific occupational disease because of presumptive or actual exposure without the corroboration of other essential factors to a correct diagnosis. One is not justified in making a diagnosis of asbestosis any more than of silicosis in the presence of a roentgenogram showing no distinctive pulmonary pathologic changes.

Associated with exposure to asbestos dust is the occurrence in the sputum and pulmonary tissues of a peculiar formation known as asbestos bodies. These asbestos bodies have been described by a number of observers and are due apparently to the action of the tissues on the asbestos fiber. Their exact significance is doubtful, but it is commonly agreed at the present time that they are not diagnostic of pulmonary fibrosis and indicate merely that the individual has been exposed to asbestos dust.[8] Some observers believe that, when these asbestos bodies appear in the sputum in clumps, they indicate actual disintegration of lung tissue.

Dr. Miller[9] of the United States Public Health Service describes the technic that he employs in the intraperitoneal injection of finely divided dusts in a state of suspension. Miller defined three types of reaction: absorptive, inert and proliferative. The first is produced by relatively harmless or inactive dusts, the second by dusts that might cause pulmonary fibrosis, and the third as the typical reaction of silicosis. Recently Miller[10] described the effects of the injection of three varieties of asbestos; namely, chrysotile, crocidolite and amosite. Chrysotile is Canadian asbestos and, as previously stated, is largely magnesium silicate. Crocidolite and amosite contain only a small quantity of magnesium, containing instead iron silicate in approximately the same quantity.

The three types of asbestos produced the same type of reaction; namely, the one described by this investigator as inert. He states:

> We have been assuming that the dusts producing this inert reaction cause pneumoconiosis of the diffuse fibrosis type as distinct from the proliferative reacting dusts which cause nodular fibrosis. This assumption, as you know, has not been proven by corresponding animal experiments with the same dusts, but results from observation of pathologic material from autopsies. . . . I believe that the gross behavior of asbestos in the tissues has further strengthened the value of our classification of dusts and the intraperitoneal test as a means of determining the harmful dusts by its clinical correlation.

Much work remains to be done before asbestosis is spoken of as authoritatively as is silicosis. In the meantime, asbestos plants are being cleaned up and the dust is being controlled. This, together with the smaller number of persons employed, implies that there will probably never be the wealth of clinical material that has been available in silicosis. It is by no means certain that asbestosis progresses as does silicosis after withdrawal from dust exposure, nor does infection seem to be as closely and intimately associated with asbestosis as with silicosis. The answer to these and other problems resulting from exposure to silicate dusts demands further study both in the field and in the laboratory.

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Silicosis and Asbestosis

BY VARIOUS AUTHORS

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esses in which dust control measures can be readily instituted. As soon as the hazard was realized, industrial firms fabricating asbestos took energetic steps to control the dust so that it is probable that cases of asbestosis will become uncommon. A recent report from the United States Public Health Service details dust control methods instituted in an asbestos textile plant which illustrates in a striking manner what can be done to alleviate dust.[23]

Asbestosis has not appeared to be as serious a disease in the United States as it has in England, judging from the various reports from the latter country. Asbestos "mattress making," mentioned in the British reports as a serious hazard, is a process not found in this country. The role of infection in asbestosis is not as clearly defined as in silicosis and the clinical evidence available indicates that tuberculosis does not have the intimate relationship with asbestosis that is so conspicuous in silicosis.[24 25]

Effects of Silicosis upon the Families of Silicotics

From the earliest times, when silicosis was first described as a disease of miners, the fact that the wife did not contract tuberculosis from the husband was mentioned and the many recent investigations have not upset this general conclusion. Hoffman [26] stated in 1922, "The frequency of pulmonary tuberculosis among immediate relatives of granite cutters, but particularly wives, daughters, and mothers, is much less than, as a mere matter of probability, would be assumed to be the case." This was borne out by the later studies of McFarland [27] and Russell.[28]

Cummings of the Saranac Laboratory, who investigated this subject in connection with the Picher Clinic, commented on the fact that, despite the wretched living conditions, and the lack of cleanliness and sanitation in many homes of advanced silicotics whose sputum was loaded with tubercle bacilli, no

adult type tuberculosis was found among exposed children up to the age of sixteen.

Nevertheless, these various testimonies do not exclude the necessity of treating tuberculo-silicosis as a communicable disease and of making follow-up investigations of members of families of advanced or disabled silicotics, to determine the presence of tubercle infection or the desirability of segregating contacts from the diseased member of the family. Sound public health procedure should be the rule and reliance should not be placed upon the supposed immunity of the family because the tuberculosis is complicated by or consequent upon silicosis.

Control and Prevention of Silicosis

The control of silicosis is both an engineering and a medical problem, as are so many other situations that concern the public health. Both the engineer and the physician, with their technicians, have their parts to play. In some instances, the medical phase may predominate ; in others, the engineering, but it is seldom that they can be separated or be independent of each other if a thorough job of prevention is to be done.

Engineering Control

The development of silicosis is dependent upon the dosage of silica and the matter of avoiding a dangerous quantity might be thought simple. The lay mind, familiar with the definiteness of the hazard of carbon monoxide, and other toxic substances producing acute effects, usually fails to appreciate the inherent difficulties of establishing a threshold limit for a substance producing a chronic disease whose development is measured in terms of years rather than hours.

From time to time, critical comment is directed toward the industrial hygienist by the engineer because the former has failed to inform the latter of the safe or threshold limit of

INDUSTRIAL HYGIENE AND TOXICOLOGY

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of aluminum by physicians for therapeutic or prophylactic purposes, makes only metallic aluminum available. Some investigators, notably Hannon,[73] have reported marked success in the treatment of patients with severe disability. However, when purely objective criteria have been used to measure disability the results have been somewhat less striking. Gardner, Dworski, and Delahant[74] have suggested that variations in response to aluminum therapy may be due in part to the nature of the dust that has caused the silicosis. They suggest that dust which is primarily free silica may react with aluminum more readily than dust which contains high percentages of iron. The use of aluminum generally in industry for the therapy or prophylaxis of silicosis should be contemplated only with the utmost caution.

I. ASBESTOSIS

Asbestos is a hydrated magnesium silicate. More than 90 per cent of the raw mineral used in this country and Great Britian is produced in the Canadian chrysotile mines. Asbestos is used in two general types of manufacturing processes. It may be used either by itself or mixed with other insulating materials such as diatomaceous earth for fireproofing, packing, or insulating; or it may be combined with cotton and woven as a textile for fireproof and heat-resistant clothing and other substances. Lanza[75] estimates that there are about 10,000 persons exposed to asbestos in the United States. Most observers feel that the incidence of asbestosis in American asbestos workers is quite low. Asbestosis has, however, been reported more frequently in England and Canada.

Lanza, McConnell, and Fehnel[76] concluded from their study of asbestosis that: Prolonged exposure to asbestos dust causes pulmonary fibrosis different from that produced in silicosis and demonstrable by roentgenogram. Clinically it appears to be milder than silicosis.

Definite cardiac enlargement frequently was found to be associated with asbestosis.

A predisposition to tuberculosis, due to asbestos dust, was not indicated although it was not known to what extent asbestosis may add to the mortality from pneumonia and acute nontuberculous pulmonary infections.

Symptoms. The onset of asbestosis, as of silicosis, is slow although symptoms are apt to be somewhat more marked than in silicosis. In silicosis there may be marked x-ray findings with little complaint of symptoms, whereas the opposite is likely to be the case in asbestosis. As in silicosis, dyspnea is the cardinal symptom. Anorexia occurs frequently in advanced stages, and cyanosis and clubbing of the fingers are apt to appear with greater constancy in asbestosis.

Pathology. The fibrosis in asbestosis is diffuse and tends to predominate in the basal portions of the lungs in contrast to the generalized nodular fibrosis of silicosis

[73] J. W. G. Hannon, *Trans. Can. Inst. Mining Met.*, *48*, 180 (1944).
[74] L. U. Gardner, M. Dworski, and A. B. Delahant, *J. Ind. Hyg. Toxicol.*, *26*, 211 (1944).
[75] A. J. Lanza, *J. Am. Med. Assoc.*, *106*, 368 (1936).
[76] A. J. Lanza, W. J. McConnell, and J. W. Fehnel, *U. S. Pub. Health Repts.*, *50*, 1 (1935)

with a predominance in the upper portions of the lung. Bronchiectasis and bronchiolectasis are frequent, especially in the more fibrous portions. Whereas the proliferation of fibrous tissue is caused by chemical action in silica exposure, it is induced by mechanical action in asbestos exposures. Gardner[77] found that the fibrosis-producing character of asbestos could be almost eliminated by grinding the fibers so that no particles more than 2 μ in length were present. As previously mentioned, asbestos is classified among the inert dusts.

Microscopic Anatomy. Johnstone[78] described the microscopic appearance of the lungs somewhat as follows: in the early phases of the disease there is thickening of the alveolar septa which results from fibroblastic proliferation. The alveolar spaces contain numerous phagocytes. With progression of the disease fibrosis becomes more marked; the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few alveoli that remain in the area of fibrosis are lined with low cuboidal epithelium giving them an almost glandular appearance.

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures described first by McDonald.[79] These bodies are 20 to 100 μ in length and are bulbous on one or both ends so that they appear club- or dumbbell-shaped. They are brownish in color, do not stain, and give a Prussian-blue reaction for iron. Simson[80] has produced these bodies in guinea pigs by experimental exposure to atmosphere containing asbestos. Lynch[81] concludes that these "curious bodies" signify exposure to asbestos dust but do not necessarily indicate asbestosis.

X-Ray Examination. For an excellent review of roentgenographic findings in both silicosis and asbestosis the reader might well refer to Pendergrass.[82] Characteristic differences in the roentgenographic findings in silicosis and asbestosis are tabulated below:

Asbestosis	Silicosis
Fibrosis diffuse (film may have ground glass appearance from pleural involvement).	Fibrosis nodular.
Findings may be either bilateral or unilateral.	Findings characteristically bilateral.
Lesions largely in the lower one half or two thirds of the lung fields.	Lesions predominately in the upper two-thirds of the lung fields.
Emphysema in upper portion of lung fields.	Emphysema in lower portion of lung fields.

Control. Prevention of asbestosis depends entirely upon preventing exposure to concentrations of dust sufficiently high to produce the characteristic reaction.

[77] L. U. Gardner, *Ind. Med.*, **9**, 45 (1940).
[78] R. T. Johnstone, *Occupational Diseases*. Saunders, Philadelphia, 1942
[79] S. McDonald, *Brit. Med. J.*, **2**, 1025 (1927).
[80] F. W. Simson, *Brit. Med. J.*, **1**, 885 (1928).
[81] K. M. Lynch, *J. Am. Med. Assoc.*, **109**, 1947 (1936).
[82] E. P. Pendergrass, "Roentgen-Ray Diagnosis in Silicosis and Asbestosis," in A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

Dreessen, DallaValle, Edwards, Miller, and Sayers[83] have found evidence to indicate that 5,000,000 particles per cubic foot of air is a satisfactory figure for the maximum permissible atmospheric concentration of asbestos to which workers may be exposed. Definite permissible standards, however, have not been established.

VI: Dust Causing Minimal Fibrosis or No Fibrosis

Gardner[77] has pointed out that any known inorganic dust other than free silica and the asbestos silicates may produce nonspecific dust reactions, as described on page 480. X-ray markings may be found in other conditions such as chronic infection and heart disease. No interference with pulmonary function or disability is produced by dust of this type and there is no influence on susceptibility to tuberculosis.

A. SILICATES

McCord,[84] from a number of sources, has compiled a list of the commonly used silicates, which is presented here:

Olivine—a magnesium silicate widely present in all basic rocks. Many varieties of this stone are known.

Calcium silicate—nonexistent in nature, but a common product of industry; may be found at blast furnaces and in production of cement and hydrated limes. Other silicates of calcium are known.

Willemite—a zinc silicate (rare).

Sodium silicate (meta)—the well-known water-soluble silicate, commonly called water glass. This is widely used in industry, notably as a filler in soaps. This is the only crystalline silicate of sodium, the others being amorphous glass.

Tremolite—a magnesium-calcium silicate.

Asbestos—tremolite, actinolite, chrysotile, or amianthus, all of which are essentially magnesium silicates.

Jade—another form of magnesium-calcium silicate.

Crocidolite—"blue asbestos." It is a sodium iron silicate.

Talc—a hydrated magnesium silicate with extensive industrial application.

Soapstone—a form of talc.

Agalite—a variety of talc resembling asbestos. It is used in the coating of paper, and as a paper filler.

Meerschaum—also called sepiolite. It is closely akin to talc.

Serpentine—hydrated silicate of magnesium. It is a common building stone.

Sillimanite—aluminum-containing silicate. The source of many stone tools of the stone age

Topaz—a semiprecious stone of silicate origin. Chemically it is an aluminum fluosilicate.

Fuller's earth—a hydrated silica-aluminum compound, associated with ferric oxide.

Kaolin (kaolinite)—many related silicates of mixed constituency.

Clays—hydrated aluminum silicates containing iron. Titanium, quartz and mica, are likely to be present in clays.

[83] W. C. Dreessen, J. M. DallaValle, T. I. Edwards, J. W. Miller, and R. R. Sayers. *U.S. Pub. Health Bull.* No. 241 (1938).

[84] C. P. McCord, *Ind. Med.*, 2, 4 (1933).