FILE NAME: State of the Art Literature (SAL)

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2. Since material leaving picking machines is productive of considerable dust, storage rooms receiving such material should be exhausted. Workers who must enter such rooms while material is being blown should be equipped with approved types of respirators. No material should be removed from storage rooms while the picking machine is in operation.

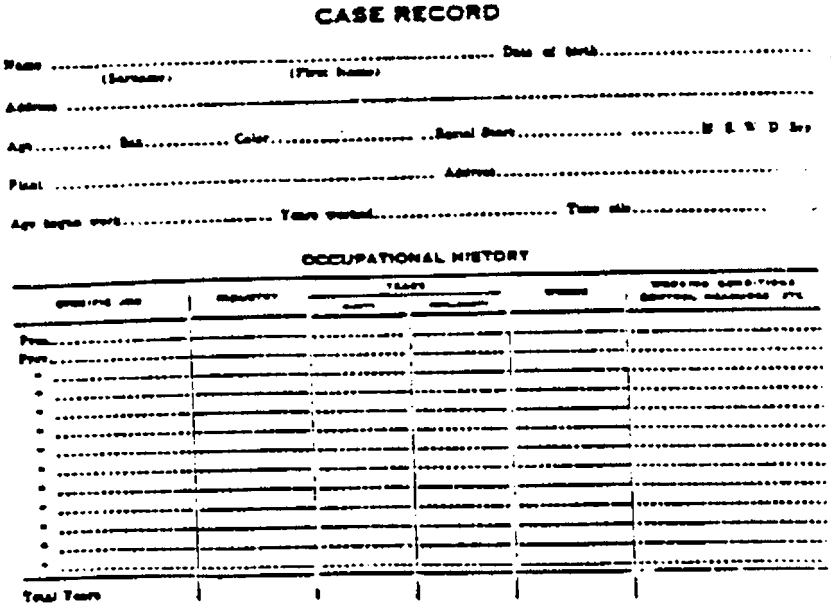
3. Clean air should replace the dust-laden air removed by exhaust systems attached to the equipment. This requires, in event of recirculation of air, that dust concentrations entering any room be kept at levels comparable to that found in outdoor air.

4. Periodic studies of the condition of the working environment appear necessary to determine whether the control methods adopted are constantly adequate. This requires that dust concentrations be determined for each operation. Ventilation measurements are equally important. The work of inspection and review should be performed by persons trained for such studies.

STATISTICAL DESCRIPTION OF EMPLOYEES

OCCUPATIONAL HISTORIES

At the start of the medical examination the physicians questioned each worker to obtain a complete record of all the jobs at which he had worked. This information was recorded on the portion of the physical examination form reproduced in figure 27. After the basic census data (name, age, sex, color, and marital status) had been recorded the worker was asked how old he was when he first began


CASE RECORD

Name ..... Date of birth.....
(Surname) (First name)

Address .....

Age..... Sex..... Color..... Marital status..... M S W D Sep

Plant ..... Address.....

Age began work..... Years worked..... Time off.....

OCCUPATIONAL HISTORY

Occupying job	Industry	Years		Wages	Working conditions (chemical, mechanical, etc.)
Pres.					
Prev.					
Total Years					

FIGURE 27.—FORM USED FOR RECORDING OCCUPATIONAL HISTORY

industrial work. This figure was subtracted from his present age, and a line of questioning was begun to elicit information on the work done in the intervening years. No occupational history was regarded as complete until all of this time could be accounted for by definite entries. The job he held at the time of the study was entered on the top line of the occupational history, together with the number of years he had held it, the name of the department in which he worked, and any other pertinent data that could be obtained. Next, he was asked what he had done previously, and again a complete entry was made. The name of the factory was entered for each period of em-

ployment in the asbestos textile industry. After each job had been accounted for, the worker was asked what he had done before that.

If a man had worked in an industry in which workers are known to be exposed to pneumoconiosis-producing dust, entries were made in the last column to show what materials he handled and whether or not dust-control measures were in use. Information on previous employment in dusty trades is of paramount importance in a study such as this. Any cases of prolonged exposure to pneumoconiosis-producing dust other than asbestos must be treated separately.

After the field study had been completed, an engineer assigned dust exposure values to each worker, following a procedure illustrated in table 18. In assigning these values, consideration was given to each job a worker had held in an asbestos plant.

The importance of the occupational history in a study of this kind can scarcely be overemphasized. It is the connecting link between the engineering and medical studies. The correlations between medical findings and dust exposure that adequate occupational histories make possible provide means for estimating the dust concentrations to which workers may be exposed safely.

Only 23 persons reported previous employment in an industry in which workers are known to be exposed to pneumoconiosis-producing dust. Such periods of employment usually were so short that no deleterious effects would be expected. Twenty-one persons had normal lung-field markings. Only one of these 23 persons had asbestosis at the time of this study, a man who had worked 3 years in a foundry and 10 years in asbestos textile plants.

COMPARISON WITH OTHER INDUSTRIAL WORKERS

Five-sixths of the employees were native-born Americans of Anglo-Saxon stock and the remainder were Negro males.

Before beginning work in asbestos textile factories, more than 200 of the 541 persons had worked at similar occupations in cotton or wool textile plants. A large proportion of the employees had worked on farms either before beginning industrial work or in intervals between periods of industrial employment.

About 15 months before this study was begun, approximately 150 workers in these asbestos textile factories were replaced by workers with little or no previous asbestos exposure. Although an effort was made to examine as many as possible of these former workers, less than half could be examined. Partly as a result of these personnel changes, the group of employees examined in this study had an unusually low average age. There was an abnormally large percentage of workers with less than 5 years' employment in the asbestos textile industry and an abnormally small percentage of persons who had worked 10 years or more in this industry.

TABLE 21.—*Age distribution of asbestos workers*

Age	Number of workers			Total
	White males	White females	Negro males	
Total	238	114	44	396
Under 20 years	14	12	1	27
20 to 24 years	35	24	5	64
25 to 29 years	32	20	4	56
30 to 34 years	26	14	4	44
35 to 39 years	18	11	3	32
40 to 44 years	14	10	4	28
45 to 49 years	10	4	3	17
50 to 54 years	4	4	1	9
55 to 59 years	4	2	1	7
60 to 64 years	1	1	0	2
65 years and over	1	0	0	1
Unknown age	0	2	0	2
Average	32.05	30.41	32.44	[illegible]
Standard deviation	9.99	10.39	9.92	[illegible]

The low average age of this group of asbestos textile workers must be kept in mind in making comparisons of health conditions in these factories with health conditions in American industry in general.

TABLE 22.—*Average age of white male industrial workers*

Industry	Average age	Public Health Service Bulletin	Industry	Average age	Public Health Service Bulletin
Asbestos textile	32.1	[illegible]	Glass	35.1	[illegible]
Iron and steel	42.0	208	Gas	34.4	[illegible]
Granite cutting	39.8	147	Clay	34.2	[illegible]
Anthracite mines	39.3	221	Cement	33.9	[illegible]
Fur cutting	38.2	204	Rubber	33.1	[illegible]
Pottery	38.1	118	Chemical	33.1	[illegible]
Post office	37.6	143	Garment	32.5	[illegible]
Forestry	37.6	148			

The average age of white male industrial workers is less in the asbestos textile factories studied than it is in 14 other industries in which the Public Health Service has made similar studies. White males employed in asbestos textile factories average 32.1 years, white females 30.4 years, and Negro males 32.4 years of age.

TABLE 23.—*Length of employment in the asbestos textile industry*

Sex and color	Total	Years employment in asbestos textile industry						
		Less than 1	1 to 4.9	5 to 9.9	10 to 14.9	15 to 19.9	20 to 24.9	25 to 29.9
Total	511	74	240	112	47	26	7	5
White males	431	44	201	92	42	23	7	4
White females	111	19	31	15	5	3	0	1
Negro males	69	11	8	5	0	0	0	0

Correspondingly, the average length of employment in the asbestos textile industry is relatively short. Only 18.8 percent of the employees have been employed in the asbestos textile industry more than 10 years. In the 13 industries included in table 22 for which information on length of employment is readily available, only two industries have as large a percentage of employees who have worked less than 10 years. This is partly accounted for by the fact that the asbestos textile industry is of comparatively recent origin in this country. Only one of the plants studied had been in operation before 1920.

MEDICAL STUDY

PROCEDURE

A medical examination was made of each worker employed in the asbestos textile factories of North Carolina during the winter of 1935–36. A group of former asbestos workers was also examined in order to obtain a complete sample of asbestos-exposed workers. Office workers and other employees not exposed to asbestos dust were included in these examinations. They are classified as controls. Included in this control group, which numbered 76 persons, were 27 persons seeking employment in the asbestos industry for the first time.

Before making any medical examinations the two physicians made an inspection tour of each factory in which this study was made in order to familiarize themselves with the various jobs or occupations involved in the manufacturing process. This is a necessary prerequisite for taking a usable occupational history.

Space for examination and fluoroscopic rooms and laboratory was obtained at a location as convenient as possible for all concerned. An improvised darkroom was used for fluoroscopy and for processing the X-ray films. In an adjoining room, the medical examinations were made by two physicians approximately according to the following schedule: Four persons at 9 a. m.; four at 10:30 a. m.; four at 1 p. m.; four at 2 p. m.; and four at 3 p. m. In other words, about 30 minutes were required per examination. The routine used in the study of pulmonary tuberculosis was followed in the examination of the chest. After the history and physical findings had been recorded, the group of four was fluoroscoped and chest roentgenograms were taken. A few examinations were made in the evening, and other adjustments were made in the schedule so as not to interfere too seriously with the work routine of the employees.

At plant A, the urine of workers was examined with particular attention to amount of silica excretion. In addition to this metabolic study, routine examinations were made of the sputum of workers in plants B and C for presence of acid-fast bacilli and asbestosis bodies.

Sections of the physical examination form used in this study have been reproduced in several places in this bulletin as a means of indicating the information that was collected.

PAST MEDICAL HISTORY

Each person was questioned in order to find out whether he had previously had certain diseases which are of importance in the study of chest diseases.

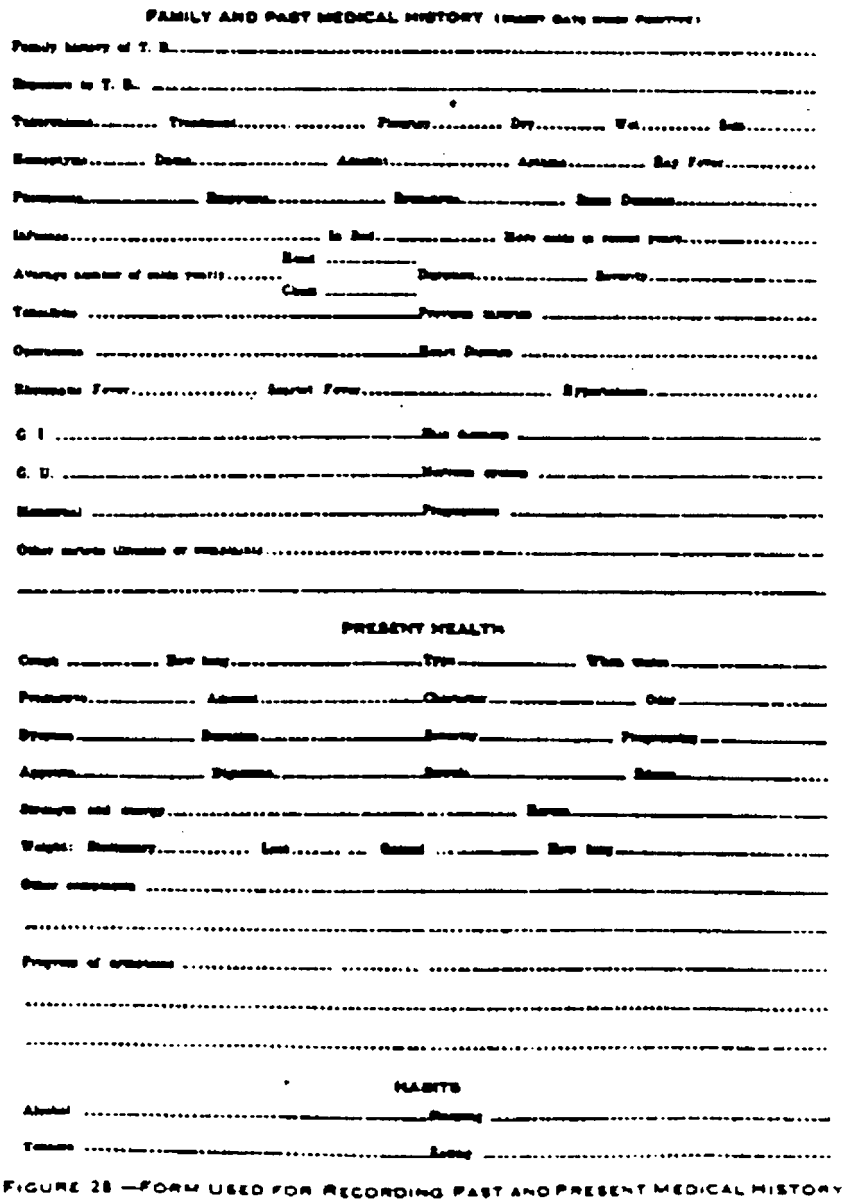


FIGURE 28.—FORM USED FOR RECORDING PAST AND PRESENT MEDICAL HISTORY

Tabulations of previous illness did not indicate that pneumonia, pleurisy, respiratory diseases other than the common cold, digestive

disturbances, or prolonged contact with an active case of tuberculosis had made people more susceptible to asbestosis. Frequent, disabling colds were more common among persons with advanced pulmonary fibrosis than they were in persons with little or no fibrosis, but this probably is a result of the fibrosis rather than a predisposing influence.

SYMPTOMS OF ASBESTOSIS

The chief symptoms usually associated with asbestosis are progressive dyspnoea, cough, hemoptysis, emaciation, weakness, poor chest expansion, curved finger nails or clubbed finger tips, chest pain, and cyanosis. In these respects asbestosis resembles silicosis. Early cases, of course, would have relatively few of these symptoms and an advanced case would have almost every symptom listed. The appearance of these symptoms can be explained on the basis of a more or less general fibrosis of the lungs and concomitant pathological changes such as emphysema, bronchiectasis, and fibrous pleurisy. The cardinal symptoms of asbestosis are cough and dyspnoea. These two symptoms occurred together and in combination with other symptoms far more frequently than one would expect on the basis of chance alone.

TABLE 24.—*Number of persons who had certain pairs of disorders, compared with the number who would be expected to have them on the basis of chance alone. Where the differences are statistically significant the observed numbers appear in boldface type*

Disorder	Persons affected		Cough		Hemoptysis		Loss of weight		Chest pain		Weakness		Loss of appetite	
	Number	Percent of total examined	Observed	Calculated	Observed	Calculated	Observed	Calculated	Observed	Calculated	Observed	Calculated	Observed	Calculated
Dyspnoea	122	30.34	94	36.4	36	10.9	25	10.0	11	5.6	34	11.1	6	1.6
Cough	130	32.31	.....	.....	32	11.6	22	12.9	13	6.7	28	12.5	5	1.5
Hemoptysis	36	8.42	.....	.....	.....	.....	9	3.4	3	1.6	11	4.5	1	0.4
Loss of weight	41	8.84	.....	.....	.....	.....	.....	.....	4	2.7	10	3.7	2	0.6
Chest pain	19	4.80	.....	.....	.....	.....	.....	.....	.....	.....	9	1.7	3	0.3
Weakness	42	9.45	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	6	0.6
Loss of appetite	8	1.80	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....

As table 24 shows, 94 of the 122 people who complained of dyspnoea had a cough also. On the basis of chance alone, assuming there was no causal connection between the two symptoms, one would expect that 36 people would have both conditions.[1] Actually, the two conditions are found together far more frequently than that, and it is safe to assume that there is some sort of causal relationship. Similar considerations hold for most of the other pairs of impairments.

Typically, the cough of asbestosis is dry and nonproductive. In

[1] This value has been calculated on the assumption that [illegible]

some cases there is scanty, viscid expectoration. Severe paroxysms of coughing occasionally occur in cases of asbestosis having serious disability. At such times highly tenacious sputum is raised.

Usually a complaint of dyspnoea was entered on the records only when breathing was labored after an exercise test (see p. 65) carried on in the presence of the examining physicians. The high percentage of persons with dyspnoea who were able to be at work is undoubtedly due to the sedentary nature of much of the work in asbestos textile factories.

Blood-streaked sputum occurred frequently among persons exposed to asbestos dust although there were only two active cases of pulmonary tuberculosis in the group. These two cases have not been included in tables 25 and 26 and figures 29 and 30. The absence of pulmonary tuberculosis makes hemoptysis a more significant finding. Its presence seems to indicate that bronchiectatic and emphysematous processes are going on in the lungs. The presence of asbestosis bodies in the sputum (which will be discussed in a later section) also can be interpreted as a sign of disintegrative processes in pulmonary tissue.

Weakness and loss of weight are closely related symptoms. Independently of age and other factors, these symptoms increase in frequency with increasing dust exposure. They are accompanied by a general bodily appearance not unlike that of fibroid phthisis.

Nineteen of the 447 asbestos-exposed people complained of a pain in their chests which is best described by the term "substernal distress." It was vaguely defined and not as a rule very well localized, and it seemed to be an ache or a feeling of oppression rather than a sharp pain. It did not seem to be of cardiac origin. Only one of the 24 persons with a past history of pleurisy complained of a chest pain of this kind. It appears that substernal distress may be connected with the cough or the dyspnoea that accompany asbestosis (table 24).

The symptoms of asbestosis are listed in table 25, where their incidence is classified according to the total dust exposure of the persons affected. For purposes of comparison, the employees of asbestos textile factories have been divided into five groups, which differed in dust exposure, one of them being a control group of employees who were not exposed to asbestos dust. A number representing the total dust exposure was assigned to each dust-exposed employee by multiplying the number of years he had worked in the asbestos industry by the average dust concentration to which he presumably had been exposed. His total dust exposure is thus expressed in million particle years. If a worker had changed from one occupation to another, a dust exposure value in million particle years was calculated for each occupation, and these values were added together to obtain his total dust exposure.[1]

[1] An explanation of this term is given in the section entitled "Determination of dust exposure."

The percentage of workers who complain of cough, dyspnoea, blood-streaked sputum, and other symptoms increases rapidly with increasing asbestos-dust exposure. In all cases the percentage of workers who are affected by a particular symptom is larger than the percentage in the control group. Two of the cases of dyspnoea found

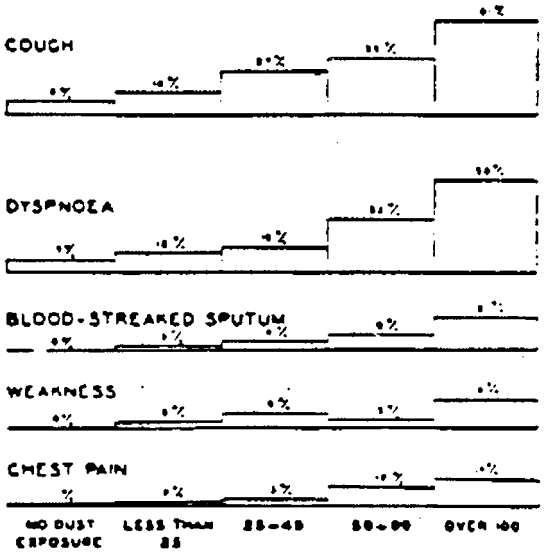


FIGURE 29.—PERCENTAGE OF PERSONS CLASSIFIED BY DUST EXPOSURE, WHO HAD CERTAIN DISORDERS

in the control group were accompanied by heart defects severe enough to have caused shortness of breath.

TABLE 25.—*Classification of present complaints according to the dust exposure of the affected persons*

Present complaint	Not exposed to dust	Million particle-years dust exposure									
		Less than 25		25 to 49		50 to 99		Over 100		Total	
	Number	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Total	50	108	100	51	100	17	100	10	100	186	100
Cough	6	8	7	21	41	12	71	8	80	49	26
Dyspnoea	5	7	6	13	25	10	59	7	70	37	20
Weakness	0	0	0	4	8	3	18	4	40	11	6
Loss of weight	4	5	5	3	6	2	12	3	30	13	7
Blood-streaked sputum	0	0	0	2	4	3	18	3	30	8	4
Chest pain	1	1	1	2	4	1	6	2	20	6	3
Loss of appetite	0	0	0	0	0	1	6	1	10	2	1

[1] Data not available for all workers.

Another indication that these symptoms are an integral part of the symptom complex of asbestosis is to be found in the relation of

these symptoms to the fibrotic changes that occur in the lungs. The degree of fibrosis can be estimated from the lung-field markings seen on X-ray examination, a subject which will be more fully discussed in a later section. For the present it will be sufficient to say that as a result of long-continued exposure to asbestos dust the normal or first-degree exaggeration of linear pulmonic markings become coarser and more widespread (second-degree linear exaggeration). Upon longer or more intensive exposure a fine, interstitial fibrosis sets in which gives a hazy, ground-glass appearance to the lung field (first-

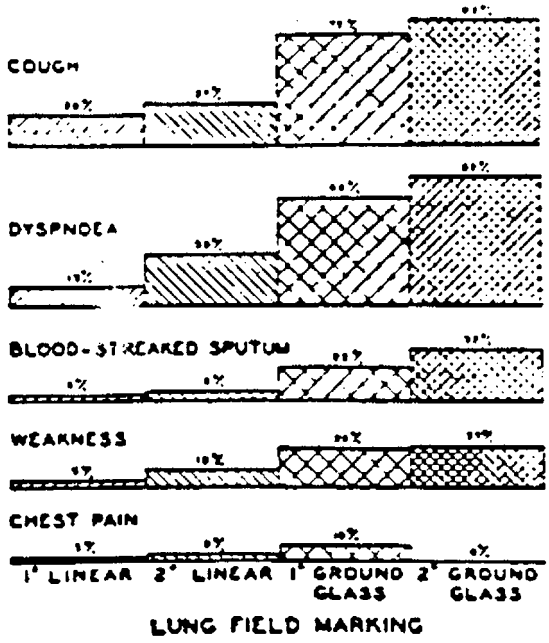


FIGURE 30.—PERCENTAGE OF PERSONS CLASSIFIED BY LUNG-FIELD MARKINGS WHO HAD CERTAIN DISORDERS

degree ground glass) and eventually almost completely obscures the linear markings (second-degree ground glass).

Asbestos-exposed people have been classified into four groups, according to their lung-field markings, and the percentage of persons having certain symptoms has been calculated for each group. Here again, the incidence of these symptoms increases greatly with increasing fibrotic change. Such a relation is entirely to be expected for dyspnoea, since the disease diminishes the volume of the lung available for the respiratory function. The high incidence of the other symptoms in the presence of fibrotic changes that have progressed to the ground-glass stage is additional evidence of degenerative changes in the lungs.

TABLE 26.—*Classification of present complaints according to lung-field markings of the affected persons*

Present complaint	Lung-field markings								Total
	First-degree linear		Second-degree linear		First-degree ground glass		Second-degree ground glass		
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	
Total	312	100	78	100	80	100	22	100	492
Cough	63	20	21	27	36	45	13	59	133
Dyspnea	42	13	28	36	34	43	15	68	119
Weakness	15	5	9	12	13	16	6	27	43
Loss of weight	23	7	6	8	2	3	4	18	35
Blood-streaked sputum	17	5	5	6	11	14	2	9	35
Chest pain	10	3	4	5	5	6	0	0	19
Loss of appetite	1	1	1	1	0	0	0	0	2

X-RAY FINDINGS

LUNG-FIELD MARKINGS

The classification of lung-field markings used in the preceding section is part of a scheme that was used in a study of anthracosilicosis

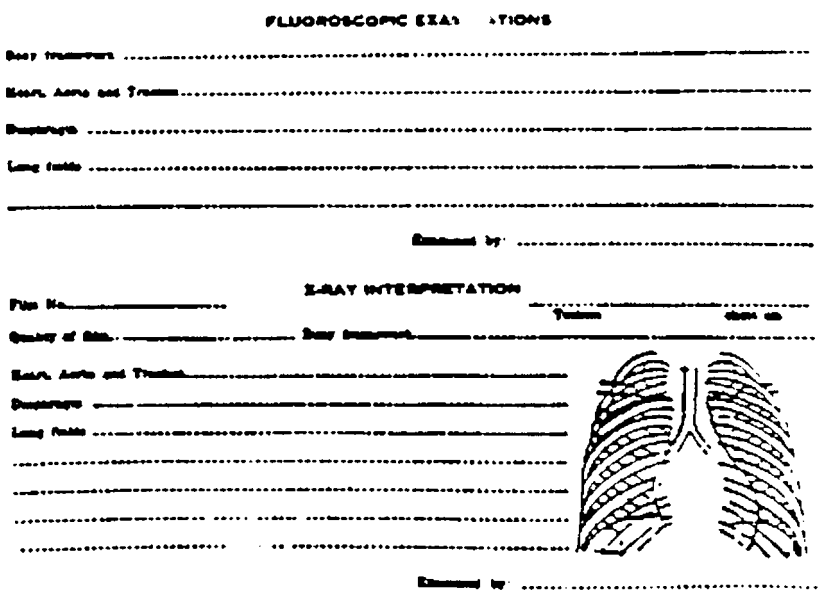


FIGURE 31.—FORM USED FOR RECORDING X-RAY FINDINGS

(42, 51). It is, of course, highly desirable to have a system of classification which will help in deciding whether a given type of lung-field marking represents an early or a late stage of a pneumoconiosis, and this scheme has been drawn up to illustrate the probable sequence of lung-field markings that occurs during long-continued exposure to a

pneumoconiosis-producing dust. During medical studies of anthracite miners, for instance, all of the stages represented in figure 32 were found. Among asbestos-exposed workers, on the contrary, no case was found which had progressed beyond the ground-glass stage. This constitutes a difference between asbestosis and silicosis that Merewether (23), Lanza (18), and Pancoast (29), among others, have commented on. Further investigation will be needed to decide whether this difference is due to a peculiarity of asbestos dust or

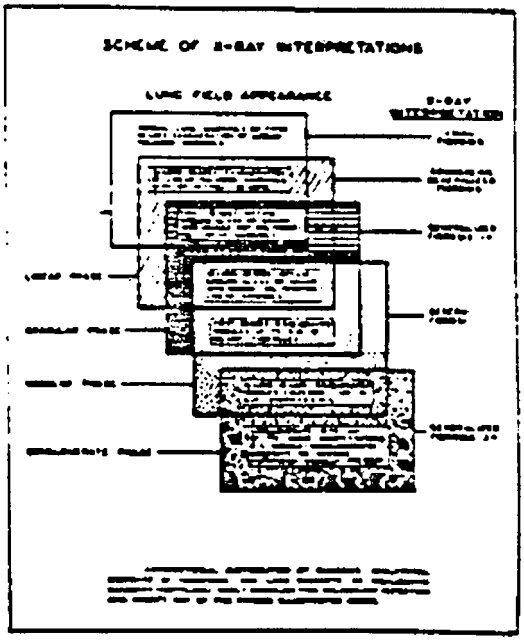


FIGURE 32.—SCHEME USED IN CLASSIFYING X-RAY MARKINGS

whether it is due to the rare occurrence in asbestos textile mills of the excessively high dust exposures (100 to 300 million particles per cubic foot and more) found in certain other dusty trades.

In a typical case of asbestosis the lower half of the chest X-ray film has a fine, granular appearance which Merewether (23) aptly describes as "ground glass." Not infrequently, there is more fibrosis on the right side than on the left. The presence of emphysema in the apices, which results in increased radiolucence, tends to exaggerate the cloudiness of the bases of the lungs. Emphysema precedes the ground-glass appearance of the bases. Chest X-rays of individuals with pneumoconioses induced by other silicates, with the exception of mica,[1] do not regularly show the basal localization of fibrosis that is so characteristic of asbestosis.

[1] Unpublished data.

Occasionally, in an asbestotic chest, the heart has a shaggy outli when viewed roentgenographically, a phenomenon discussed by oth investigators. This seems to be of more frequent occurrence wh the worker has been exposed to dust containing a high proportion fibers (the dust produced in twisting or broadcloth weaving, for i stance) than when he has been exposed to short asbestos partic

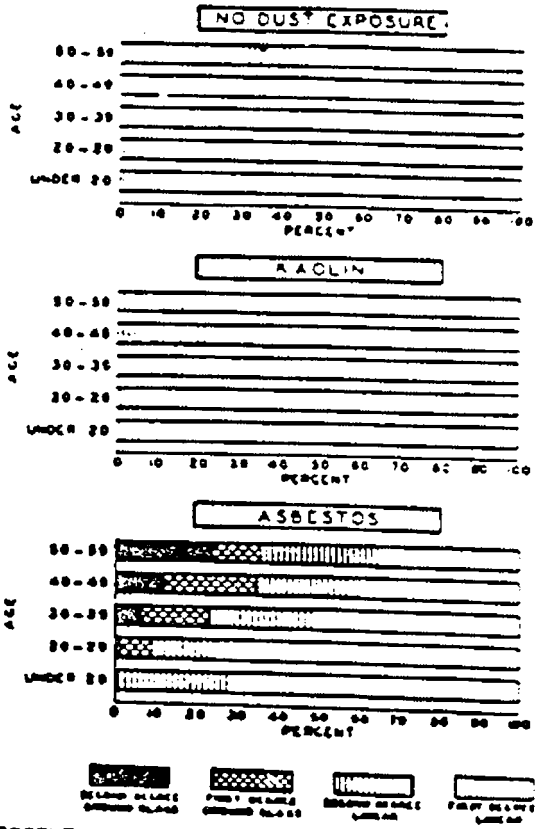


FIGURE 33.—PERCENTAGE OF MALES, CLASSIFIED BY AGE, WHO HAD CERTA LUNG-FIELD MARKINGS. 229 MEN HAD NO PREVIOUS INDUSTRIAL DUST E POSURE 80 HAD BEEN EXPOSED TO KAOLIN DUST, AND 357 HAD BEEN EXPOSE TO ASBESTOS DUST.

such as those produced in carding. Further study would be necessary in order to establish this point.

Unlike silicosis, nodular and conglomerate shadows are not found i asbestotic workers in the absence of tuberculosis or other infections The principal difference discernible on an X-ray film between an early and an advanced stage of asbestosis lies in the density of the ground-glass markings and the degree to which the linear pulmonic markings have been obliterated. Although first-degree and second-degree ground-glass markings have been tabulated separately in several tables published in this report, it is often difficult to decide whether a

particular film represents an early or an advanced stage of pulmonary fibrosis.

There is, of course, an increase in the prevalence of lung fibrosis with advancing age. Because ground-glass appearance of the lung fields which is indicative of fine, interstitial pulmonary fibrosis, is a most important diagnostic sign of asbestosis, it is necessary to evaluate the relation of this condition to age and other relevant factors. From unpublished data collected by the Public Health Service, tabulations of lung-field markings in relation to age were made on two groups of men. One group comprised 229 men who had never been employed in a dusty trade; the other group included 80 men engaged in mining and refining kaolin by wet methods. The percentage of men who have a second-degree exaggeration of the linear pulmonic markings increases with advancing age. It is significant, however, that in the entire group of 309 men there were no cases of ground-glass lung-field markings. Other studies confirm this. Dreessen (43a) examined 80 men exposed to marble dust; 18 percent had second-degree linear markings, but none had ground-glass or nodular markings. Similar data are available (42) for 201 men employed in portland cement mills who were examined by Gardner and Sampson. No cases of granular markings or of discrete, nodular fibrosis were found.

In every age group the proportion of asbestos-exposed men who had advanced stages of pulmonary fibrosis is greater than in the men not exposed to asbestos dust. The percentage of men with ground-glass lung-field markings increases with advancing age. In view of the data for the two occupational groups just discussed and of the relation between this condition and length of employment, it appears that the reason why the incidence of this type of pulmonic markings is correlated with age is that the older people have been exposed to asbestos

TABLE 27.—*Relation of lung-field markings to age: Men seeking employment in a dusty trade for the first time, kaolin workers, and men employed in asbestos textile factories*

Age	No previous dust exposure			Kaolin workers			Asbestos workers				
	Number of men	Percent with first-degree linear markings	Percent with second-degree linear markings	Number of men	Percent with first-degree linear markings	Percent with second-degree linear markings	Number of men	Percent with first-degree linear markings	Percent with second-degree linear markings	Percent with [illegible]	[illegible]
Total examined	229	92	8	80	96	21	279	61	39	61	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
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dust for the longest time. It would be important to know whether susceptibility to asbestosis varies with age, but these data do not seem to be extensive enough to test this point satisfactorily.

The prevalence of ground-glass lung-field markings increases greatly with increasing length of employment, as figure 34 shows.

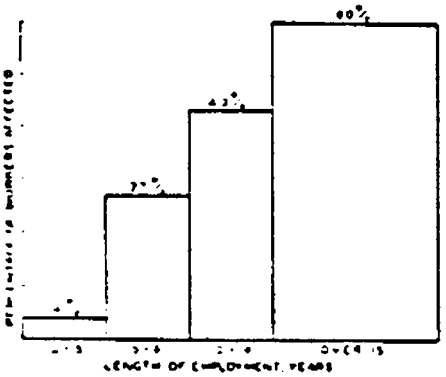


FIGURE 34.—RELATION OF GROUND-GLASS LUNG-FIELD MARKINGS TO LENGTH OF EMPLOYMENT.

The incidence of ground-glass lung-field markings also increases with increasing dust exposure.

In figure 36, which is based on table 28, the heights of the vertical bars represent the percentages of asbestos workers in different exposure groups who had ground-glass lung-field markings of either first or second degree. The 76 controls have been excluded from this tabulation and a number of workers whose dust exposure was not known have also been omitted. Many of the percentage values must be viewed with caution because they are based on small numbers of workers. This is particularly true in the groups with more than 10 years' employment.

Considering the four dust exposure groups one at a time, there is a consistent and regular increase in the percentage of persons with advanced stages of lung fibrosis with increasing length of employment. Age, of course, also increases with length of employment, but these fibrotic changes cannot be ascribed to advancing age because, as table 27 shows, fibrotic changes of this degree are not to be expected in workmen of comparable age who are not exposed to siliceous dust.

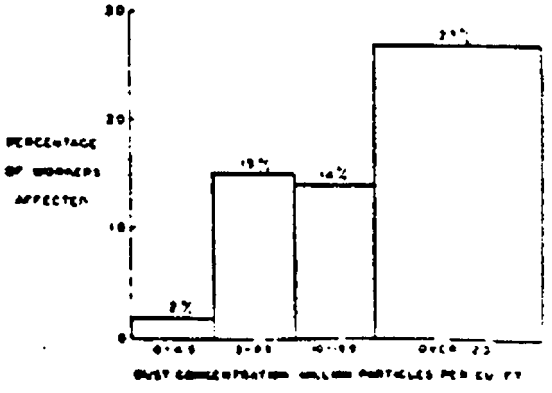


FIGURE 35.—RELATION OF GROUND-GLASS LUNG-FIELD MARKINGS TO DUST EXPOSURE

Obviously, there are great differences between individuals in the time that elapses from their first exposure to asbestos dust and the time that fibrotic changes are evident on X-ray examination. In some persons this was much less than 5 years and in others it appears

to be more than 10. Although it is impossible to account for these differences entirely, it is possible that one factor may be the difference in physical exertion that different occupations require. In a sedentary occupation, shallow breathing might be adequate to meet a person's respiratory needs; such a person would inhale less air, and consequently fewer asbestos particles, than a person engaged in vigorous muscular work who was obliged to breathe deeply. Some of the individual differences to be found in these data may be due to differences in the total amount of asbestos-dust inhaled.

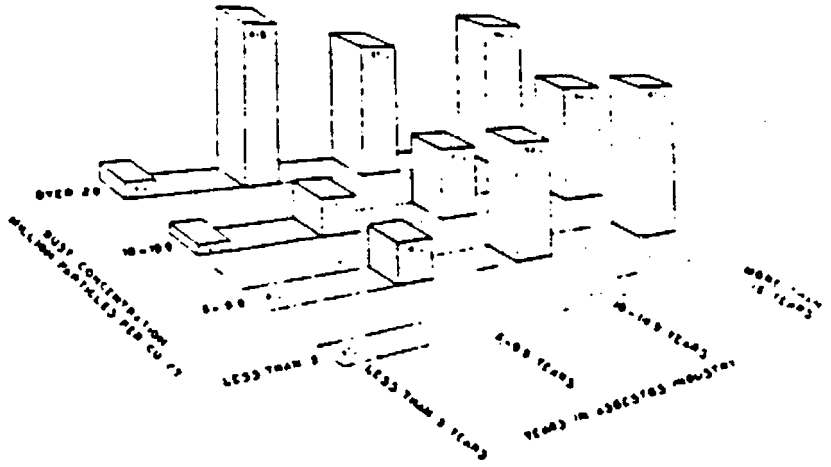


FIGURE 38.—INCIDENCE OF GROUND-GLASS LUNG-FIELD MARKINGS IN RELATION TO LENGTH OF EMPLOYMENT AND DUST CONCENTRATION

TABLE 28.—*Proportion of asbestos textile workers with ground-glass lung-field markings*

Dust exposure (million particles per cubic foot)		Years in asbestos industry			
		0 to 4.9	5 to 9.9	10 to 14.9	Over 15
0 to 4.9	Affected	0	0	0	0
	Examined	52	29	6	1
	Percentage	0.0	0.0	0.0	0.0
5 to 9.9	Affected	0	1	1	4
	Examined	61	38	11	4
	Percentage	0.0	2.6	9.1	100.0
10 to 19.9	Affected	3	4	5	7
	Examined	50	18	11	7
	Percentage	6.0	22.2	45.5	100.0
20 or more	Affected	4	12	6	4
	Examined	41	25	11	4
	Percentage	9.8	48.0	54.5	100.0

It will be noted that the relation between dust exposure and the incidence of ground-glass lung-field markings is not a simple one. Even if length of employment is disregarded, as in figure 35, the incidence of ground-glass markings is proportionately lower at 10 to 19.9 million particles per cubic foot than it is at the next higher or

next lower dust concentration. Although it is probable that these differences represent sampling errors due to small numbers of exposed persons, it may be that asbestos fibers excite a different, and possibly more severe, reaction than asbestos particles. If this were true, the total particle count tabulated above would not be a completely satisfactory measure of the pneumoconiosis-producing power of asbestos dust.

It is not unreasonable to suppose that the amount of pulmonary fibrosis might be more closely related to the weight of inhaled asbestos dust than to any other physical property of the dust. The number of dust particles can be used as a rough estimate of the weight of most dusts (laboratory studies have shown that the two quantities are highly correlated). This may not be as good a measure in the case

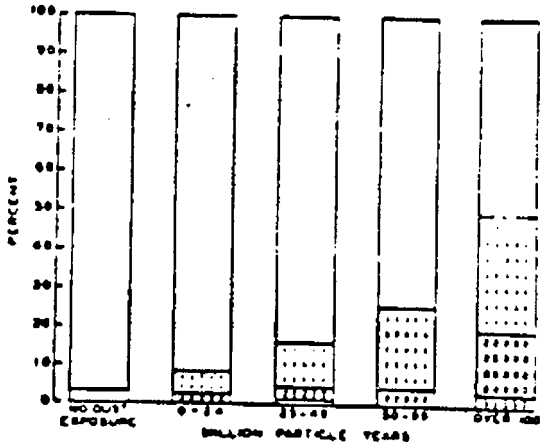


FIGURE 37.—RELATION OF DIAPHRAGMATIC FIXATION TO DUST EXPOSURE. THE PERCENTAGE OF PERSONS IN EACH EXPOSURE GROUP WHO HAD FIRST, SECOND, OR THIRD DEGREE DIAPHRAGMATIC FIXATION HAS BEEN REPRESENTED BY THE HEIGHT OF VERTICAL BARS.

of asbestos because, as table 6 shows, there are many long fibers in the dust suspended in workroom air. The difficulties encountered in measuring fibers have already been discussed (p. 23) and only one additional point need be mentioned here. A fiber 50 microns in length is capable of reaching the lungs (table 37); it would be counted as one particle in making dust counts, and yet it would have approximately 50 times the weight of a fragment of the same fiber only 1 micron in length. The 2 particles would have the same influence on the dust count, but one would carry 50 times as much asbestos to the lung as the other if both were inhaled and retained. Unfortunately, measurements of the weight of dust in the air were not made. If they had been, it is possible that the relation between asbestosis and dust exposure might have been altered.

DIAPHRAGM

During fluoroscopic examination of the chest the degree of movement of the diaphragm was observed and recorded. The percentage of people with impaired diaphragmatic movement increased with increasing length of dust exposure, and the proportion of workers with severe degrees of impairment likewise increased.

TABLE 29.—*Occurrence of diaphragmatic fixation in relation to million particle years of exposure* [1]

PERCENT

Degree of fixation	Controls	Million particle years			
		Under 25	25 to 49	50 to 99	Over 99
Normal	97	92	84	74	[illegible]
−	3	8	12	22	[illegible]
−−	0	2	3	1	[illegible]
−−−	0	0	1	0	[illegible]

NUMBER OF CASES

Degree of fixation	Controls	Million particle years			
		Under 25	25 to 49	50 to 99	Over 99
Total tested	76	196	56	55	[illegible]
Normal	74	182	73	41	[illegible]
−	2	11	10	12	[illegible]
−−	0	3	3	2	[illegible]
−−−	0	0	1	0	[illegible]

[1] The percentage values in the upper half of this table were obtained by dividing the values labeled "total tested" by the corresponding values in the lower half of the table.

This is shown in figure 37 where asbestos workers have been divided into five groups (one of them a control group), according to the total amounts of asbestos dust to which they have been exposed during their working life. Only two of the 76 controls had any impairment of diaphragmatic movement. For each group the percentage with normal diaphragmatic movement, and slight (+), moderate (−−), or severe (−−−) diaphragmatic fixation has been calculated. These percentages have been represented by the heights of vertical bars.

Severe forms of diaphragmatic fixation accompanied advanced stages of lung fibrosis. Among 295 persons with first-degree linear lung-field markings, 9.5 percent had impaired diaphragmatic movement. Of the 22 persons with second-degree, ground-glass lung-field markings, 68 percent had impaired diaphragmatic excursion. This is a reasonable finding, since lung fibrosis and diaphragmatic limitation appear to be part of the same process.

Diaphragmatic abnormalities, such as peaking, did not occur to any significant extent in asbestos workers although they are common among persons with silicosis. In a study of granite workers carried on by the Public Health Service (48, p. 87): "Irregularities of the diaphragm were rather constant. Forty percent of cases had evidence of it, and as the condition advanced there was an increase in changes of the diaphragmatic shadows. There were instances in which angulation of the diaphragm was in evidence, and others which showed indications of extensive adhesions." In a study of anthracite miners carried on by the Public Health Service (51, p. 58): "Decrease in the motibility of the diaphragm as demonstrated by fluoroscopy was frequently observed. This malfunction, varying from slight to practical fixation, increased with the severity of the pulmonary pathology. Not infrequently adhesions between the visceral and diaphragmatic pleura could be seen." On the contrary, in asbestosis, with the exception of one man (fig. 48), there was no roentgenographic evidence of diaphragmatic peaking. It appears that the limitation of movement, which is a definite part of the symptom complex of asbestosis, may be due to inelasticity of the lungs and binding of the lungs to the parietes and the mediastinal structures by fibrous pleuritis. (See pathological section.)

PHYSICAL FINDINGS

The objective symptoms that are most frequently observed and most useful in establishing a diagnosis of asbestosis are general appearance of ill health (particularly any signs of loss of weight), the condition of the finger nails, and diminished chest expansion. These symptoms obviously appear late in the course of the disease. The useful chest signs seem to be limited to adventitious sounds. Diminished breath sounds, prolonged expiration and crackling rales over the bases of the lungs and interscapular regions seem to accompany asbestosis. Various rales and adventitious sounds were heard in the chests of 5 percent of the persons in the control group. Rales were noted in 14 percent of the asbestos-exposed persons who had normal lung-field markings or who had only first-degree linear exaggeration. However, in the group of persons with ground-glass lung-field markings (persons who had asbestosis), 49 percent had rales or other adventitious sounds. In about two-fifths of the cases, rales persisted after cough.

Changes in the percussion note are hard to evaluate, probably because the condition tending to give a dull note is counterbalanced by generalized emphysema which produces hyper-resonance. The absence of localized areas of conglomerate fibrosis and localized areas in which there is marked compensatory emphysema obviously accounts for the lack of more definite findings.

CHEST EXPANSION

Chest expansion decreases with increasing dust exposure and with increasing pulmonary fibrosis. Asbestos-exposed men were divided into four groups according to dust exposure, and the average chest expansion and the average chest girth at expiration were calculated for each group. The same men were classified into four groups on the

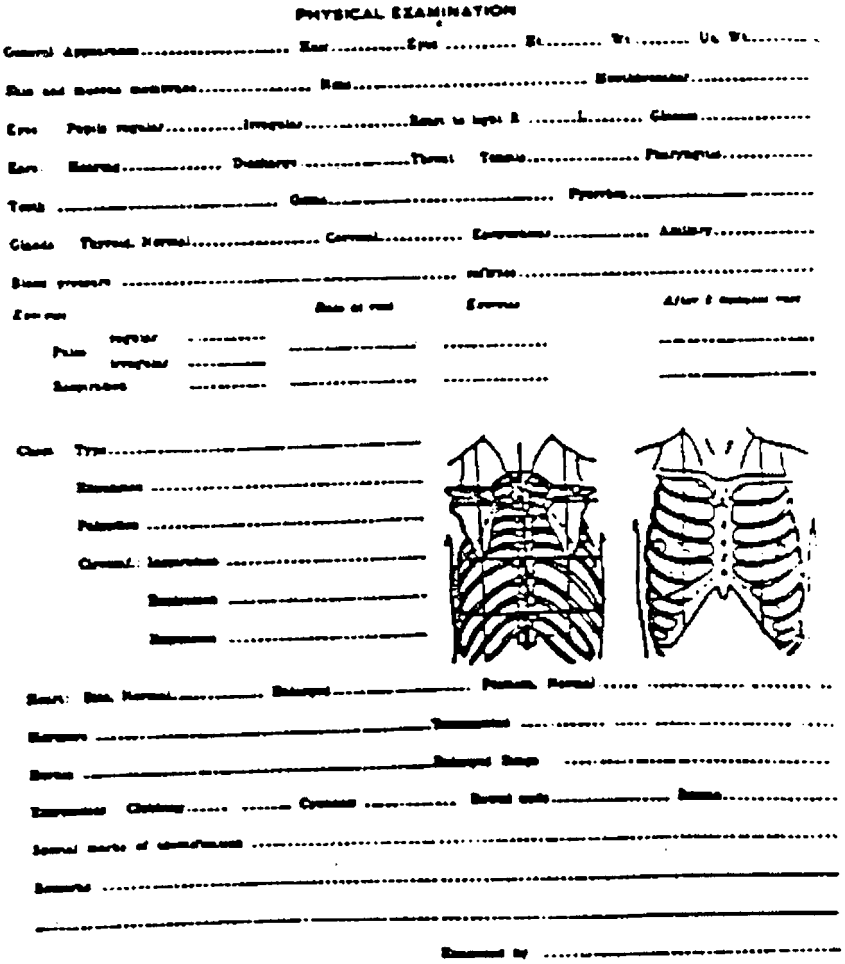

PHYSICAL EXAMINATION

FIGURE 38.—FORM USED FOR RECORDING PHYSICAL FINDINGS

basis of lung-field markings, and similar calculations were made. The decrease in average chest expansion cannot be attributed to any lack of large-chested men in the groups with high dust exposure or advanced pulmonary fibrosis. On the contrary, the average chest girth increases slightly with increasing dust exposure and with increasing pulmonary fibrosis. Several factors may have contributed to the

latter phenomenon and the available data provide no means for discriminating between them. Since the trend is slight it may be disregarded. The ratio of chest expansion to chest girth decreases with increasing dust exposure and with increasing pulmonary fibrosis.

TABLE 30.—*Relation of average chest expansion to dust exposure in male asbestos workers*

	Dust exposure million particle years			
	Less than 25	25 to 45	50 to 99	Over [illegible]
Chest expansion, inches	3 12	3 [illegible]	2 [illegible]	2 [illegible]
Girth at expiration, inches	34 [illegible]	34 [illegible]	34 [illegible]	34 [illegible]
Ratio of expansion to girth	.092	.091	.079	.059

TABLE 31.—*Relation of average chest expansion to lung-field markings in male asbestos workers*

	Lung-field markings			
	First-degree [illegible]	[illegible]	First-degree [illegible]	Second-degree [illegible]
Chest expansion, inches	3 [illegible]	3 [illegible]	2 [illegible]	2 [illegible]
Girth at expiration, inches	34 [illegible]	34 [illegible]	34 [illegible]	34 [illegible]
Ratio of expansion to girth	.088	.076	.059	.059

Chest expansion tends to decrease somewhat with advancing age, but the average chest expansion is less in asbestos-exposed men than it is in men of comparable age who have not been so exposed.

TABLE 32.—*Average chest expansion of males*

Age	Exposed to [illegible]		Employed in asbestos textile factories but not exposed to dust		Exposed to asbestos dust	
	Number of men in age group	Average chest expansion	Number of men in age group	Average chest expansion	Number of men in age group	Average chest expansion
		Inches		Inches		Inches
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

Externally, the thorax shows evidence of limited respiratory movement; the supra- and infra-clavicular fossae are conspicuous; the anterior-posterior diameter of the chest is increased; the substernal angle may be more obtuse; evidence of wasting or loss of subcutaneous fat may be present; certain of the accessory muscles of respiration

may appear to be hypertrophied. In other words, the general appearance of the chest is that of fibroid phthisis plus emphysema. Among the conditions that may reduce chest expansion are pleural adhesions and the relative inelasticity of fibrotic lung tissue.

RESPIRATORY RATE

The direct effect of pulmonary fibrosis on respiratory rate may be seen in table 33. Before the exercise test the respiratory rate of males with ground-glass lung-field markings was higher, but not significantly so, than the respiratory rate of males with first degree linear exaggeration of pulmonic markings. The exercise test consisted of having the subject stand with one foot on a box 14 inches high and to raise his other foot to the level of the top of the box 25 times in 30 seconds. The respiratory movements were counted during a 15-second period preceding the test, during a 15-second period immediately after exercise, and during a 15-second period 2 minutes after the test. Exercise raised the respiratory rate of all four groups of men, but the rise in respiratory rate was dependent upon the degree of pulmonary fibrosis, the men with ground-glass lung-field markings being affected most. After 2 minutes' rest, the fall in respiratory rate was again dependent on pulmonary fibrosis. The respiratory rate returned most closely to the preexercise rate in men with normal pulmonic markings or with first-degree linear exaggeration of lung-field markings. The return to the preexercise rate was slower in men with asbestosis (ground-glass lung-field markings).

TABLE 33.—*Relation of respiratory rate to lung-field markings in male asbestos workers*

Average respiratory rate	Lung-field markings			
	First-degree linear	Second-degree linear	First-degree ground-glass	Second-degree ground-glass
Number of men examined	260	62	31	
Before exercise	19.45±0.17	19.32±0.32	20.23±0.50	21.70±0.71
After exercise	25.04±.24	25.32±.63	26.14±1.05	27.[illegible]
2 minutes after exercise	20.41±.17	21.29±.32	21.45±.76	21.[illegible]

TABLE 34.—*Influence of anthracosilicosis on the respiratory rate of anthracite miners*[1]

Average	Examined in chest roentgenology survey	Anthracosilicosis
Number of men examined	[illegible]	[illegible]
Rate before exercise	19.72±0.10	20.41±0.30
Rate after exercise	26.09±.13	27.[illegible]
Rate after rest	22.21±.10	23.[illegible]

[1] These data were not included in Public Health Bulletin No. 251.

Similar relations were found in a study of anthracite miners. In men with anthraco-silicosis the respiratory rate was definitely higher than in essentially normal men before exercise, after exercise, and after rest.

CLUBBING OF FINGERS AND CURVING OF NAILS

Many asbestos-exposed workers, particularly those with long dust exposures, had a deformity of the finger nails. The terminal phalanges may become so bulbous that the finger resembles the end of a drumstick, a condition conveniently described by the term clubbed fingers. Usually it is accompanied by a downward curving of the end of the nail.

The affection is almost always bilaterally symmetrical and often involves the toes as well as the fingers. It is common in pulmonary tuberculosis. In bronchiectasis, empyema, and congenital heart disease it may be seen in its most exaggerated form. Such exaggeration, however, was comparatively rare in this study. In a study of 2,711 (31) anthracite workers, the deformity, regardless of degree, was observed in about a third of the workers having anthraco-silicosis, but was present in only 4 percent of the controls. The real significance of the affection has not been determined. Norris and Landis (62) state that in all probability it is a manifestation of long-standing toxemia associated with phthisis or bronchiectasis. The bronchiectatic and emphysematous changes noted in the asbestotic lung at autopsy probably account to a great extent for the occurrence of the change in the workers with asbestosis.

TABLE 33.—*Number and percent of asbestos-exposed workers who had clubbed fingers or curved finger nails, classified by lung-field marking*

Degree of clubbing and curving	Lung-field markings								Total
	First-degree linear		Second-degree linear		First-degree ground glass		Second-degree ground glass		
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	
Total	313	100	79	101	50	100	22	101	464
Normal	267	85	62	79	31	62	14	64	374
+	42	13	10	13	13	26	6	27	71
+ +	4	1	5	6	5	10	2	9	16
+ + +	1	0	2	3	1	2	0	0	4

Five controls (6 percent) had curving of the nails. This percentage was much higher (19.2) in the asbestos-exposed group. Thirty-nine percent of the workers with first- or second-degree ground-glass lung-field markings had this condition.

HEART DEFECTS

Taking age into consideration, blood pressure values conformed closely to the values found on examination of 10,143 male industrial workers (66).

Heart defects were no more numerous than in industrial groups in general. They were tabulated in relation to dust exposure and pulmonary fibrosis in the same way as the impairments just dis-

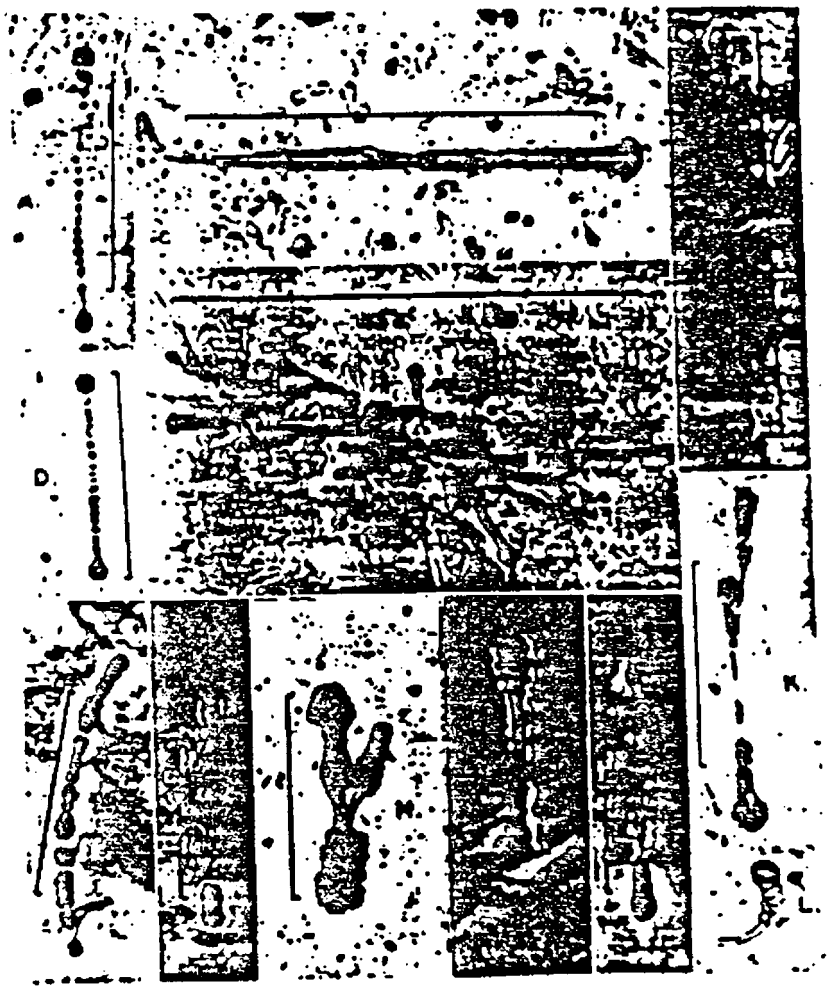


FIGURE 39.—PHOTOMICROGRAPHS OF ASBESTOSIS BODIES FOUND IN SPUTUM. ENLARGED 530 TIMES EXCEPT E, WHICH IS ENLARGED 310 TIMES. A SCALE RULED IN UNITS OF 50 MICRONS, HAS BEEN DRAWN BESIDE EACH ASBESTOS BODY

cussed, but no simple relationships could be found. A careful cardiac study (including electrocardiograms) was made on one group of asbestos workers. Many workers with advanced cases of asbestosis had normal hearts and no evidence was found which would relate heart impairments to asbestos dust exposure.

ASBESTOS CORNS

Asbestos corns are wartlike callosities, 1 mm to 5 mm in size, usually located on the palmar surfaces of hands or fingers and occasionally on soles of feet or on the legs (see fig. 39a). Their presence on the palmar surfaces of the hands is explained by the fact that workers, on breaking threads of asbestos, force tiny slivers of asbestos under the skin where it initiates a foreign body response. This response is characterized by hyperplasia and hyperkeratization. Table 36 shows that the occupations in which asbestos corns are most common are ones in which workers test the tension of moving threads with their fingers, or in which they are obliged to break threads with their hands or to knot threads together.

TABLE 36.—*Percentage of asbestos workers who had asbestos corns, classified by occupation*[1]

Occupation	Number employed	Number with asbestos corns	Percent with corns
Twisters	[illegible]	25	[illegible]
Drawers	[illegible]	[illegible]	[illegible]
Twine and braid [illegible] weavers	[illegible]	11	[illegible]
Inspectors, menders, and finishers	[illegible]	[illegible]	[illegible]
Cloth weavers	[illegible]	[illegible]	[illegible]
Crushers	[illegible]	[illegible]	[illegible]
Yarn spoolers and winders	[illegible]	11	[illegible]
Mule and ring spinners	[illegible]	[illegible]	[illegible]
Carders	[illegible]	[illegible]	[illegible]
Preparers, stenographers, and clerks	[illegible]	[illegible]	[illegible]
Pickers and pilers	[illegible]	[illegible]	[illegible]
Willowers and slubbermen	[illegible]	[illegible]	[illegible]
Card[illegible]	[illegible]	[illegible]	[illegible]
Foremen and [illegible]	[illegible]	[illegible]	[illegible]
Carpenters, yarnmen, and night watchmen	[illegible]	[illegible]	[illegible]
Managers and superintendents	[illegible]	[illegible]	[illegible]
Preemployment	[illegible]	[illegible]	[illegible]
Total	[illegible]	[illegible]	[illegible]

[1] Does not include 47 recently discharged workers.

Microscopically, Dewirtz (10) states that the corns present a picture of slow chronic local irritation. The horny and Malpighian strata of the skin are increased. Round-cell infiltration is noted particularly in the papillary layer of the dermis. Asbestos fibers or crystals are also seen and they are regarded by Dewirtz as the agent responsible for the thickening of the rete mucosum, basal layers, and the giant-cell formation. Gloyne (16) has also found asbestos fiber in the corns but noted that asbestosis bodies of the type observed in asbestosis lungs and sputum were absent. The workers have themselves called these little tumors "asbestos corns," or asbestos warts. Not infrequently the corns become very tender, particularly when pressure is applied to them in the course of handling tools or machinery. On account of the pain, the workers will attempt to remove the spicule of mineral with a pen knife or some other sharp instrument. Should the fiber be incompletely removed, it recurs with greater hyperplasia

because of the added trauma. Most of the workers so afflicted insist that every bit of fiber must be removed before the corn is cured. Only occasionally, however, is medical relief sought, and in such cases Dewirtz (10) advises complete surgical removal.

LABORATORY FINDINGS

ASBESTOSIS BODIES

Unusual structures have been observed by several investigators on histological study of the lung tissue of men who had died from asbestosis but no detailed description appeared until 1927 when Cooke (9) and McDonald (22) published photomicrographs and discussed the conditions under which asbestosis bodies are formed. Their papers apparently awakened interest in the subject and several papers on asbestosis bodies appeared soon afterward.

The following review of the literature on asbestosis bodies is far from complete, but it will serve to indicate the kind of information that has been put on record thus far. There is good evidence to show that these bodies originate as a cellular response to an inhaled asbestos fiber. The core of the body is an asbestos fiber which is surrounded by protein de[illegible]its. Apparently asbestosis bodies are formed only in the lungs. [illegible]ney have been recovered from the feces (36), suggesting that they pass unchanged through the gastrointestinal tract. They have not been demonstrated in the asbestos corns which form on the hands of asbestos workers (10, 57a), nor in the peritoneum of experimental animals (57) into which the dust has been injected. Gardner (57) found the "bodies" in the lungs of dusted guinea pigs, but for some unaccountable reason found them to be sparse in similarly exposed rabbits. The bodies can usually be demonstrated in the sputum of patients with asbestosis, although, according to Lynch and Smith (38), the sputum may be negative on repeated examinations even in well-established cases. For cases of this type, lung puncture (40) has been suggested as a diagnostic procedure.

Asbestosis bodies are variable in form (fig. 39*a*) and in size (table 37). Characteristically, they are slender, elongated, segmented structures with bulbous ends which give them a dumbbell or drumstick shape. Occasionally, a forked or Y-shaped body such as the one marked *H* in figure 39 is encountered. A rosette such as the one illustrated in the center of figure 39 was observed only once in the examination of sputum samples from 281 persons. Rosettes, however, are common in sections of lung tissue. Stewart, Tatersall, and Haddow (41) say,

> We suggest that the findings of a large number of clumps of asbestosis bodies in the sputum as here described is a clear indication of disintegration of lung tissue whether by a process of simple suppurative bronchopneumonia, as in our first case, or as a result of secondary tuberculous infection. In either case it is reasonable to conclude that there is also a definite underlying asbestosis

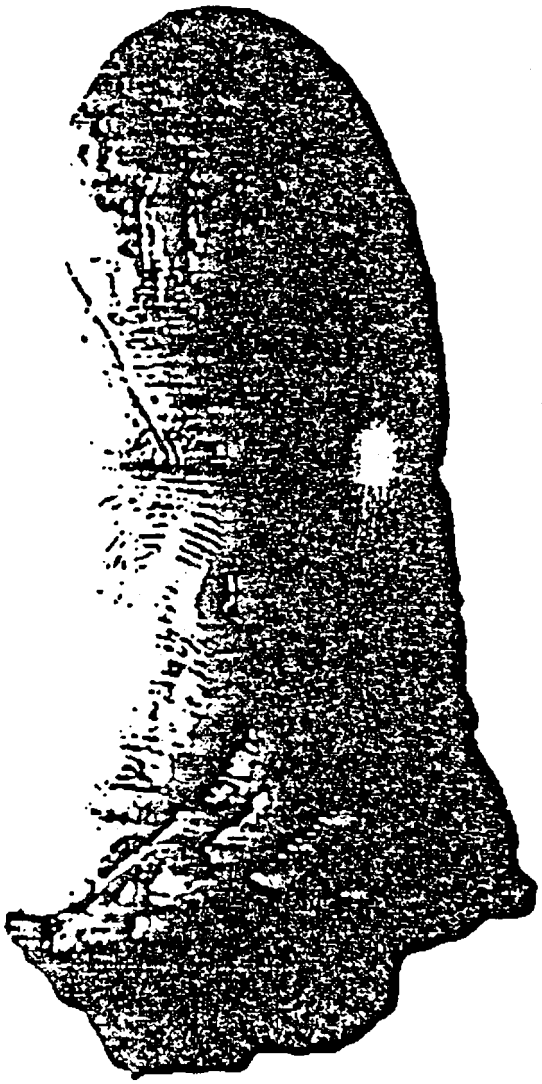


FIGURE 39A —ASBESTOS CORN ON THUMB OF 36-YEAR-OLD MAN EMPLOYED 1 YEARS IN THE CARD ROOM OF AN ASBESTOS TEXTILE FACTORY PREVIOUSLY EMPLOYED 14 YEARS IN COTTON AND WOOLEN TEXTILE MILLS PHOTOGRAPH OF MOULAGE 2X

The asbestosis bodies illustrated in figure 39 show some of the variations in size and shape seen in sputum smears. Five of these asbestosis bodies (*A, C, D, E, K*) were found on the same slide. This sample of sputum was obtained from a 37-year-old mule spinner who had been employed in the asbestos industry for 7½ years, exposed to an average dust concentration of 8 million particles per cubic foot. Because he made no complaint of any of the major symptoms of asbestosis and because his lung fields were second-degree linear, no diagnosis of asbestosis was made. The bodies labeled *G* and *H* came from another mule spinner, a 37-year-old man employed 2½ years in an average dust concentration of 7.9 million particles per cubic foot. Except for a cough he had none of the symptoms of asbestosis and his lung fields were essentially normal.

In several photomicrographs of the "asbestosis body," the asbestos fiber may be seen clearly. The bodies are somewhat fragile, and fragments (such as *L* in fig. 39) often occur. Unstained specimens are golden yellow or golden brown. They do not stain with the aniline dyes ordinarily used in histology, but they stain blue (prussian-blue reaction) when treated with potassium ferrocyanide.[4]

Asbestosis bodies can be demonstrated on sectioning [illegible] tissue. Since they arise as a tissue reaction to an inhaled asbesto[illegible]er, their size is important because it indicates the length of the asbestos fibers that may reach the lungs. The greatest length of the first 108 asbestosis bodies found during the study of prepared sputum specimens was measured on camera-lucida drawings. Every asbestosis body was measured and the frequency distribution of these measurements appears below.

TABLE 37.—*Frequency distribution of the lengths of the first 165 asbestosis bodies found in sputum smears*

Length in microns	Number of [illegible] bodies of the length specified	Length in microns	Number of [illegible] bodies of the length specified
Total measured	165	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]

[illegible] method was used in the general study of tissue lesions in sputum. A sample of sputum is digested with an equal volume of concentrated antiformin, centrifuged at [illegible] speed, the antiformin is poured off, and replaced with the following mixture: 2 percent potassium ferrocyanide, 1 part; 2 percent hydrochloric acid, 1 part. The tube is shaken and allowed to stand for [illegible] hour. It is then centrifuged and the material mounted in a wet preparation. With this technique the asbestos bodies are stained brilliant blue. If mounted without treatment with potassium ferrocyanide and hydrochloric acid, the bodies are golden yellow in color.

The asbestos bodies ranged in length from 10 to 128 microns; their average length was 35.1 microns. After completing this series, routine measurements were discontinued since this seemed to be a large enough sample to indicate the sizes of asbestosis bodies most frequently found. The largest body found in subsequent study of sputum specimens measured 184 microns in length. There seems to be no completely satisfactory explanation for the absence of asbestosis bodies measuring less than 10 microns in length.

Asbestos appears to differ from other pneumoconiosis-producing dusts with respect to the size of the largest particle that may reach the lungs. The largest reported upper limit of size for rock dust in the gold mines of South Africa and in the granite (48) and anthracite (51) industries in this country is 14 microns. The data of table 37 are in harmony with a concept that has been gaining support in recent years, namely, that particles beyond 10 microns in size reach the alveoli. Measurements indicate that the diameter of air sacs may be from 150 to 300 microns in adults, or, in other words, of such a size that asbestos fiber or resulting asbestosis bodies may be readily accommodated.

Asbestosis bodies were found more frequently in the sputum of persons who complained of a persistent cough than in the sputum of persons without a cough. This is a reasonable finding, since the act of coughing tends to raise materials from the lungs. Half of the 56 people who complained of dyspnoea had asbestosis bodies in their sputum. Less than a fifth of the 225 asbestos-exposed people who did not make a complaint of dyspnoea had asbestosis bodies in their sputum. Almost the same relationship holds for a complaint of cough.

Asbestosis bodies were found in the sputum of 46.9 percent of persons whose condition was diagnosed as asbestosis. A much smaller proportion, 24.3 percent, of essentially normal asbestos-exposed persons had asbestosis bodies in their sputum. Thus, it appears that the presence of asbestosis bodies is evidence of exposure to asbestos dust, but it is not necessarily a diagnostic sign of asbestosis. Asbestosis bodies were found in the sputum of persons who had been exposed to asbestos dust for as short a time as 3 months. Of the workers employed less than 1 year, 22.5 percent had asbestosis bodies in their sputum. A larger percentage, 38 percent, of workers employed more than 5 years had these bodies in their sputum. In evaluating these percentages it must be remembered that they are based on the examination of a single sputum specimen from each individual. If several specimens could have been collected and examined, the percentage of persons with asbestosis bodies would almost certainly be much higher.

No asbestosis bodies were found in sputum samples from any of the 76 persons tested who were not exposed to asbestos dust.

The incidence of asbestosis bodies increased with increasing dust exposure.

TABLE 38.—*Relation of dust concentration to occurrence of asbestosis bodies*

Weighted dust exposure	Number exposed	Number with asbestosis bodies	Percent with asbestosis bodies
Total exposed.....	251	[illegible]	[illegible]
0 to 4.9	[illegible]	[illegible]	[illegible]
5 to 9.9	[illegible]	[illegible]	[illegible]
10 to 19.9	[illegible]	[illegible]	[illegible]
20 and over	[illegible]	[illegible]	[illegible]

RESULTS OF TESTS ON URINE

Each person employed in the three asbestos plants studied was asked to bring a 24-hour sample of urine in a metal container provided for that purpose. In addition to the standard clinical tests, the silica content was determined by the method of King and Dolan (59).[1] Of the 416 persons exposed to asbestos dust at the time of the study, urine analyses are available for 403, and these results have been kept separate from the results of urine tests made on 67 persons whose work did not expose them to asbestos dust.

Albumin was found in the urine of 10 of the 248 white males tested; in the urine of 10 of the 98 white females; and in the urine of 3 of the 57 negro males tested. Only one sample of urine (from a white male) contained sugar (Benedict's test). These rates of incidence are no higher than in the controls and, insofar as published data are available for comparison, they seem to be no higher than the rates generally found in industrial populations.

TABLE 39.—*Relation of urinary silica to dust exposure*

Urinary silica	Controls	Dust exposure, million particles per cubic foot		
		[illegible]	[illegible]	[illegible]
Milligrams silica per liter.....	[illegible]	[illegible]	[illegible]	[illegible]
Milligrams silica per day.....	[illegible]	[illegible]	[illegible]	[illegible]
Number tested.....	[illegible]	[illegible]	[illegible]	[illegible]

Silica was present in concentrations ranging from [illegible] per 100 cc. Average urinary silica concentrations for [illegible] persons whose dust exposure varied widely [illegible] that there was any simple relation between the exposure to [illegible] dust (which consists of hydrated magnesium silicate) and the concentration of urinary silica. Rather, it appears that [illegible]

[1] The [illegible] method [illegible] amount of silica present.

dust-exposed persons fluctuate above and below the value for the controls. The total daily silica output was calculated by multiplying silica concentration by the volume of urine excreted in 24 hours; these values (table 39) also show no simple relation to dust exposure.

Urinary silica analyses are available for two other groups of people whose diet resembled that of asbestos workers in consisting largely of vegetables. The urine of seven male college students living in the same city averaged 2.6 mg per 100 cc (based on single specimens, not 24-hour samples). An earlier survey of an industrial plant in Tennessee made by the Public Health Service [4] showed that the daily silica output averaged 29.59 ± 0.20 mg, a value very close to the mean silica output of nonexposed asbestos workers.

Diet plays an important part in influencing urinary silica concentration as King and Dolan (39) have demonstrated on experimental animals. A predominantly vegetable diet, with its high silica content, leads to a high urinary silica output. It may be that the dietary intake of silica was so great as to totally mask any possible effect of silicate absorption through the lungs. In any case, the phenomenon reported by King and Dolan for gold miners (39) and by the Public Health Service (51) for anthracite miners, namely, a higher silica excretion in men exposed to silica dust than in nonexposed controls, does not appear here. The urinary silica concentration of the asbestos workers is higher than that reported for the two occupational groups just mentioned, and this appears to be largely due to dietary differences.

Silica concentration increased with increasing specific gravity. The silica content is, of course, too low to influence the specific gravity directly. Instead, it appears that the two values may vary concomitantly, and that the drinking of large quantities of water, for instance, would lower both the specific gravity of the urine subsequently excreted and its silica content as well.

TABLE 40.—*Relation of silica content of urine to specific gravity*

Specific gravity	Average silica content	Number of samples
	Mg per 100 cc	
1.000 to 1.009	2.14	29
1.010 to 1.019	2.66	104
1.020 to 1.029	3.20	104
Over 1.030	3.10	21

THE DIAGNOSIS OF ASBESTOSIS

In preceding sections of this bulletin the signs and symptoms of asbestosis have been treated one at a time or two at a time in order to show their relationship to dust exposure. In this and the two following sections the symptom complex of asbestosis and its relation to dust exposure will be discussed.

[4] United States Steel

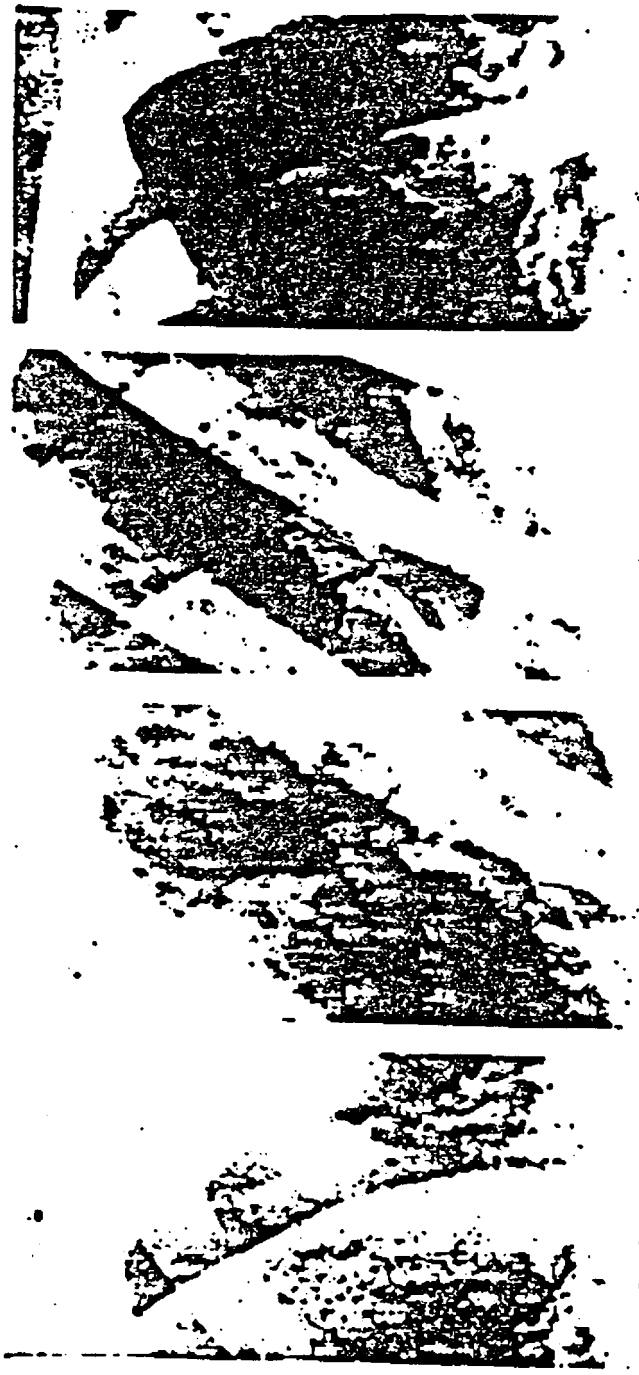


FIGURE 40.—PORTIONS OF CHEST X-RAY FILMS WHICH ILLUSTRATE THE SUCCESSIVE CHANGES BROUGHT ABOUT BY LONG-CONTINUED INHALATION OF ASBESTOS DUST

In order to show these markings on a larger scale than would be possible if the entire film were reproduced and to illustrate this system of classification, figure 40 has been prepared.

Symptoms.—Dyspnoea, cough, blood-tinged sputum, pain in the chest, weakness, decreased chest expansion, curving of the finger nails and adventitious chest sounds are symptoms which also occur in silicosis. It is possible to differentiate pulmonary asbestosis from silicosis by history of exposure to asbestos, the presence of asbestosis[1] bodies in the sputum or lungs, and by the type and distribution of the X-ray markings. With reference to asbestosis bodies in the sputum, the data show that they may appear after 3 months' exposure. They show evidence of exposure to dust, but not established disease. Lynch and Smith (38) reported difficulty in demonstrating the bodies in an advanced case. The presence of rosettes as illustrated in figure 39 has been reported to be of greater diagnostic significance

No advanced cases of asbestosis of the bed-ridden type were observed in the present study. Reports (13, 20, 26, 32) of fatal American asbestosis cases which were relatively uncomplicated by the presence of other disease processes are essentially similar to reports of uncomplicated British cases (9, 14, 34, 35). Patients in the terminal stage are usually reported to be emaciated, markedly dyspnoeic, cyanotic, and usually have a severe cough with wiry mucoid sputum.

REPRESENTATIVE CASE HISTORIES

Plates made from representative X-ray films appear as figures 41 to 51. Roentgenograms of a normal chest (figure 41) and of a chest showing advanced silicosis (figure 42) have been included for purposes of comparison. In figures 43 to 51 which represent asbestotic patients, an abstract from the case history and physical examination accompanies the X-ray. In viewing these roentgenograms it should be remembered that the ground-glass lung-field markings that are characteristic of asbestosis are exceedingly difficult to photograph and reproduce. It will be noted that the distribution of the shadows is not quite as general as in silicosis.

[1] Wade has reported bodies that similar bodies in the sputum of anthracite miners, but they seem to differ morphologically from asbestosis bodies.

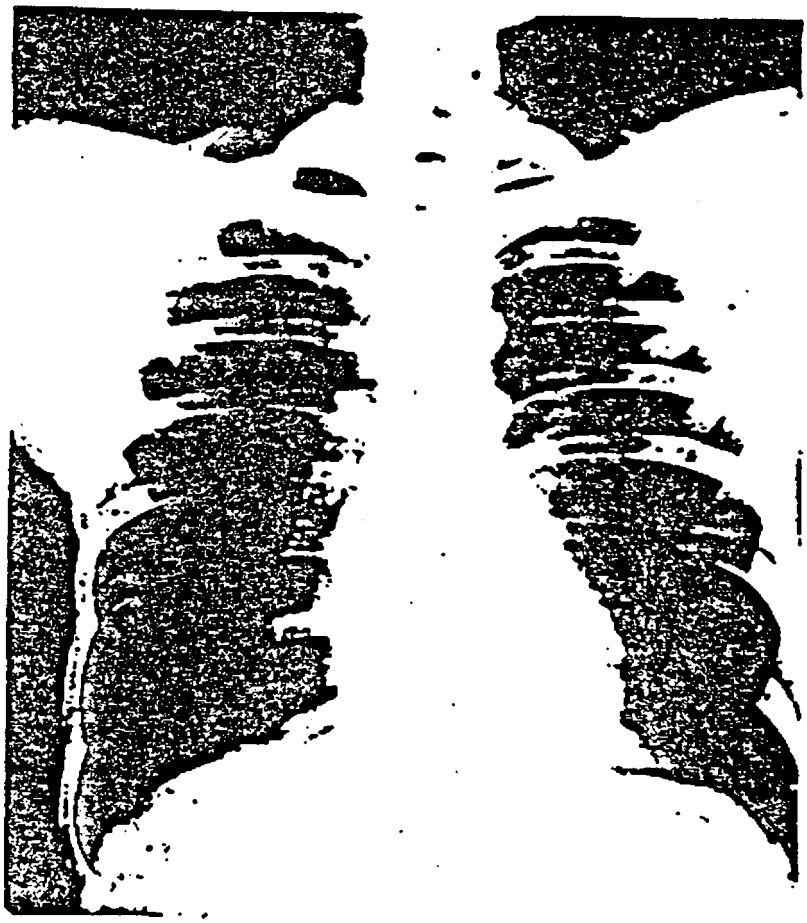


FIGURE 41.—NORMAL PULMONIC MARKINGS. WHITE MALE, AGE 23.

Note the linear type of pulmonic markings usually seen in the chest roentgenogram of an average industrial worker not exposed to a dust hazard. This man had worked 3 years in the decorating department of a pottery and 2 years at general outdoor labor.

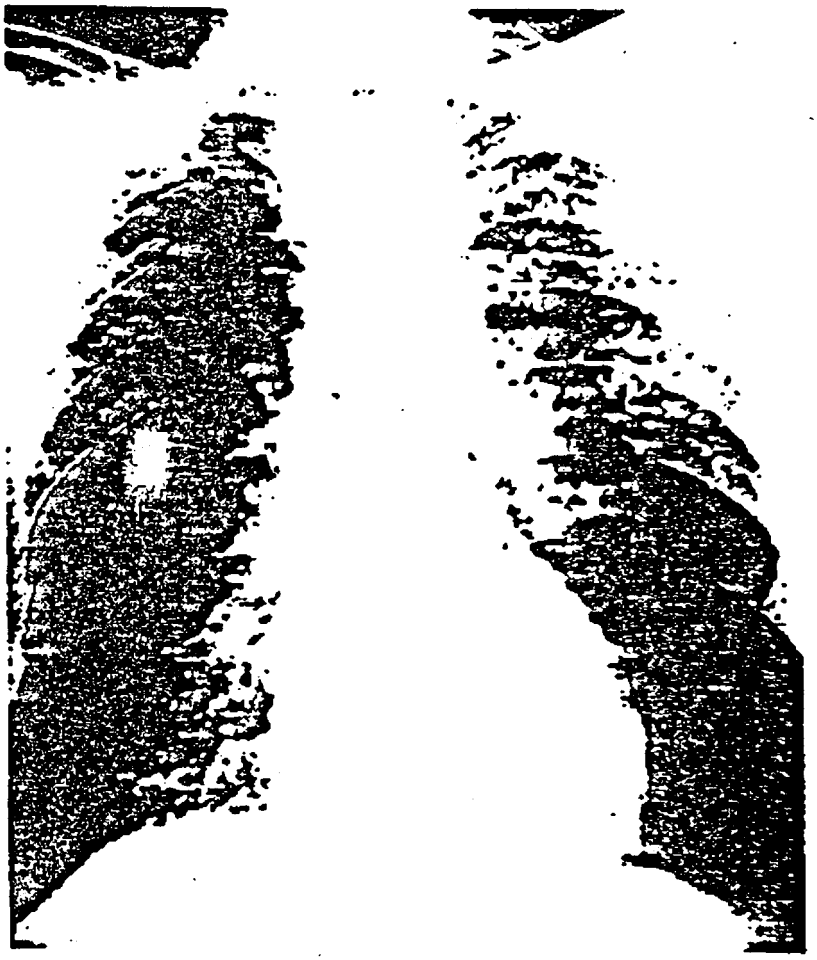


FIGURE 42.—SILICOTIC PULMONIC MARKINGS. WHITE MALE, AGE 51

Note coarse nodular appearance, with beginning coalescence of these shadows in the upper half of the left. Moderate emphysema both bases and slight mediastinal distortion. Nodular markings of the type here shown were not noted in asbestos employees.



FIGURE 43.—EARLY ASBESTOSIS. WHITE MALE, AGE 29

Occupational history.—Carrier (asbestos), 4½ years; nonindustrial trades, 5½ years.
Estimated dust exposure.—(a) 35.3 million particles per cubic foot. (b) Million particle years, 159.

No significant past medical findings or complaints.

Physical examination.—Intermediate, slender white male. Height, 67½ inches; weight, 142 pounds. Rather long chest; expansion, 2 inches. No changes in fremitus, breath sounds, or percussion note. Blood pressure, 108/78. Pulse rate, 98, 120, and 80 before, immediately after, and 2 minutes after functional exercise. Urinary silica, 5.2 mg per 100 cc (24-hour output, 34.3 mg). Routine urinalysis negative; specific gravity 1.022.

X-ray.—First-degree ground-glass appearance not quite obliterating the coarse linear markings. Note the fine, closeset reticular markings just off each hilum and the apices remaining quite clear.

Diagnosis.—Early asbestosis without symptoms complicated by hypertensive heart disease.

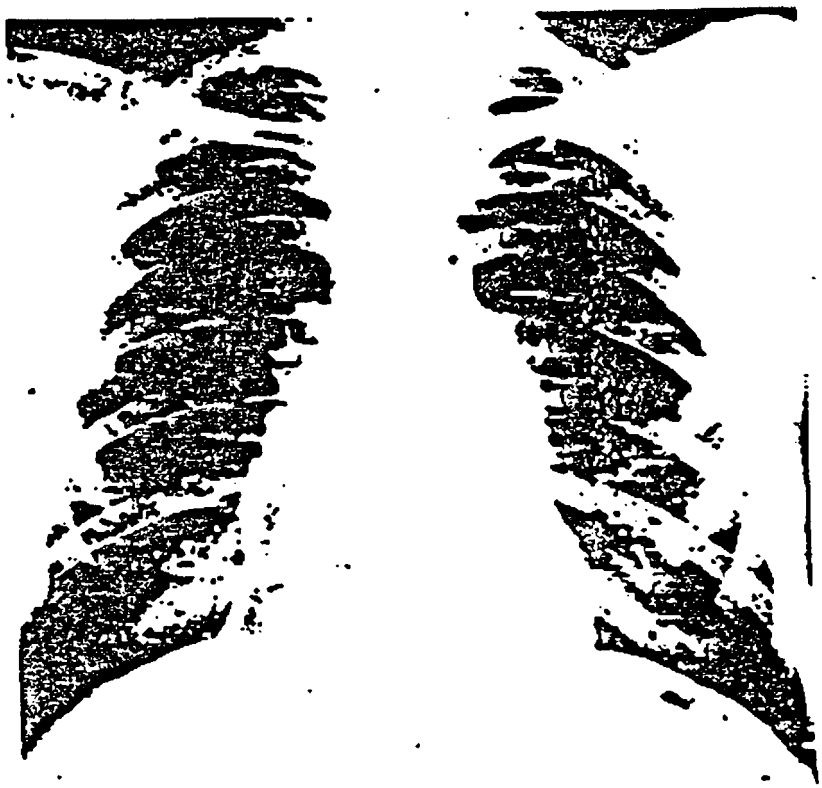


FIGURE 44.—EARLY ASBESTOSIS. WHITE MALE. AGE 39.

Occupational history.—Mule spinner (asbestos), last 15 years; roving attendant (asbestos), 1 year; nondusty trades, 8½ years.

Estimated dust exposure.—(*a*) Weighted average, 10.5 million particles per cubic foot. (*b*) Million particle years, 168.2.

Past medical.—Shrapnel wound right parietal region of head, October 1918. Bilateral pneumonia as a child.

Complaints.—Slight weakness.

Physical examination.—Intermediate build, fairly well nourished. Height, 66½ inches; weight, 144 pounds. Chest expansion, 3½ inches. Blood pressure, 130-82. Pulse rate, 88, 120, and 84, and respiratory rate, 16, 24, and 18 (respectively before, immediately after, and 2 minutes after functional exercise test). Intermediate chest type with slight increase in A-P diameter of chest. Few nonpersistent crackling rales at right base. Asbestos worn on palmar surface of left wrist. Slight cyanosis of lips and nails. A dusky red cast to eyes. Sputum negative for *M. tuberculosis* and asbestos bodies. Urinary silica, 3.6 mg per 100 cc (24 hours, 21.5 mg).

Fluoroscopy.—Moderate (2—) limitation of diaphragmatic excursion. Hila moderately increased in size and density.

Xerox.—Note the fine-to-coarse granularity or ground-glass appearance, not obliterating the linear markings, giving general reticular appearance to lung fields. Generalized fibrosis 1—.

Diagnosis.—Asbestosis 2.

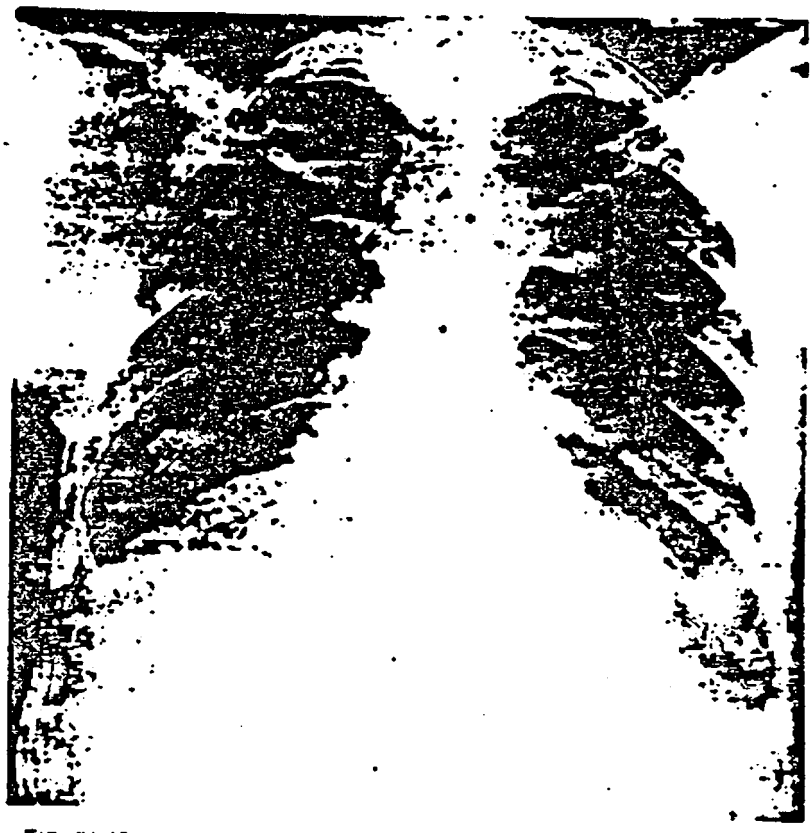


FIGURE 45.—MODERATELY ADVANCED ASBESTOSIS. WHITE MALE. AGE 46.

Occupational history.—Brassicloth weaver (asbestos), 7 years 3 months. Previously in nondusty trades, 15¼ years including 10 years as farmer.

Estimated dust exposure.—(a) Weighted exposure, 18 million particles per cubic foot. (b) Million particle years, 109.2.

Past medical.—Tonsillectomy, 1915. Pleurisy, right, March 1926.

Complaints.—Dry, hacking morning cough for 3 years productive thick mucoid sputum. Slight dyspnoea, 2 to 3 years. Blood-tinged sputum.

Physical examination.—Height, 70½ inches. Weight, 160 pounds. Blood pressure, 150 ±2. Pulse rate, 72, 88, and 68; respiratory rate, 24, 28, and 24 before, immediately after, and 2 minutes after functional exercise test. Patient completed only about half of prescribed exercise. Chest expansion, 1 inch. Shoulders somewhat rounded. Crackling rales heard over both lungs on quiet breathing, and these persist after cough; 2+ curved finger nails; 3.1 mg silica per 100 cc single specimen.

Fluoroscopy.—Left side of diaphragm moves more freely than right; 2+ limitation of excursion, especially on right.

X-ray.—Shows second-degree ground-glass appearance obliterating usual linear pulmonic markings. It also shows preponderance of fibrosis in the bases of lung and shaggy appearance of heart shadow.

Diagnosis.—Asbestosis 3 (moderately advanced).



FIGURE 46.—MODERATELY ADVANCED ASBESTOSIS. WHITE MALE. AGE 40.

Occupational history.—Mule spinner (asbestos), last 13 years; nondusty trades, 4½ years; idle, 4½ years.

Estimated dust exposure.—(a) Weighted average, 13.3 million particles per cubic foot. (b) Million particle years, 173.7.

Past medical.—Influenza and pneumonia, 1919. Frequent disabling colds.

Complaints.—None.

Physical examination.—Intermediate white male. Height, 70½ inches; weight, 156 pounds. Intermediate chest shape; expansion, 2 inches. Dry, musical rales over bases of lungs. Urinary silica, 3.6 mg per 100 cc (24-hour output, 80 mg). Sputum not examined.

Fluoroscopy.—Showed slight limitation of diaphragmatic excursion right and left with first-degree diffuse ground-glass appearance of the lung fields.

X-ray.—Shows first-degree granular appearance not obliterating the linear markings. It illustrates the fine reticular fibrosis associated with this condition and the tendency to be most dense at the bases of the lungs.

Diagnosis.—Asbestosis 3.



FIGURE 47.—MODERATELY ADVANCED ASBESTOSIS. WHITE MALE, AGE 49.

Occupational history.—Mule spinner (asbestos), 25 years, working 10 to 11 hours a day; nondusty trades, 9 years.

Estimated dust exposure.—(a) Weighted average, 7.9 million particles per cubic foot. (b) Million particle years, 221.2.

Past medical.—Typhoid fever at age of 11. Influenza, 1918. Dry pleurisy, right in 1931; second attack in 1935 on left. More than average number of chest colds each year. Kidney colic, 1931.

Complaints.—Slight dyspnoea, especially in climbing stairs. Slight morning cough past 2 to 3 years productive of small amount of white frothy sputum.

Physical examination.—Tall, fairly well-developed man. Height 70 inches, and weight, 172 pounds. Blood pressure, 140 80. Pulse rate, 80, 108 and 80; respiratory rate, 24, 32, and 24 (respectively before, immediately after, and 2 minutes after functional exercise test). Chest is long, and well developed with some flaring of the costal margins. Chest expansion, 1½ inches. Numerous crackling rales at bases on deep breathing and after cough. Sputum (single specimen) was negative for acid-fast bacilli and asbestos bodies. Urinary silica, 4.2 mg per 100 cc (24-hour output, 23.0 mg).

Fluoroscopy.—Diaphragm almost fixed and lung fields interpreted as showing a first-degree ground-glass appearance.

X-ray.—Note the fine grainy or ground-glass appearance fairly well limited to the lower two-thirds of each lung field.

Diagnosis.—Asbestosis 3.

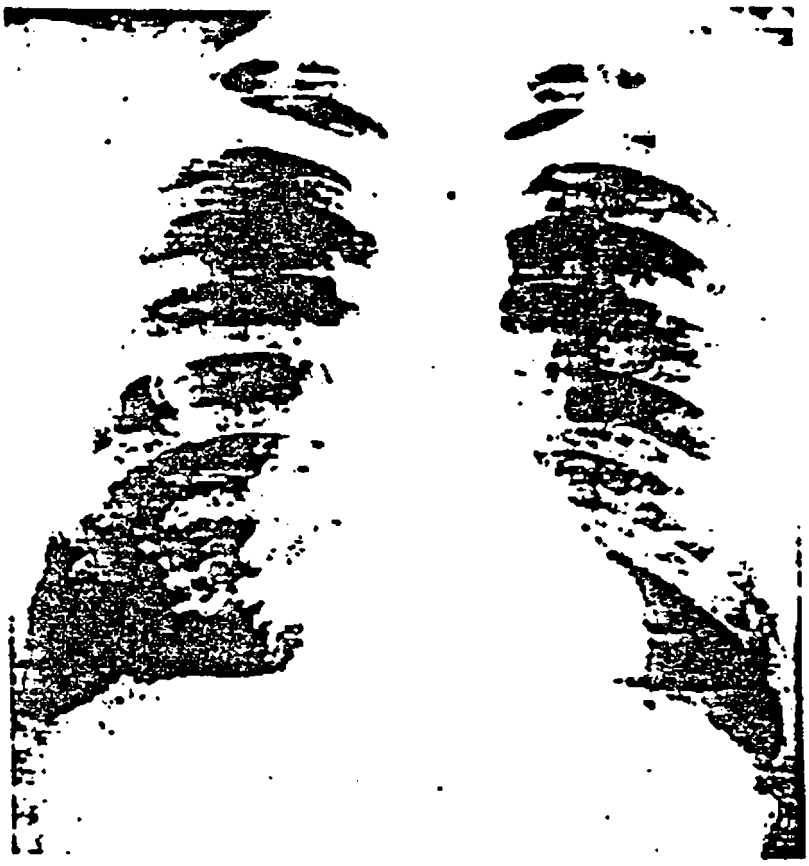


FIGURE 48.—MODERATELY ADVANCED ASBESTOSIS. WHITE MALE. AGE 39.

Occupational history.—Broadcloth loom room (asbestos), 14 years; nondusty trades, 7 years.

Estimated dust exposure.—(a) Weighted average, 40.7 million particles per cubic foot. (b) Million particle years, 439.7.

Past medical.—Bilateral pneumonia as a child. Sinus disease for the past 2 years. Typhoid fever, 1911.

Complaints.—Dry, hacking, morning cough productive of 2 ounces of white, viscid sputum, past 2 years. Shortness of breath, 2½ years.

Physical examination.—Fairly well-developed, slender, white man. Height, 72 inches; weight, 163 pounds. Blood pressure, 104/70. Pulse rate, 68, 76, and 68. Respiratory rate, 21, 30, and 30 (before, immediately after, and 2 minutes after functional exercise test). Chest is long, slender type. Expansion, 1¾ inches. Heart sounds are distant with a pulmonic second sound somewhat accentuated.

Fluoroscopy.—Peaking of the diaphragm both sides. 3 cm excursion upon forced respiration.

X-ray.—Note the deformity of diaphragm. This case is the only one showing definite deformity in the group. The lung fields were interpreted as showing accentuated granular diffuse grainy or ground-glass appearance with emphysema.

Diagnosis.—Moderately advanced asbestosis (asbestosis 3).

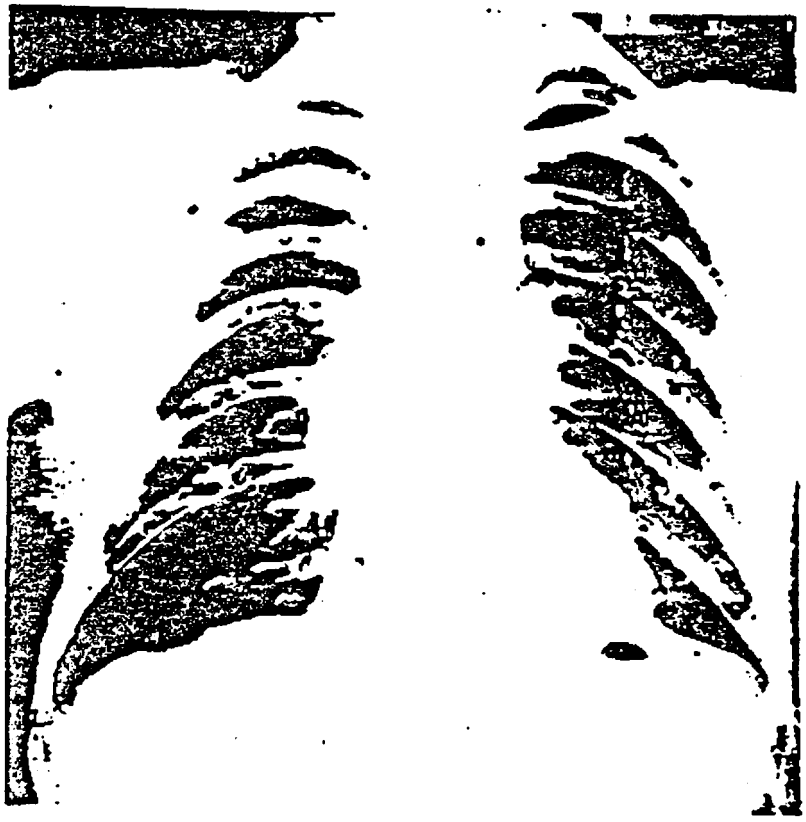


FIGURE 49.—ADVANCED ASBESTOSIS. WHITE MALE, AGE 48.

Occupational history.—Mule spinner (asbestos), 17½ years (1½ years in same room with carding machines; 11 years he worked 11 hours a day; nondusty trades, 17 years.

Estimated dust exposure.—(a) Weighted average, 10.6 million particles per cubic foot. (b) Million particle years, 103.8.

Past medical.—Frequent chest colds. Aerophagia for several years. Malaria about 1926. Influenza, 1929 and 1932.

Complaints.—Cough, 6 years' duration, possibly somewhat improved since quitting work in asbestos. Severe paroxysms of cough in morning, causing him to vomit frequently and productive of small amount of tenacious mucoid sputum. Moderate dyspnoea since 1928, unchanged. Lost 15 to 20 pounds in weight and associated weakness before quitting work in asbestos.

Physical examination.—Slender fairly well-developed and nourished white male. Height, 68 inches; weight, 131 pounds. Rather long narrow chest. Chest expansion, 1½ inches. Tactile and voice fremitus apparently normal. Slight impaired resonance lower half of each lung. Numerous persistent dry crackles mostly over lower portions of lungs. Faint breath sounds lower one-half right and left. 1+ curving of finger nails. Blood pressure, 135/80. Pulse and respiratory rate, respectively, 72, 100, and 68 and 28, 32, and 28. (Before immediately after, and 2 minutes after the functional exercise test). Moderate dyspnoea following this exercise. Urinary silica (single specimen), 2 mg per 100 cc. Sputum negative for acid-fast bacilli and asbestosis bodies.

Fluoroscopy.—2+ lag of diaphragmatic excursion right and left. Rather poor radiolucency lower lung fields.

X-ray.—Shows secondary-degree grain appearance with almost complete obliteration of lung markings. Concentrated fibrosis 2+.

Diagnosis.—Well-established asbestosis.

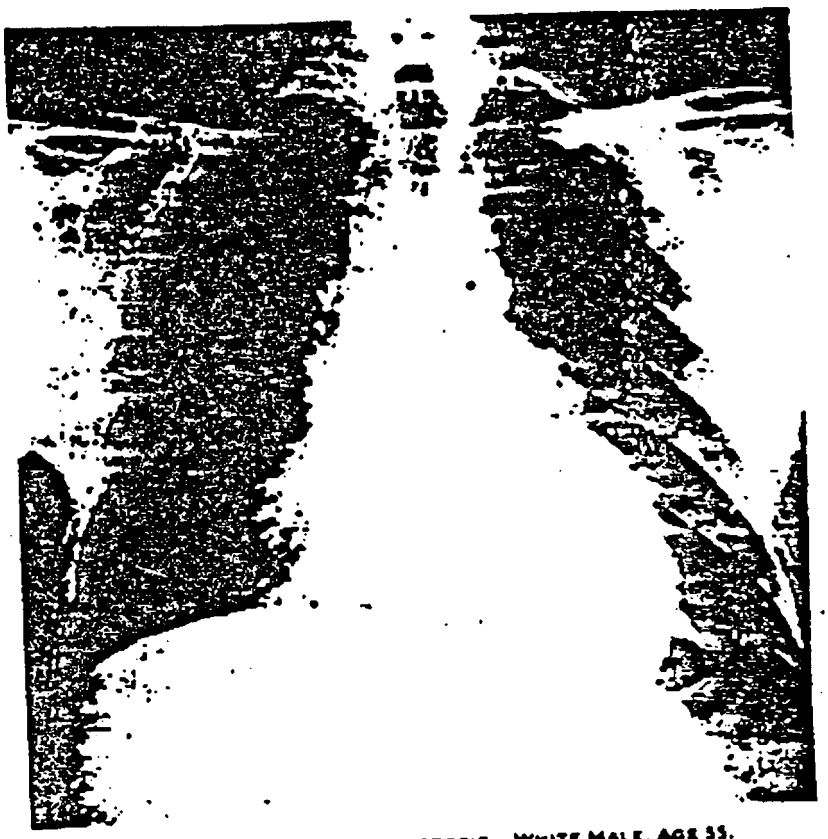


FIGURE 50.—ADVANCED ASBESTOSIS. WHITE MALE, AGE 55.

Occupational history.—Preparation department (asbestos), 14 years; insulation trades, 27 years.

Estimated dust exposure.—(a) Weighted average, 38.9 million particles per cubic foot. (b) Million particle-years, 363.

Past medical.—Maxillary sinuses drained in 1930. Typhoid fever at age 21.

Complaints.—Dry cough, 10 years, productive of 2 ounces of white sputum over a period of 24 hours. Rather severe dyspnea, 4 years. He must walk very slowly to prevent dyspnea. Occasional edema of ankles for 4 years.

Physical examination.—Height, [illegible]. Weight, 142 pounds. Blood pressure, 112/70. Moderate arteriosclerosis. Pulse rate, [illegible]. Respiratory rate, 48, 54, and 51 [illegible]. Patient stopped [illegible] exercise test. Patient stopped [illegible] exercise, but expressed a willingness [illegible]. Breathing is labored and shallow. [illegible] over the lower half of both lungs [illegible] with the aortic second sound [illegible] second. First sound at the apex is [illegible] 2+ curving of the finger nails.

Fluoroscopy.—Aortic shadow [illegible] 1+ increase in size. Diaphragm [illegible] 3 cm on forced excursion.

X-ray.—Shows diffuse [illegible] finely linear markings [illegible].

Diagnosis.—Advanced asbestosis.



FIGURE 31.—ASBESTOSIS WITH FIBROID TUBERCULOSIS. WHITE MALE AGE 38.

Occupational history.—Mule spinner (asbestos), 11 years 6 months; nondusty trades, 12 years.

Estimated dust exposure.—(a) Weighted average, 7.9 million particles per cubic foot. (b) Million particle years, 88.5.

Past medical.—Received sanatorium treatment for tuberculosis 8 months in 1930. Had pneumonia as a child. Appendectomy in 1916.

Complaints.—Dry cough past 5 years, worse at night. Slight dyspnea, 8 years.

Physical examination.—Very thin man. Weight, 131 pounds. Blood pressure, 100/70. Pulse rate, 72, 80, and 68. Respiratory rate, 16, 16, and 20 (before, immediately after, and 2 minutes after functional exercise test). Asthenic type of chest. Some deformity of chest due to scoliosis. Chest expansion, 2½ inches. Breath sounds are slightly increased over the extreme right upper. Very few dry rales right supra-scapula fossa. Heart: The pulmonic second sound is much greater than the aortic second.

Fluoroscope.—Diaphragm moves freely and equally on both sides. On forced respiration, expansion is 5 cm on right and 4 cm on left.

Diagnosis.—Moderately advanced asbestosis with arrested fibroid tuberculosis.

INCIDENCE OF ASBESTOSIS

Preceding sections of this bulletin have stressed the importance of the X-ray findings in establishing a diagnosis of asbestosis. The reason, of course, is that X-ray examination is the most direct means now available for detecting the primary effect of inhaled asbestos dust on the body; namely, initiation of interstitial fibrosis of lung tissue. Classification of asbestosis into a series of stages which follow one another in their development is a difficult matter because it depends to a great degree on the interpretation of relatively minor changes in the lung-field markings. Other investigators have encountered the same difficulty. Lanza, McConnell, and Fehnel (17) and Fulton, Dooley, Matthews, and Houtz (13) set up a category of doubtful cases of asbestosis. Stage 1 in the accompanying tabulation more or less corresponds to their category of doubtful cases. It comprises cases in which diagnosis rests largely on X-ray evidence. Stage 2 represents early cases of asbestosis, and stage 3 designates moderately advanced cases. More advanced cases (stage 4) are relatively infrequent. Stage 5 was reserved for bed-ridden cases.

TABLE 41.—*Relation of asbestosis to lung-field markings*

X-ray findings	Asbestosis absent	Stages of asbestosis				Total
		1	2	3	4	
Total	373	36	28	16	7	447
First-degree linear	267					267
Second-degree linear	65	11	3			79
First-degree round spots	6	13	22	9	4	54
Second-degree ground glass	6		1	5	2	14

Table 41 illustrates the relationship of stages of asbestosis to the X-ray findings. Although diagnoses of early stages of asbestosis were occasionally made in the absence of definite ground-glass lung-field markings when cough, dyspnoea, and objective findings were present in sufficiently advanced degree, diagnoses of the later stages of asbestosis were not made unless unmistakable ground-glass markings were present in the lung fields.

The incidence of asbestosis increases rapidly with increasing dust exposure. A fifth of the workers with an exposure of 50 to 99 million particle years, and half of the workers having an exposure of more than 100 million particle years had asbestosis. In comparison with anthracosilicosis these are exceedingly low dust-exposure values.

The relation of asbestosis to dust concentration and length of employment, in table 43, is of great practical importance because it shows what working conditions are most likely to cause asbestosis and how long a period of exposure may elapse before the disease becomes evident.

TABLE 42.—*Relation of asbestosis to total dust exposure* [illegible]

Diagnosis	Million particle years				Total
	Less than 25	25 to 49	50 to 99	Over 100	
Essentially normal	191	81	43	32	[illegible]
[illegible]	5	2	4	12	[illegible]
[illegible]	8	1	1	23	[illegible]
[illegible]	0	0	6	11	[illegible]
[illegible]	0	0	0	[illegible]	[illegible]
Total	[illegible]	84	54	105	441

[illegible] exposure could not be estimated and 3 persons with active pulmonary tuberculosis [illegible] have been excluded from this table.

TABLE 43.—*Occurrence of asbestosis in relation to dust concentration and length of employment*

PERCENTAGE WITH ASBESTOSIS

Dust exposure, million particles per cubic foot		Years in asbestos industry		
		0 to 4.9	5 to 9.9	Over 10
0 to 4.9	Affected	2	1	0
	Exposed	84	19	3
	Percentage	[illegible]%	[illegible]%	[illegible]%
5 to 9.9	Affected	0	6	13
	Exposed	70	37	19
	Percentage	0%	[illegible]%	[illegible]%
Over 10	Affected	8	22	21
	Exposed	134	43	34
	Percentage	[illegible]%	[illegible]%	[illegible]%

The percentage of persons with asbestosis increases greatly with increasing length of employment. Lanza, McConnell, and Fehnel (17) reported a similar trend; their percentage values are higher than these. There are two explanations for the small number of persons employed 15 years or more. This group of asbestos textile factories has been in operation from 6 to 16 years, and because of the relatively short life of the industry in this section of the country comparatively few persons have been exposed to asbestos dust for a long time. Another explanation is suggested by the fact that almost three-fourths of the workers employed more than 15 years have the early stages, at least, of a disabling disease, asbestosis. Since there is no satisfactory way of locating and examining persons who drop out of an industry because of disability incurred therein, the percentage values of the foregoing tables are necessarily minimal estimates of the prevalence of asbestosis.

The incidence of asbestosis increases with increasing dust concentration. In their report of a similar study carried on in Pennsylvania asbestos textile factories, Fulton, Dooley, Matthews, and Houtz (15) found that 8 percent of the workers exposed to an average dust concentration of 5 million particles per cubic foot had asbestosis; 22 percent of the 17-million-particle group; and 57 percent of the 44-million-particle group had asbestosis.

THRESHOLD CONCENTRATION OF ASBESTOS DUST

From a practical standpoint, one of the most important results of a medical and engineering study such as this is the definition of safe working conditions in the industry under study. Ideally, a threshold concentration of dust should be the highest dust concentration that would not produce pneumoconiosis in originally healthy workmen during their entire working life. The chief difficulty in this study, as in most of the earlier studies of the Public Health Service, is that very few workmen are exposed for a long period of time to low concentrations of asbestos dust. Because of the importance of the problem, it seems to be desirable to use such data as are at hand to define tentative safe working conditions that may serve as standards for the guidance of factory managers and engineers until more complete data are available.

In an analysis of this kind the cases that occur at relatively low dust exposures are of especial significance because they indicate what the safe limits of exposure may be. These cases must be examined individually, as well as statistically, in order to be sure that there are no unusual features that would complicate statistical analysis. Below 2.5 million particles per cubic foot, the interpretation of table 43 offers no difficulties. None of the 39 persons exposed to dust concentrations below 2.5 million particles per cubic foot had a case of asbestosis, although, as a matter of fact, only 6 persons had been employed more than 5 years. Three doubtful cases of asbestosis fell in the range 2.5 to 4.9 million particles.

Because clean-cut cases of asbestosis were found only in dust concentrations exceeding 5 million particles per cubic foot, and because they were not found at lower dust concentrations, 5 million particles per cubic foot may be regarded tentatively as the threshold value for asbestos-dust exposure until better data are available.

In order to find out the levels to which asbestos-dust concentrations have been reduced in practice, an engineering study (7) was made in an asbestos textile factory in which dust control equipment has been recently installed. This shows that means are already available for reducing the dust exposure of a majority of asbestos textile workers to less than 5 million particles per cubic foot.

The data of table 43 provide a simple means of comparing asbestosis with other pneumoconioses. In a previously reported study of the Public Health Service (51) it was found that workmen in anthracite mines could be divided into three groups which differed with respect to the nature of the dust to which they were exposed. The percent-

TABLE 44.—*Percentages of workers exposed to certain concentrations of asbestos dust in asbestos textile factories where dust control measures are used to a limited extent and in factories where effective dust control is practiced*

Dust concentration million particles per cubic foot	Limited use of dust-control measures [1]	Extensive use of dust-control measures [2]
Over 10	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]

[1] Data from table 43.
[2] Data from [illegible].

ages of men having any stage of anthraco-silicosis (1, 2, or 3) have been tabulated separately for each group in relation to average dust concentration and length of employment. In passing, it may be of interest to call attention to the difference between the pneumoconiosis-producing power of the three kinds of dust encountered in anthracite mines. If one compares groups of men with the same dust exposure and the same length of employment, in general the percentage of men with anthraco-silicosis increases with the free silica content.

TABLE 45.—*Percentage of men having anthraco-silicosis* [1]

Dust count in million particles per cubic foot	1,435 anthracite miners exposed to dust containing less than 5 percent free silica			602 men employed on haulageways who were exposed to dust containing about 12 percent free silica			131 rock workers exposed to dust containing about 45 percent free silica		
	Years in industry			Years in industry			Years in industry		
	Less than 15	15 to 24	Over 25	Less than 15	15 to 24	Over 25	Less than 15	[illegible]	Over [illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

[1] From table 40, p. 78, Public Health Bulletin No. 221.
[2] Less than 50 men examined.

As in asbestosis, the incidence of pneumoconiosis among anthracite miners increases with increasing length of employment. There is a much more definite increase in the incidence of anthraco-silicosis with increasing dust concentration than there is in asbestosis, probably

because men were exposed to a much greater range of dust concentrations in anthracite mines than in asbestos textile factories.

In comparison with the three kinds of dust encountered in anthracite mines, relatively low concentrations of asbestos dust and relatively short lengths of employment in the asbestos textile industry result in pneumoconiosis.

There was no free silica (quartz) in the dust encountered in the asbestos textile factories.

According to table 43, 16.3 percent of 69 former asbestos workers and 378 men and women employed at the time of the study in three asbestos textile factories had asbestosis. (Persons who were not exposed to appreciable quantities of asbestos dust have not been included.) Eight percent of the 378 workers employed at the time of study had asbestosis. This does not completely represent the incidence of asbestosis because about 150 employees were discharged from these factories 15 months before the present study was made. Many of the discharged asbestos workers had asbestosis, as the reports of Shull (31), Donnelly (12), and McPheeters (23) show. Although an attempt was made to examine as many as possible of these discharged cases, only 69 could be examined, 43 persons of whom had asbestosis according to the diagnostic standards employed in this study. One assumes that the number of persons employed in these factories was the same before these 150 persons were discharged as it was when the study was made, and if one assumes that 62 percent of all the discharged cases had asbestosis, then 50 cases of asbestosis escaped observation. Adding these to the 73 that were found, dividing the sum by 447, and multiplying by 100 gives an estimated percentage of 27.5. This is not an unusual percentage value. In numerous studies of the dusty trades the percentage of workers with pneumoconiosis has been found to be between 20 and 25 percent. Two such studies may be mentioned. Of 7,722 miners and men seeking employment in lead and zinc mines at Picher, Okla., who were examined in 1927–28 (49), 21.3 percent had silicosis. In a study of anthracite miners (51), "The prevalence of anthraco-silicosis among the entire group of employees was found to be about 23 percent."

Other investigators have reported the incidence of asbestosis in asbestos textile factories. Lanza, McConnell, and Fehnel (17) reported, "Of the total of 126 X-ray examinations, 4 were diagnosed as second-degree asbestosis, 33 as first degree, 39 as doubtful, and 20 as negative." The persons he studied "were selected more or less at random from among those having more than 3 years of employment in the industry." Since Lanza did not examine all the employees in the factories he studied, percentage values calculated from his data are not directly comparable with rate of incidence for the present study.

Fulton, Dooley, Matthews, and Houtz (15) noted that "Asbestosis was found in 14 persons (12 men, 2 women), or 25 percent of the exposed group."

INCIDENCE OF ASBESTOSIS IN DIFFERENT OCCUPATIONS

The percentage of persons affected by asbestosis varies in different occupations. The numbers of persons employed in many of the occupational groups are so small that caution must be exercised in making comparisons, but the data seem to be of sufficient interest to warrant publication. Classified by departments, the preparation department has the highest percentage of asbestotic persons. Carding is the principal occupation in this department. Although only 8 of the 72 carders had been employed more than 10 years, 29.2 percent had asbestosis. Eleven carders had advanced asbestosis. No other occupational group had so large a proportion of advanced cases.

TABLE 46.—*Number and percentage of workers having asbestosis, classified according to department and occupation*

Department and occupation	Number employed	Percent with asbestosis	Number with asbestosis		
			Stage 1	Stage 2	Stages 3 and 4
Preparation	102	25			
Crushers	5		0	0	0
Willowers and shakermen	5		1	0	1
Pickers and pluers	20		2	0	1
Carders	72		4	8	11
Spinning, twisting, and winding	216	14			
Mule and ring spinners	65		3	9	5
Twisters	84		1	2	2
Yarn spoolers and winders	63		2	2	0
Cleaners	4		1	0	0
Weaving	89	14			
Cloth weavers	43		3	4	4
Tape and braid loom weavers	41		1	1	0
Creelers	13		1	0	0
Finishing	18	11			
Inspectors, examiners, and folders	15		0	1	0
Burlers and menders	3		1	0	0
Superintendence and utility	29	3	0	1	0
Shipping and outside labor	17	0	0	0	0
Office and others	31	0	0	0	0
Total	502	10	23	25	24

In the spinning, twisting, and winding department, the mule and ring spinners have a higher proportion of asbestotic persons than the average, 24.6 percent. Twenty of the sixty-five mule and ring spinners had been employed more than 10 years.

A third occupation with an excessive prevalence rate for asbestosis is cloth weaving, in which 25.6 percent of the workers are affected. Only 4 of the 43 workers had been employed more than 10 years.

Lanza (18) has called attention to a point of interest, namely, that British investigators have found asbestosis more severe and menacing than it was in the United States. He adds that this difference is probably more apparent than real. Lynch and Smith (20) have reported 3 fatal cases whose duration of employment in asbestos textile mills was 11½, 17, and 21 years in occupations having dust exposures of the order of dry weaving. Stock (32) reported a fatal case in a man 27 years of age who worked 8 years as a carder in an asbestos plant. Egbert (13) has also given complete findings of similar case in a man 30 years of age who had worked 13 years as a carder and who died 4 years after onset of symptoms and 3½ years after discontinuing work.

These examples of American fatalities, some in young individuals, suggest that the period of dust exposure before disability or death ensues is as short as is the case in silicosis.

Bridges (63) in the factory inspector's reports of Great Britain has shown from year to year (table 47) that the duration of employment of fatal cases with asbestosis is only half that of cases with silicosis.

TABLE 47.—*Silicosis and asbestosis in Great Britain*

	Number of deaths	Average age at death	Duration of employment (years)		
			Maximum	Minimum	Average
Through 1933					
Silicosis	311	55.6	62	2.3	33.1
Silicosis with tuberculosis	205	52.4	62	2.0	31.7
Asbestosis	32	41.9	28	1.5	12.6
Asbestosis with tuberculosis	21	37.1	29	.9	9.2

More than 90 percent of the raw mineral used in the manufacture of asbestos textiles in this country and Great Britain is Canadian chrysotile, indicating that very little difference is to be expected from the standpoint of biological reaction in the two countries. As near as dust concentrations in the various operations of asbestos manufacture of British practice can be compared with results obtained in this country, no greater differences are found than exist in the inherent limitations of the dust-sampling devices. It would seem, therefore, that quite negligible differences exist in the manifestations of the disease here and abroad with the possible exception of associated or complicating tuberculosis.

PATHOLOGICAL FINDINGS IN ASBESTOSIS

Specimens for pathological study were furnished through the courtesy of Dr. J. Rush Shull, Charlotte, N. C., and Dr. H. F. Easom, director of the Division of Industrial Hygiene of the North Carolina State Board of Health.

The pathology of asbestosis is characterized by diffuse, interstitial, pulmonary fibrosis and by the presence of asbestosis bodies in the lungs.

GROSS APPEARANCE

In gross appearance the lungs vary in color from brownish to pale gray. Usually, they are not distorted like silicotic or anthracosilicotic lungs unless an extensive amount of long-standing fibrosis is present. Often the lungs are firm and heavy, but softer areas can sometimes be felt on palpation. Crepitus depends on the amount of fibrosis. The pleura may be thickened and fibrous tags may be scattered over the surface. In the cases described in this report, thickening of the pleura and the presence of fibrous adhesions are more frequently found on the right lung than on the left. Adhesions to the diaphragm are sometimes present.

On section, small, dark gray, irregularly shaped areas are seen on the cut lung surface. They are rather evenly distributed and they are of approximately the same size. Visible strands of connective tissue of varying size run through the lung parenchyma, forming a more or less regular, fibrous network, and enclosing the dark gray, nodular areas and lung tissue which show alveoli. Areas of well-marked emphysema are noted in the apices and outer portions of the lower lobes, a feature that was referred to in the discussion of the X-ray findings. The bronchi and bronchioles appear to be distended and their walls are often thickened. In one case, this bronchiectasis is most marked in the upper lobe, particularly in the apex.

Congested areas are occasionally seen on gross inspection of some of the lung sections. They are probably terminal.

The hilar lymph nodes are not markedly increased in size. They are pinkish gray and contain about the usual amount of anthracotic pigment found in a city dweller.

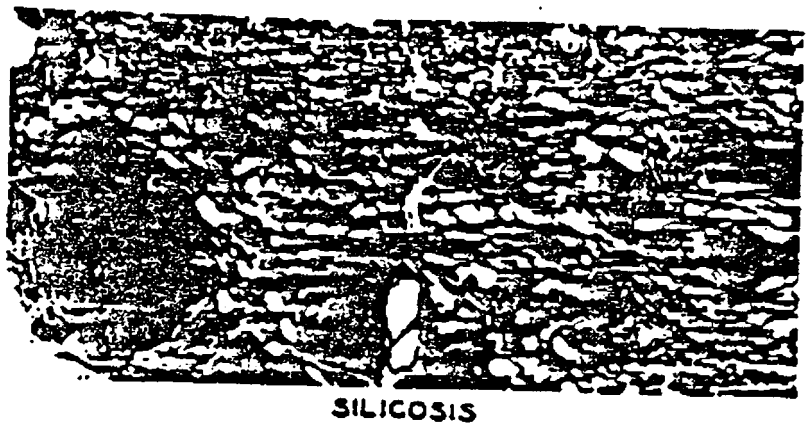
MICROSCOPIC APPEARANCE

The pleura, when thickened, is made up of adult fibrous connective tissue cells. The collagen fibers are usually well developed and may show hyalinization. In some sections the thickened pleura is vascular.

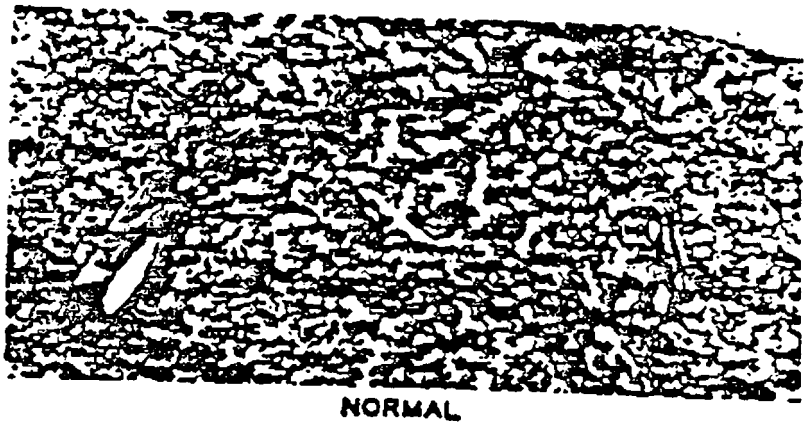
the blood vessels generally being small capillaries. Areas of variable size, containing anthracotic pigment, are occasionally found in the subpleural connective tissue.

Throughout the parenchyma of the lung a diffuse interstitial fibrosis is present, which varies with the asbestos-dust exposure of the individual. The areas of fibrous tissue are irregular or stellate in shape and may contain small blood vessels. The cellular elements consist of adult fibrous tissue cells, fibroblasts, some collagen, and often numerous macrophages. The strands of fibrous tissue represent an increase in the interlobular connective tissue. The fibrosis is usually rather uniformly distributed throughout the entire lung. In areas of considerable fibrosis, atelectasis is noted due to the contraction of the fibrous tissue. The alveoli occurring between areas of fibrosis are generally distended and show a moderate to marked emphysema. The alveolar walls are thickened by fibrous tissue, and in some sections hyalinization and the loss of nuclei occur. The alveoli are lined by a single layer of epithelial cells. The bronchial walls and the walls of the bronchioles may be thickened, and a slight to moderate lymphocytic infiltration is present occasionally. The smaller bronchi and bronchioles are frequently quite distended. The walls of the small blood vessels are thickened, particularly the adventitia, which contains an increased amount of fibrous tissue. Sections through emphysematous blebs show a fairly thick, fibrous, connective-tissue wall, lined by a single layer of flat or low cuboidal, epithelial cells. The blebs, measuring as much as 7 mm in diameter in the specimens studied, are apparently of bronchial origin.

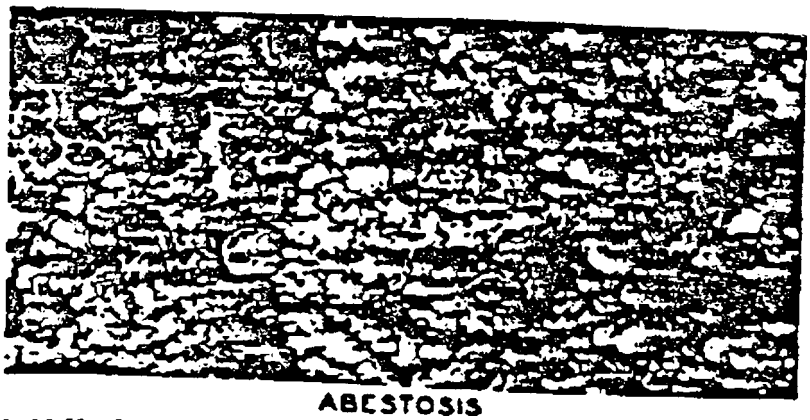
Asbestosis bodies are found in almost all parts of the pulmonary tissue. They occur most frequently in the areas of fibrosis and are accompanied by a greenish black pigment. They are also noted in the connective tissue about the small bronchi, in the alveolar walls, and in the alveoli. Macrophages and giant cells are usually present when the asbestosis bodies are found in the alveoli and often surround or engulf the bodies. These cells also contain a considerable amount of anthracotic pigment, and the dark greenish black pigment commonly found in asbestosis. A brownish yellow pigment in lesser amounts is also present. The asbestosis bodies occur in the lungs in a variety of shapes. The most frequent is a beaded rod with large rounded ends. Spindle shapes with beaded centers are also noted. Rosette formation is relatively frequent. There appears to be no relation between the number of asbestosis bodies and the amount of pulmonary fibrosis. In one case in which there is exceedingly little fibrosis, the asbestosis bodies are particularly numerous, while in another case, showing extensive fibrosis, they are quite infrequent. These bodies are golden yellow in color and are stained blue with potassium ferrocyanide, showing the presence of ferric iron. With this



SILICOSIS



NORMAL



ABESTOSIS

FIGURE 52 —PHOTOMICROGRAPHS OF LUNG SECTIONS SHOWING THE PRESENCE OF NODULAR FIBROSIS AND EMPHYSEMA IN A SILICOTIC LUNG AND DIFFUSE FIBROSIS AND EMPHYSEMA IN AN ASBESTOTIC LUNG. A SECTION OF A NORMAL LUNG IS SHOWN FOR COMPARISON. MAGNIFICATION 16X

stain some fine blue strands have been noted which are apparently asbestos fibers in the process of becoming asbestosis bodies. By the use of polarized light, fine fibers are often demonstrated in the tissue which are identified as asbestos. These may be seen in the peribronchial connective tissue, the interstitial connective tissue, the



FIGURE 53.—ASBESTOSIS BODY IN INTRA-ALVEOLAR CONNECTIVE TISSUE. PRACTICALLY NO FIBROSIS IN THIS AREA. CASE H-21. MAGNIFICATION 1,000X. (ARMY MEDICAL MUSEUM NO. 32295.)

alveolar walls, and in the alveolar spaces. The blue staining fibers and the asbestosis bodies are not visible when viewed with crossed Nicol prisms. Other fine crystalline material is sometimes noted.

The presence of complicating tuberculosis is often difficult to demonstrate. Unless caseation is well defined, the tubercles may



FIGURE 55.—CASE H-21. RIGHT LUNG. MARKEDLY THICKENED PLEURA WITH FUSION OF INTERLOBAR PLEURA. DENSE FIBROUS TAGS OVER ENTIRE SURFACE.



FIGURE 57.—CASE H-21. SECTION OF RIGHT LUNG. ADVANCED ASBESTOSIS. DISTENDED BRONCHI AND AIR SPACES AT BOTH APEX AND BASE. DIFFUSE FIBROSIS THROUGHOUT LUNG SUBSTANCE AND THICKENED PLEURA.



FIGURE 58.—CASE M 21. SECTION OF LEFT LUNG. DISTENDED BRONCHI AND AIR SPACES AT APEX, MATTED UNIFORMLY DISTRIBUTED FIBROUS AND EMPHYSEMATOUS AREAS THROUGHOUT REST OF LUNG

Case H-23

(Figs. 59 to 63)

Male. Negro, aged 33 years.

Cause of death.—Pneumonia.

Occupational history.—Worked as cleaner and sweeper in the cardroom of an asbestos plant for 13 years prior to death. Previously, roofer (asphalt), ½ year.

Estimated dust exposure.—*a*. 35.3 million particles per cubic foot; *b* million particle years, 458.9.

Fluoroscopy.—Moderate (2+) lag of diaphragm right and left; first degree ground-glass appearance.

X-ray.—Note obliteration of costophrenic angle on right. Moderate granular appearance right base. Interpreted as commencing generalized fibrosis (fig. 59). Note the definite evidence of fibrosis at the right base and obliteration of the right costophrenic angle. This evidence of increased fibrosis and fibrous pleurisy is also shown in the necropsy material (figs. 62 and 63).

Medical history.—Influenza, 1918 (duration, 2 weeks). Frequent colds. Medical history negative until 2 weeks before death when he developed pneumonia.

Physical examination.—Slender, colored male. Height, 70½ inches. Weight, 151 pounds. Long chest; right side slightly smaller. Expansion poor (1¼ inches). Moderate dullness over bases on percussion. Persistent crackling rales over bases of lungs. Palpation and fremitus, negative. Faint breath sounds over bases. Blood pressure, 140/90. Pulse rate, 76, 80, and 68. Respiratory rate, 28, 28, and 24 (before, immediately after, and 2 minutes after exercise). Slight transient dyspnoea immediately after exercise). Asbestosis bodies in sputum. Urinary silica, 3.3 mg per 100 cc (33 mg per 24 hours).

Diagnosis.—Moderately advanced asbestosis (stage 3) 15 months before death.

Pathological examination showed moderately advanced asbestosis. A moderate degree of diffuse pulmonary fibrosis and emphysema was noted accompanied by many asbestosis bodies. Acute diffuse pneumonitis was present.

Chemical examination of lung tissue.—Sample from right lung—ash 2.69 percent, silica 0.44 percent, silica in ash 16.3 percent.



FIGURE 39.—CHEST X-RAY OF CASE M-23 15 MONTHS BEFORE DEATH



FIGURE 60.—CASE H-23. RIGHT LUNG. ENTIRE PLEURAL SURFACE COVERED BY DENSE FIBROUS TAGS



FIGURE 62.—CASE M 23. SECTION OF RIGHT LUNG. MODERATELY ADVANCED ASBESTOSIS. FIBROUS AREAS AND EMPHYSEMA UNIFORMLY DISTRIBUTED THROUGHOUT THE LUNG SUBSTANCE.



FIGURE 63.—CASE H-23. SECTION OF LEFT LUNG SHOWS LESS INVOLVEMENT THAN RIGHT LUNG (FIG. 62).

Case H-25

(Figs. 64 and 65)

Male, white, aged 27 years.

Cause of death.—Lobar pneumonia.

Occupational history.—Began work at age of 16 years. Broadcloth weaver (asbestos), 1 year. Truck driver, 3 months. Broadcloth weaver (asbestos), 2 years. Truck driver, 3 years. Broadcloth weaver (asbestos), 4 years.

Weighted average dust exposure.—49.7 million particles per cubic foot, or 347.9 million particle years.

Medical history.—Past history negative. Had influenza, prior to death, which developed into pneumonia.

X-ray examinations.—Fi[illegible] made 15 months and 1 month (fig. 64) before death showed first-degree ground glass which was interpreted as commencing generalized fibrosis.

Medical examination.—The principal abnormalities found on physical examination 15 months before the death of the patient were second-degree curved nails, second-degree cyanosis, and decreased fremitus on both the right and left side. The heart size and heart sounds were normal; blood pressure, 120 74. On exercise the pulse rate rose from 88 to 116 and 2 minutes later had fallen to 84. The respiratory rate rose on exercise from 18 to 30 and returned to 22. The patient had a chest girth at expiration of 37½ inches and a chest expansion of 2 inches. He was a thick-chested man 68½ inches tall, weighing 195 pounds. Fifteen months before death this case was diagnosed as early asbestosis (stage 2).

Pathological examination showed early asbestosis. A diffuse interstitial fibrosis with little emphysema and bronchiolectasis and numerous asbestosis bodies was noted. Lobar pneumonia was shown to be the cause of death.

Chemical examination of lung tissue.—Sample from right lung—ash 2.44 percent, silica 0.21 percent, silica in ash 8.42 percent. Sample from left lung—ash 2.44 percent, silica 0.27 percent, silica in ash 11.17 percent.

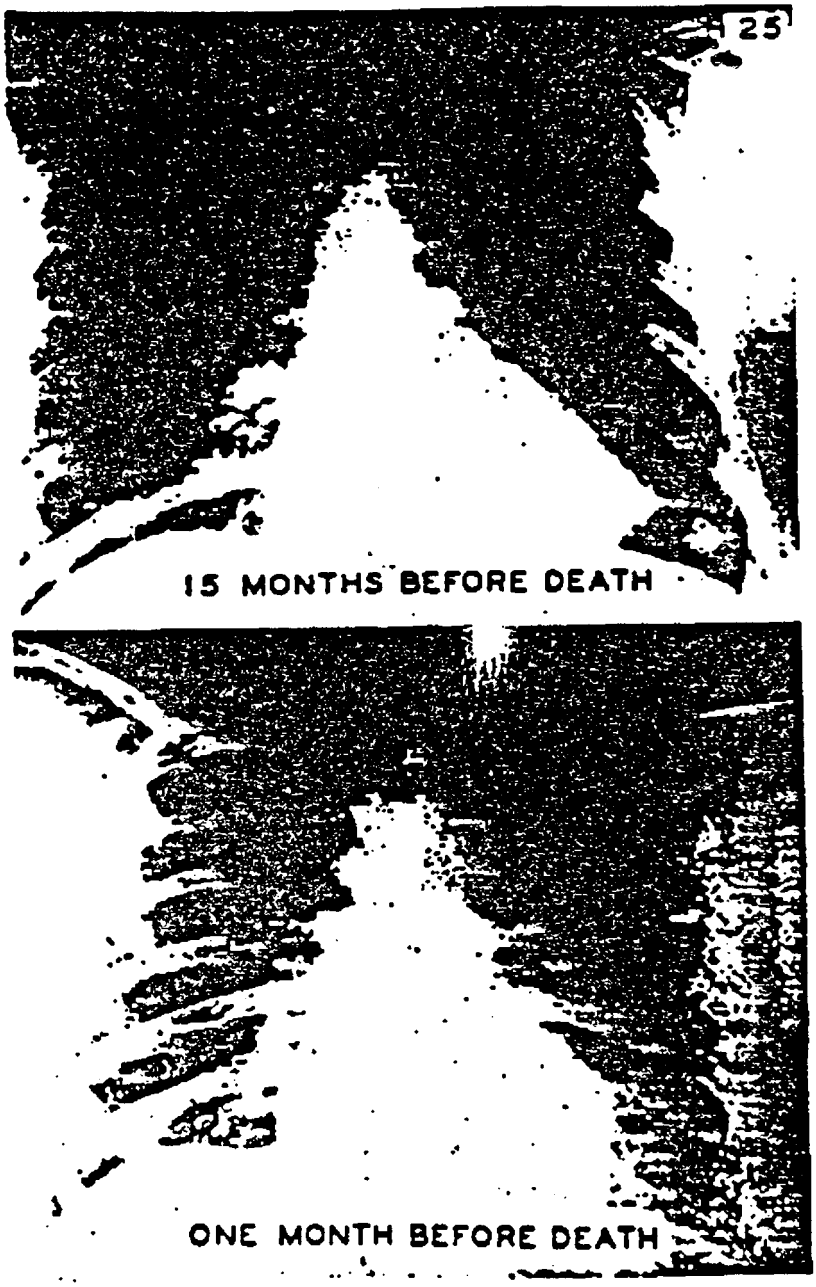


FIGURE 64.—CASE H-25. CHEST X-RAYS TAKEN 15 MONTHS AND 1 MONTH BEFORE DEATH. PART OF THE DIFFERENCE BETWEEN THE TWO ROENTGENOGRAMS IS DUE TO A DIFFERENCE IN X-RAY TECHNIQUE



FIGURE 65.—CASE H-25. SECTION OF RIGHT LUNG. EARLY ASBESTOSIS. UNIFORM DISTRIBUTION OF FIBROUS AREAS THROUGHOUT LUNG SUBSTANCE.

ACKNOWLEDGMENTS

Grateful acknowledgments are due the employers and employees of the asbestos textile plants in which this study was made for their interest and cooperation.

Howard F. Brubach, laboratory assistant, prepared X-ray chest films, conducted clinical and chemical tests on urine and sputum samples, collected the atmospheric dust samples used for chemical analysis, made the photomicrographs reproduced in this bulletin, and materially contributed to the progress of this study in these and in other ways. Junior Statistical Clerk Marjorie W. Hertford coded and tabulated the medical findings and prepared the graphs and tables included in this report. Associate Chemist F. H. Goldman made chemical analyses on samples of asbestos dust and samples of lung tissue.

SUMMARY OF MEDICAL FINDINGS

A medical examination was made of each employee in three asbestos textile plants employing 423 men and 118 women. These persons were much younger than the average industrial employee and they had been employed in this industry for relatively short lengths of time. Almost half of these workers had been previously employed in cotton or woolen textile mills. Only 23 persons had been employed in a trade in which workers are known to be exposed to inorganic dust, and only 1 of these 23 persons had pneumoconiosis. For this reason, it is believed, asbestos-dust exposure can be held responsible for the cases of pneumoconiosis that were found in these three factories.

Asbestosis is a form of pneumoconiosis caused by long-continued inhalation of asbestos dust. The primary effect of asbestos dust on the body is to set up an interstitial, pulmonary fibrosis. This may be seen at autopsy, and reports on three cases appear in the pathological section of this bulletin. This type of fibrosis may also be detected by X-ray examination of the chest. On the X-ray film the shadows cast by this type of fibrosis resemble ground glass in appearance and usually extend over the lower portions of the lung fields, frequently being heavier on the right side. Unlike silicosis, nodular fibrosis has not been detected in asbestos workers.

As in silicosis, the chief symptoms of asbestosis are progressive dyspnoea, variable cough, substernal chest pain, decreased chest expansion, emaciation, weakness, clubbed finger tips, and curved finger nails. Most of these symptoms seem to be the result of the pulmonary fibrosis set up by inhaled asbestos dust, and the concomitant pathologic changes, such as emphysema, bronchiectasis, and bronchiolectasis, which can be demonstrated at autopsy.

The percentage of persons affected by asbestosis or by any one of these symptoms depends upon the dust exposure. In all cases the percentage of persons affected by any given sign or symptom of asbestosis is greater than in control groups of industrial workers who were not exposed to inorganic dust.

A characteristic of asbestosis is the finding of asbestosis bodies in the lungs and in the sputum. Although it does not appear from these data that a finding of asbestosis bodies can be used to establish a diagnosis of asbestosis, nevertheless it is evidence of asbestos exposure.

Only three cases of asbestosis, all of them diagnosed as doubtful or borderline cases, were found to be exposed to dust concentrations of less than 5 million particles per cubic foot. (These three individuals had dust exposures of about 4 million particles per cubic foot.) Above 5 million particles per cubic foot, numerous cases of well-marked asbestosis were found. It would seem that if the dust concentration in asbestos factories could be kept below 5 million particles (the engineering section of this report has shown how this may be accomplished), new cases of asbestosis probably would not appear.

ANNOTATED BIBLIOGRAPHY

To facilitate reference to the papers listed in this bibliography, a brief note has been appended to each citation which indicates the kind of information that may be sought for profitably. So far as possible, no information that can be gained from the title has been repeated in the annotation and consequently the length of the note is no measure of the merit of the article.

In the interests of completeness, a number of papers have been listed here which were not referred to in the text. The papers on lung cancer as a possible complication of asbestosis are an example. No cases of lung cancer were found in this study, but it appears that it would be a profitable thing to look for whenever autopsy material is available for study. For that reason papers dealing with the subject have been included.

ENGINEERING REPORTS

1. Annual report of the chief inspector of factories and workshops for England and Wales. H. M. Stationery Office, London, England. 1910.

   Contains a section on observations made by Dr. Collis on the effects of exposure to asbestos dust on 40 workers in an asbestos textile factory.

2. Bloomfield, J. J., and J. M. DallaValle. The determination and control of industrial dust. Public Health Bulletin No. 217. 1935.

   Discusses generally-accepted methods of dust quantification and methods in use for controlling industrial dust.

3. Hatch, Theodore, and S. P. Choate. Statistical description of the size properties of nonuniform particulate substances. Jour. Franklin Inst. 207: 369–387. 1929.

   Gives statistical parameters for the calculation of total surface area, volume, and mass of a given distribution of nonuniform dust particles.

4. Fulton, W. B., R. L. Houtz, A. Dooley, and J. L. Matthews. Asbestosis. Part I. The collection and counting of dust encountered in asbestos fabricating plants. Pennsylvania Dept. Labor and Industry Bull. 37. 1934.

   Discusses the measurement of asbestos dust and its physical properties. Engineering study of four plants.

5. Hatch, Theodore, and C. B. Moke. The mineralogical composition of airborne foundry dust. Jour. Ind. Hyg. 18: 91–97. 1936.

   A petrographic study of air-borne foundry dust according to particle size. Composition of dust reported to vary with size.

6. Hurlbut, C. S., and C. R. Williams. The mineralogy of asbestos dust. Jour. Ind. Hyg. 17: 280–284. 1935.

   Discusses the composition of settled dust samples obtained in factories manufacturing asbestos textiles and other asbestos products.

7. Page, R. T., and J. J. Bloomfield. A study of dust control methods in an asbestos fabricating plant. Public Health Reports 52: 1713–1727. 1937.

"This investigation included a study of atmospheric dust concentrations in the factory workrooms, and a study of the exhaust systems used to remove asbestos dust."

ASBESTOSIS

8. Alwens, W. Ueber Asbestose der Lungen. Munchener med. Wochenschr. 82: 1797–1800. 1935.

X-ray and medical study of 35 employees of a German asbestos textile factory. 22 cases of asbestosis were found. X-ray examination showed flocculent markings but no nodulation.

8a. di Biasi, W. Zur pathologischen Anatomie der Lungenasbestose. Archiv fur Gewerbepath. und Gewerbehyg. 8: 139–155. 1937.

A 50-year-old man employed 21 years in an asbestos factory died of circulatory insufficiency after seeking treatment for shortness of breath. On autopsy the right side of the heart was enlarged. There were pleural adhesions. The fibrosis seen on gross inspection and confirmed on histological examination was most extensive in the lung bases. Sanders and Bygden [41a] have reported on the asbestosis bodies found in this case.

8b. Bureseh, Annemarie. Pneumonokoniose, Berufskrankheit und Entschadigungspflicht. Deutsche Med. Wochenschr. 57: 1100–1102; 1247–1249. 1931.

A 47-year-old man, employed 26 years in an asbestos factory, sought treatment for disabling dyspnoea, cardiac insufficiency, and cough. Clinical and X-ray findings characteristic of asbestosis are described in detail. Died as a result of right ventricular insufficiency. Post-mortem examination revealed pulmonary fibrosis, marked hypertrophy and dilatation of the right ventricle and little hypertrophy of the left ventricle.

9. Cooke, W. E. Pulmonary asbestosis. Brit. Med. Jour. 1927 (Vol. 2): 1024–1025. 1927.

A 33-year-old woman employed in an asbestos factory over an 18-year period "complained of cough, dyspnoea, expectoration, and lassitude. An X-ray plate showed extensive fibrosis, more marked in the right lung, two calcareous glands at the root of the left lung, and two small calcareous particles in the base of the left lower lobe." On autopsy, dense pleural adhesions were found. "On section the [right] lung is seen to be fibrosed and to a large extent airless, the lung tissue being replaced by fibrous tissue." Calcareous masses and areas of caseation and cavitation were found.

10. Dewitz, A. P. Asbestwarzen. Arch. f. Dermat. u. Syph. 161: 1–5. 1930.

Description of the localization and the gross and histological appearances of asbestos corns.

11. Donnelly, J. Pulmonary asbestosis. Amer. Jour. Pub. Health 23: 1277–1281. 1933.

Partial review of previous observations on the disease. Includes case reports on three totally disabled patients taken from a series of 15 cases (4 figures—chest plates). All but 1 of the 15 had at one time been diagnosed pulmonary tuberculosis.

12. Donnelly, J. Pulmonary asbestosis: incidence and prognosis. Jour. Ind. Hyg. 18: 222–228. 1936.

Description of X-ray films of 151 asbestos workers.

13. Egbert, D. S. Pulmonary asbestosis. Report of a case with necropsy findings. Amer. Rev. Tubere. 31: 25–34. 1935.

Autopsy report on case described by W. B. Soper (Amer. Rev. Tubere. 22: 571–564. 1930). White, 30-year-old male employed as an asbestos carder 13 years complained of dyspnoea and blood-streaked sputum. No cough. Examined and X-rayed on two occasions before death which resulted from cardiac failure. On autopsy "diffuse fibrosis of lungs involving particularly upper lobe of right and portion of upper lobe of left; arrested tuberculosis; asbestosis bodies throughout lungs; few in tracheobronchial lymph nodes."

14. Ellman, P. Pulmonary asbestosis: Its clinical, radiological, and pathological features, and associated risk of tuberculous infection. Jour. Ind. Hyg. 15: 165–183. 1933.

A 43-year-old man employed in the cardroom of an asbestos factory 9 years became disabled on account of dyspnoea, accompanied by dry, irritating cough and progressive anorexia, loss of weight, and clubbing of fingers. X-ray and clinical examination. At autopsy this case was diagnosed as "advanced pulmonary asbestosis, complicated by tuberculous pleurisy."

A 31-year-old woman employed 2 years making asbestos mattresses became dyspnoeic, wasted, and cyanotic. Died of tuberculosis. Autopsy findings are presented.

15. Fulton, W. B., A. Dooley, J. L. Matthews, and R. L. Houtz. Asbestosis. Part II. The nature and amount of dust encountered in asbestos fabricating plants. Part III. The effects of exposure to dust encountered in asbestos fabricating plants on the health of a group of workers. Pennsylvania Dept. Labor and Industry Bull. 42. 1935.

Report of a medical examination of 66 workers. Bibliography of 125 papers.

16. Gloyne, S. R. The morbid anatomy and histology of asbestosis. Tubercle 14: 445–451; 493–497; 550–558. 1933.

Detailed description of pathological findings and review of literature.

17. Lanza, A. J., W. J. McConnell, and J. W. Fehnel. Effects of the inhalation of asbestos dust on the lungs of asbestos workers. Public Health Reports 50: 1–12. 1935. Reprint No. 1665.

Brief description of the findings of an engineering and medical study made in an asbestos textile plant. "X-ray films were made of 126 persons."

18. Lanza, A. J. Asbestosis. Jour. Amer. Med. Assoc. 106: 368–369. 1936.

Brief description of some of the early observations on the disease particularly as seen in United States. Also notes on clinical picture and roentgenology.

19. Laviette, Demetrius. Pulmonary asbestosis. Records of the International Conference on Silicosis held at Johannesburg, Aug. 13–27, 1930. Studies and Reports, Series F (Ind. Hyg.), No. 13, pp. 380–389.

Author's observations on workers handling pure asbestos and asbestos mixed with cotton or other vegetable dusts. Concludes that at least 5 years of exposure required to produce pneumonoconiosis; direct ratio between quantity of dust inhaled and pneumonoconiosis. Well-established case may develop in 7 to 9 years and the disease can cause death after 13 years' heavy exposure.

20. Lynch, K. M., and W. A. Smith. Pulmonary asbestosis II. Including the report of a pure case. Amer. Rev. Tubere. 23: 643–660. 1931.

White male, 70 years old, employed as an asbestos carder 11 years, sought medical treatment for chest pain, loss of weight (23 lb.) and shortness of breath. X-ray examination on 3 occasions during a 3-year period showed nothing in both lungs. Died as a result of slow, cardiac failure. On autopsy extensive fibrosis, emphysema, and bronchiectasis were found. Tuberculosis was absent.

22. McDonald, Stuart. Histology of pulmonary asbestosis. Brit. Med. Jour. 1927 (vol. 2): 1025–1026. 1927.

"The interstitial fibrosis is such as might be expected as a result of a combination of a pneumoconiotic condition and a chronic tuberculous infection." The possibility that asbestosis bodies may have been of plant or animal origin may be excluded. "The hypothesis advanced is that these bodies are portions of asbestos fibres in the process of alteration and absorption by hydrolysis, either by direct chemical action or by enzymes."

23. McPheeters, S. B. A survey of a group of employees exposed to asbestos dust. Jour. Ind. Hyg. 18: 229–239. 1936. Reports "the results of a survey of 210 persons exposed to asbestos dust."

24. Martz, L. Asbestosis and Tuberkulose der Lungen. Zeitschr. f. Tuberk. 72: 11–15. 1935.

Asbestosis complicated by tuberculosis was found in a 30-year-old woman employed in an asbestos factory during a 12-year period.

25. Merewether, E. R. A. A memorandum on asbestosis. Tubercle 15: 68–81; 109–115; 152–159. 1933.

A comprehensive review of British studies of asbestosis, including economic, mineralogical, and medical historical facts, main features of the disease, asbestosis bodies, correlations with dust exposure, risk of tuberculosis, preventive measures, and compensation.

26. Mills, R. G. Pulmonary asbestosis: report of a case. Minnesota Med. 13: 495–499. 1930.

A 58-year-old man exposed to asbestos dust in a mine 17 years previously was admitted to a hospital where he died of heart block. Marked lung fibrosis was found on autopsy. Asbestos fibers and asbestosis bodies were found on microscopic examination.

27. Musso, Giovanni. Clinical and radiological notes on pneumoconiosis due to asbestos. Silicosis—records of International Conference held at Johannesburg, Aug. 13–27, 1930. Studies and Reports, Series F (Ind. Hyg.), No. 13, p. 209.

The X-ray shows "arborization distributed bilaterally in the hilar and perihilar zones which in most advanced cases considerably reduces transparency. Sometimes there is infiltration of the lungs."

28. Oliver, Thomas. Clinical aspects of pulmonary asbestosis. Brit. Med. Jour. 1927 (Vol. 2): 1026–1027. 1927.

Case histories of "two women who are the subjects of pulmonary asbestosis, one aged 48 and the other 39."

29. Panecast, H. K., T. G. Miller, and H. R. M. Landis. A roentgenologic study of the effects of dust inhalation upon the lungs. Trans. Assoc. Amer. Physicians 32: 97–108. 1917.

The X-ray aspects of 137 individuals exposed to organic and inorganic dust are presented. Of the group exposed to inorganic dust 15 were asbestos workers. Shadows almost invariably first appeared in the right lung on a level with the hilus shadow. The appearance of the shadows is reported "softer and less sharply defined" in asbestos workers. A good description is given of the three stages which the authors recognize in the progress of pneumoconiosis.

30. Schuster, Norah H. Pulmonary asbestosis in a dog. Jour. Path. and Bact. 34: 751–757. 1931.

A dog kept in an asbestos factory 10 years "had suffered for two years from cough and dyspnoea, and for six months from progressive wasting. The dyspnoea became so distressing that he was destroyed." On examination of the lungs diffuse fibrosis, bronchiectasis, phagocytosis, and asbestos fibers were found. Asbestosis bodies were not found.

30a. Seiler, H. E., and M. D. Gilmour. A case of pulmonary asbestosis. Brit. Med. Jour. 1931 (1): 1112–1114. 1931.

A 42-year-old man employed 23 years in an asbestos factory as a carder, dry-cloth weaver, and in the manufacture of millboard, sought treatment for "cough", breathlessness, loss of weight, and lassitude. Radiographic examination revealed a diffuse fine mottling of a silicotic nature spread throughout both lungs. The patient who is the subject of these notes has died and the fact that pathological examination showed that death was due solely to cardiac failure secondary to back pressure resulting from the massive generalized fibrosis and irrespective of any added infection is an additional point of interest in this case."

31. Shull, J. R. Asbestosis: a roentgenologic review of 71 cases. Radiology 27: 279–292. 1936.

Results of medical examination of asbestos textile workers. Two autopsy reports.

32. Stock, G. A. Pulmonary asbestosis. Med. Bull. Veterans' Adm. 10: 126–129. 1933.

A 27-year-old man, employed 8 years as an asbestos carder, was admitted to a hospital because of shortness of breath and chest pain. He was underweight and had poor chest expansion. No pulmonary tuberculosis present. "The post-mortem diagnoses were: Pneumonia, acute lobar; pneumoconiosis, all lobes of both lungs; pleurisy, chronic, fibrinous, right, and chronic fibrinous with adhesions, left; interlobar, left; hypertrophy and dilatation of the heart; appendicitis, chronic." Histological examination revealed asbestosis bodies and fibrosis.

33. Stracke, H. Bericht über den Fall von Lungen-asbestosis, welcher der Arbeit des Herrn Prof. Heger zugrunde liegt. Virchow's Archiv. f. pathologische Anatomie u. Physiologie 288: 354–357. 1933.

35-year-old man employed in a German asbestos factory 20 years complained of dyspnoea, bronchitis, and loss of weight. X-ray examination showed fine, discrete markings in middle and lower portions of lung fields. Died of heart failure. On autopsy, found fibrosis, emphysema, and asbestosis bodies. No signs of tuberculosis.

33a. White, T. P. Pulmonary asbestosis. Trans. Med. Soc. State of North Carolina. 1935: 259–262. 1935.

A 45-year-old man employed 9 years in an asbestos mill sought medical treatment on account of shortness of breath, unproductive cough, and slight loss of weight. All symptoms increased in severity as time went on.

On X-ray examination 9 years later "The lung fields are negative for tuberculosis. The examination shows a marked, wide-spread fibrosis throughout both chests which reaches the periphery. The heart outline is entirely obscured by dense fibrosis and the cardio-phrenic angles are obliterated." The exact heart measurements cannot be determined because of the shagginess of the heart outline but the heart is definitely enlarged."

34. Wood, W. B., and D. S. Page. A case of pulmonary asbestosis: Death from tuberculosis two years after first exposure to the dust. Tubercle 11: 157–158. 1930.

A 21-year-old woman employed 15 months as a spinner in an asbestos factory sought treatment for "anorexia, weakness, wasting, and pain in the right side of the chest of three months' duration. She denied having cough or expectoration. There was no radiographic evidence of pneumoconiosis." On histological examination of the lungs, "in the few sections examined in which tuberculous lesions were not present, the asbestos together with a large quantity of black pigment was generally surrounded by a little fibrous tissue, but there was no gross fibrosis." Asbestos fibers and asbestosis bodies were found.

35. Wood, W. B., and S. R. Gloyne. Pulmonary asbestosis; a review of one hundred cases. Lancet 227 (2): 1383–1385. 1934.

Of 100 asbestos workers who sought medical treatment, 33 had uncomplicated asbestosis, 1 had asbestosis with active tuberculosis, 9 had asbestosis with quiescent tuberculosis, and 17 were diagnosed as doubtful cases of asbestosis. "Septic bronchitis, broncho-pneumonia, and pulmonary tuberculosis are the commonest terminal complications of the disease."

ASBESTOSIS BODIES

36. Gloyne, S. R. The presence of asbestosis bodies in the faeces in a case of pulmonary fibrosis. Tubercle 12: 158–159. 1931.

"The faeces were emulsified with four or five times their volume of 5 per cent formalin in saline. The emulsion of the faeces, when centrifuged, was found to contain a scanty number of asbestosis bodies."

37. Gloyne, S. R. A method of staining the asbestosis bodies found in the sputum of asbestos workers. Jour. Ind. Hyg. 13: 85–86. 1931.

Describes techniques employing Prussian blue and ammonium sulfide.

38. Lynch, K. M., and W. A. Smith. Asbestosis bodies in sputum and lung. Jour. Amer. Med. Assoc. 95: 659–661. 1930.

Asbestosis bodies (illustrated) were recovered from the lungs of two asbestos workers and from the sputum of four asbestos workers.

39. Page, R. C. A study of the sputum in pulmonary asbestosis. Amer. Jour. Med. Sci. 189: 44–55. 1935.

Clumps of asbestosis bodies were found in the sputum of 10 asbestos workers with advanced asbestosis. Asbestosis bodies were found in all but 1 of the 31 cases studied.

40. Stewart, M. J., and A. C. Haddow. Demonstration of the peculiar bodies of pulmonary asbestosis ("asbestosis bodies") in material obtained by lung puncture and in the sputum. Jour. Path. and Bact. 32: 172. 1929.

Asbestosis bodies were found in material obtained by puncturing the left lung with a hollow needle. Patient was a 38-year-old woman, employed in an asbestos factory 15 years, who had cough, dyspnoea, and loss of weight

41. Stewart, M. J., N. Tattersall, and A. C. Haddow. On the occurrence of clumps of asbestosis bodies in the sputum of asbestos workers. Jour. Path. Bact. 35: 727–741. 1932.

A clump of asbestosis bodies was found in the sputum of a 31-year-old woman employed 11 years in an asbestos factory. She complained of cough, dyspnoea, and anorexia. "The radiograph showed diffuse fine striation throughout the lower third of both lungs." Died of bronchopneumonia. On autopsy, "There was notable diffuse fibrosis of the greater (basal) portion of both lower lobes. The non-fibrosed tissue was somewhat emphysematous." Asbestosis bodies were most numerous in the densely fibrosed areas. No evidence of tuberculosis.

41a. Sundius, N., and A. Bygrén. Der Staubinhalt einer Asbestosislunge und die Beschaffenheit der sogenannten Asbestosiskörperchen. Archiv fur Gewerbepath. und Gewerbehyg. 8: 26–70. 1937.

Quantitative microchemical analysis of asbestosis bodies isolated from a human lung showed that the gel surrounding the asbestos fiber consisted largely of ferric oxide, phosphate, an organic substance whose chemical composition suggests that it is a protein, and water. Chemical analysis and X-ray examination of cores of asbestosis bodies indicated that the chemical composition of the asbestos fiber remained unchanged.

OTHER PNEUMOCONIOSES

42. Curtis, A. J. R. The health of cement w[illegible]ers. Indust. Med. 6: 195–200. 1937.

Results of a medical survey of 201 men exposed to dust in a cement mill. Morbidity data for 21 plants.

43. Dreessen, W. C. Effects of certain silicate dusts on the lungs. Jour. Ind. Hyg. 15: 66–78. 1933.

"The silicate dusts of tremolite talc and slate induce a fine, diffuse, bilateral fibrosis of the lungs which is definitely demonstrable in the X-ray."

43a. Dreessen, W. C. Effect of inhaled marble dust as observed in Vermont marble finishers. Public Health Reports 49: 724–732. 1934.

"Although marble dust when inhaled in the concentrations here observed [less than 26 million particles per cubic foot] produces a mild bilateral, linear fibrosis in [15 percent of the 80 men X-rayed] no serious lung changes were noted and there was no disability due to the dust, even after many years of exposure."

44. Dreessen, W. C., and J. M. DallaValle. The effects of exposure to dust in two Georgia talc mills and mines. Public Health Reports 50: 131–143. 1935. Reprint 1669.

Engineering and medical study of the relation between dust exposure and occurrence of pneumonoconiosis in talc workers.

45. Dreessen, W. C., and H. H. Jones. Anthracosilicosis. Jour. Amer. Med. Assoc. 107: 1179–1185. 1936.

Results of a medical examination of 2,711 employed and 135 disabled anthracite miners.

46. Hoffman, F. L. Mortality from respiratory diseases in dusty trades (inorganic dusts). Bull. U. S. Bur. Labs. Stat. No. 231, pp. 176–182. 1918.

Gives brief summaries of the effects of exposure to inorganic dusts, compiled from occupational mortality data available in 1918.

47. Middleton, E. L. Industrial pulmonary disease due to the inhalation of dust. Lancet 231: 1–9; 59–64. 1936.

Part I deals with silicosis, Part II with silicatosis (including asbestosis).

48. Russell, A. E., R. H. Britten, L. R. Thompson, and J. J. Bloomfield. The health of workers in dusty trades. II. Exposure to siliceous dust (granite industry). Public Health Bulletin No. 187. 1929.

An engineering, medical, and statistical study of the effects of exposure to granite dust on workers.

49. Sayers, R. R., F. V. Meriwether, A. J. Lanza, and W. W. Adams. Silicosis and tuberculosis among miners of the tri-state district of Oklahoma, Kansas and Missouri. I. For the year ended June 30, 1928. Bureau of Mines, Technical Paper 545. 1933.

Results of medical and roentgenographic examinations of 7,722 men employed in lead and zinc mines.

50. Sayers, R. R. Silicosis. Public Health Reports 49: 595–602. 1934. Reprint 1626.

Discussion of the historical, medical, and engineering aspects of silicosis.

51. Sayers, R. R., J. J. Bloomfield, J. M. DallaValle, R. R. Jones, W. C. Dreessen, D. K. Brundage, and R. H. Britten. Anthraco-silicosis among hard coal miners. Public Health Bulletin No. 221. 1933.

Medical and engineering study of incidence of pneumoconiosis in relation to dust exposure among 2,700 anthracite miners.

52. Thompson, L. R., D. K. Brundage, A. E. Russell, and J. J. Bloomfield. The health of workers in dusty trades. I. Health of workers in a Portland cement plant. Public Health Bulletin No. 176. 1928.

Includes an engineering study of dust conditions in a cement plant, records of respiratory ailments, and physical examinations of a large group of workers.

CANCER AND ASBESTOSIS

52a. Eabert, D. S., and A. J. Geiger. Pulmonary asbestosis and carcinoma. Amer. Rev. Tubere. 34: 143–150. 1936.

A 41-year-old man employed 18 years as an asbestos weaver sought medical treatment for dyspnoea, cough, and "an insidious, gradually increasing pain of aching character throughout the back." Patient lost 40 pounds within a few months. Before death, X-ray and laboratory tests were made. Besides the characteristic pathological and histological signs of asbestosis, there was a large primary carcinoma illustrated in the lower lobe of the left lung.

53. Gloyne, S. R. Squamous carcinoma of lung occurring in asbestosis: two cases. Tubercle 17: 5–10. 1935.

Description of malignant lesions noted in two cases. The lesions were small and not recognized in life. They showed prickle cells, keratinization, and cell nests. The growths advanced along the bronchial mucosa by continuous finger-like prolongations. The growths were in a portion of the lung where the asbestosis was quite advanced.

54. Gloyne, S. R. Oat cell carcinoma of lung occurring in asbestosis: case. Tubercle 18: 100–101. 1936.

Typical, extensive asbestosis was found on autopsy of a 56-year-old man employed 22½ years in the storage department of an asbestos factory. A neoplastic plant the size of a golf ball was found in the peripheral portion of the lower lobe of the left lung, continuous with the pleura. The growth

was soft, pinkish white, and fairly well demarcated. It grew outward from a center in a compact mass, destroying lung tissue as it progressed.

55. Lynch, K. M., and W. A. Smith. Carcinoma of lung in asbesto-silicosis. Amer. Jour. Cancer 24: 56–64. 1935.

Case report of white male, age 57, who had been cotton weaver 22 years and weaver in asbestos mill about 21 years. As result of exposure, he had extensive fibrosis of lungs and pleura with emphysema, bronchiectasis, and cardiac embarrassment. In addition to the diffuse fibrosis, the authors noted formation of hyalin fibrous nodules characteristic of silicosis. The pulmonary cancer appeared to originate from one of the branches of the bronchus to the right lower lobe, where squamous metaplasia of the living epithelium was observed. The duration of the cancer could not be specified, but it did not antedate the fibrosis.

MISCELLANEOUS

56. Britten, Rollo H., and L. R. Thompson. A health study of ten thousand male industrial workers. Statistical analysis of surveys in ten industries. Public Health Bulletin No. 162. 1926.

Summary of anthropometric and physiological data obtained in physical examinations, together with incidence of certain physical defects.

57. Gardner, L. U., and D. E. Cummings. Studies on experimental pneumonokoniosis. VI. Inhalation of asbestos dust: Its effect upon primary tuberculous infection. Jour. Ind. Hyg. 13: 65–66. 97–112. 1931.

"Guinea-pigs have been exposed for eight hours daily for periods as long as two and one-third years to an atmosphere containing approximately thirty-five million particles per cubic foot of asbestos dust (Canadian chrysotile) 1.5 microns and less in diameter. Rabbits and albino rats have likewise been exposed for shorter periods (330 days)."

58. Gloyne, S. R. The presence of the asbestos fibre in the lesions of asbestos workers. Tubercle 10: 404–407. 1929.

Asbestus fibers, but not asbestosis bodies, were found in asbestos cases.

59. King, E. J., and M. Dolan. Silicosis and the metabolism of silica. Can. Med. Assoc. Jour. 31: 21–26. 1934.

Urinary silica content of 56 miners was 2.2 mg per 100 cc; of 40 students 1 mg per 100 cc (single specimens). Analytical procedure is described.

60. Miller, J. W., and R. R. Sayers. The physiological response of peritoneal tissue to certain industrial and pure mineral dusts. Public Health Reports 51: 1677-1688. 1936. Reprint No. 1787.

32 dusts were classified according to the reaction produced on intraperitoneal injection into guinea pigs.

61. Miller, W. S. The lung. Charles C. Thomas, Springfield, Ill. Baltimore. 1937. pp. 57.

62. Norris, G. W., and H. R. M. Landis. Diseases of the chest and the principles of physical diagnosis. W. B. Saunders Co., Philadelphia. 5th edition. 1933.

63. Extracts from the annual report of the Chief Inspector of Factories and Workshops. 1935. Chapter III—Health. His Majesty's Stationery Dept. London.