

51

250044



Asbestosis*

R. R. SAYERS, M.D., F.A.P.H.A., AND
W. C. DREESSEN, M.D.

*Senior Surgeon, and Passed Assistant Surgeon, U. S. Public
Health Service, Washington, D. C.*

ASBESTOS is well adapted for use as a textile material because of its fibrous nature. On account of this, as well as its non-combustible and excellent insulating properties, it has come to be used in ever increasing quantities during the past 20 years. Hence, it is not surprising that Gloyne and Merewether¹ should refer to pulmonary asbestosis as a "modern disease."

The first record of a case of asbestosis seems to have been made by Montague Murray in 1900. The first complete description of the disease and of the "curious bodies" seen in lung tissue and sputum appeared in 1927 when Cooke² and McDonald³ reported 2 cases of asbestosis and listed their reasons for believing that asbestosis bodies originate from asbestos fibers that reach the lungs. Their papers aroused general interest in the subject and numerous others appeared soon afterward. Hoffman⁴ appears to have been the first American to call attention to the magnitude of the asbestosis problem. In 1918 he reported that 13 deaths from asbestosis had occurred among asbestos textile workers, and about the same time Pancoast, Miller, and Landis⁵ reported on 17 cases of asbestosis. Mills's⁶ paper was the first

pathological report on asbestosis published in the United States, and in the same year, 1930, Lynch and Smith⁷ reported on asbestosis bodies found in the sputum of asbestos workers.

The Public Health Service was requested by the State Board of Health and the Industrial Commission (Administrator of the Workmen's Compensation Act) of North Carolina to assist them in making an engineering and medical study of the health hazards in the asbestos textile industry of that state. The objectives of this study were:

1. To make a medical study of the effects of long-continued inhalation of asbestos dust on the human body.
 2. To identify the manufacturing processes that create dust, and to recommend practices for reducing the dust exposure of workers.
 3. To find out what concentrations of asbestos dust can be tolerated without injury.
- It is the purpose of this paper to review briefly the principal findings of this study.⁸

ENGINEERING FINDINGS

The main asbestos raw material used for textiles is Canadian chrysotile, which is a hydrated magnesium silicate containing no quartz.

The textile manipulations of asbestos are very similar to the production of cotton or woolen goods. The crude fiber is sent first to the preparation department, then, in the order named, to the carding machines, spinning frames, winding bobbins, twisting ma-

* Read before the Industrial Hygiene Section of the American Public Health Association at the Sixty-seventh Annual Meeting in Kansas City, Mo., October 13, 1932.

TABLE I

Summary of Results Showing the Exposure of Asbestos Textile Workers Under Controlled and Uncontrolled Working Conditions

Equipment	Number of Duct Connections	Volume of Air Handled (CFM)	Dust Concentration With Exhaust (MPPCF)	Dust Concentration Without Exhaust
Willowing (opening)	* 2	825-1,000	3.6	11.1-36.0
Piling†	1	1,025	2.0	5.4
Picking	3	2,570	6.7	34.3-74.3
Carding (primary)	3	1,420	2.0	72.3
Carding (roving)	4	1,440		
Spooling	‡ 1	46.5	2.9	13.1
Weaving (broadloom)	2	1,300	.7	4.7-49.7
Brusher calendarer	3	1,650	1.0	11.1

* Equipped with pneumatic conveyor.

† Exhausted bin or compartment.

‡ Individual cone for each spool and connected to exhaust manifold.

chines, spooling frames, and finally to the looms and miscellaneous fabricating devices.

In all, 242 dust counts were made in estimating the exposure of these asbestos workers. Only summary engineering findings, or dust concentrations as they relate to medical findings, will be discussed. Summarized results of dust concentrations under controlled and uncontrolled conditions appear in Table I. It will be noted that 74.3 million particles per cubic foot (m.p.p.c.f.) is the maximum concentration of dust encountered. Even this maximum figure is much lower than is frequently encountered in other siliceous trades where it has not been uncommon to encounter maximum dust concentrations of 1,000 m.p.p.c.f.

The dust in asbestos plants is made

up of particulate matter and fibers. The median size in microns of particulate matter ranged from 1.35 to 2.40. Particles were smallest in the carding and preparation processes, and largest in weaving. The median length of fibers in microns ranged from 7 to 16.3. As might be expected, the longest (400 μ) were noted in case of weaving and the shortest in preparation.

TABLE II

Median Length of Fibers Sampled With an Owens Jet Apparatus

Activity	Median Length of Fibers in Microns
Willowing	7.0
Picking	9.5
Carding	2.3
Twisting	12.2
Weaving (broadloom)	16.3

TABLE III

The Frequency Distribution of Particulate Dust Suspended in the Air of Asbestos Textile Plants

Type of Process Where Sample Was Taken	Median Size (in Microns)	Geometric Standard Deviation	Percentage Frequency of Each Particle Size Group (in Microns)											More	Total
			0	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5		
Preparation	1.35	1.56	5	11	37	22	10	4	2	1	0	0	0	0	100
Carding	1.35	1.57	1	9	24	25	9	16	4	5	1	5	1	5	100
Wile spinning	1.30	1.33	0	1	25	38	28	4	1	1	1	0	1	1	100
Twisting	1.22	1.24	5	30	24	14	9	5	4	5	0	1	5	1	100
Weaving (broadloom)	1.35	1.31	1	4	43	27	10	1	2	3	4	0	3	1	100
Weaving (taper)	2.40	1.64	0	1	11	24	14	12	7	13	7	5	6	1	100

The significance of dust concentrations and physical characteristics of these air contaminants will be referred to in the course of subsequent discussion.

The workers who were found particularly liable to develop severe forms of asbestosis were the willowers, pickers, carders, mule and ring spinners, twistors, and cloth weavers. Their exposure was found to be as follows:

	Dust concentration MPPCF
Willowers	11.1-36.0
Pickers	34.3-74.3
Carders and tenders	19.1
Mule spinners	2.6-7.9
Ring spinners	3.2-8.3
Twistors	3.2-13.2
Cloth weavers	
Dry	4.7-49.7
Wet	4.7-11.1

MEDICAL FINDINGS

Medical examinations were made of 541 men and women representing practically all the employees at the time of study. Five-sixths of them were native born Americans of Anglo-Saxon stock and the remainder were Negro males. The three factories studied had been in operation from 6 to 16 years. About 15 months before the study, approximately 150 workers were replaced by new ones with little or no asbestos experience. As a consequence, there was an abnormally large percentage of workers with less than 5 years' employment in the asbestos textile industry and an abnormally small percentage who had worked 10 years or more in the industry. More than 200 had worked at comparable occupations in cotton or woolen textile plants, but exposures to pneumoconiosis producing dusts were inconsequential.

Characteristics of asbestosis—Pulmonary asbestosis was the principal physical defect found on examining the 541 persons. This disease, a form of pneumoconiosis caused by long continued inhalation of asbestos dust, is

characterized pathologically by diffuse interstitial pulmonary fibrosis and the presence of asbestosis bodies in the lungs. Clinically, the chief symptoms are progressive dyspnea, variable cough, substernal chest pain, blood streaked sputum, decreased chest expansion, emaciation, weakness, clubbed fingers, or curved nails. Late in the disease, the dyspnea becomes distressing, cyanosis may occur, and there may be severe paroxysms of coughing productive of tenacious sputum. The characteristic chest X-ray shows granular or ground glass markings with more or less obliteration of usual linear pulmonary markings, localized in mid-lung and bases. The grainy appearance may become quite generalized with evidence of emphysema usually in the apices. Nodular or nodulo-conglomerate shadows of silicosis are not observed, but whether this is due to a peculiarity of asbestos dust or to rare occurrence of extremely high dust exposures, it is impossible to say. Shagreening of the heart shadow is not infrequently observed and seems to be most common in workers exposed to a high proportion of fiber (e.g., twisting, broadcloth weaving). By fluoroscopy, diminished excursion of diaphragm is seen. Peaking deformities of diaphragm, however, occur less frequently than in silicosis.

Asbestosis bodies found in the lungs and sputum are characteristic. There is good evidence that these bodies originate as a cellular response to inhaled fibers.⁹ Asbestosis bodies consist of a core of asbestos fiber surrounded by iron-containing protein deposits. They are golden yellow in color, and do not stain with ordinary histologic stains but become brilliant blue when treated with potassium ferricyanide. They are variable in form and size and characteristically are slender, elongated, segmented structures with bulbous ends which give them a dumbbell or drum-

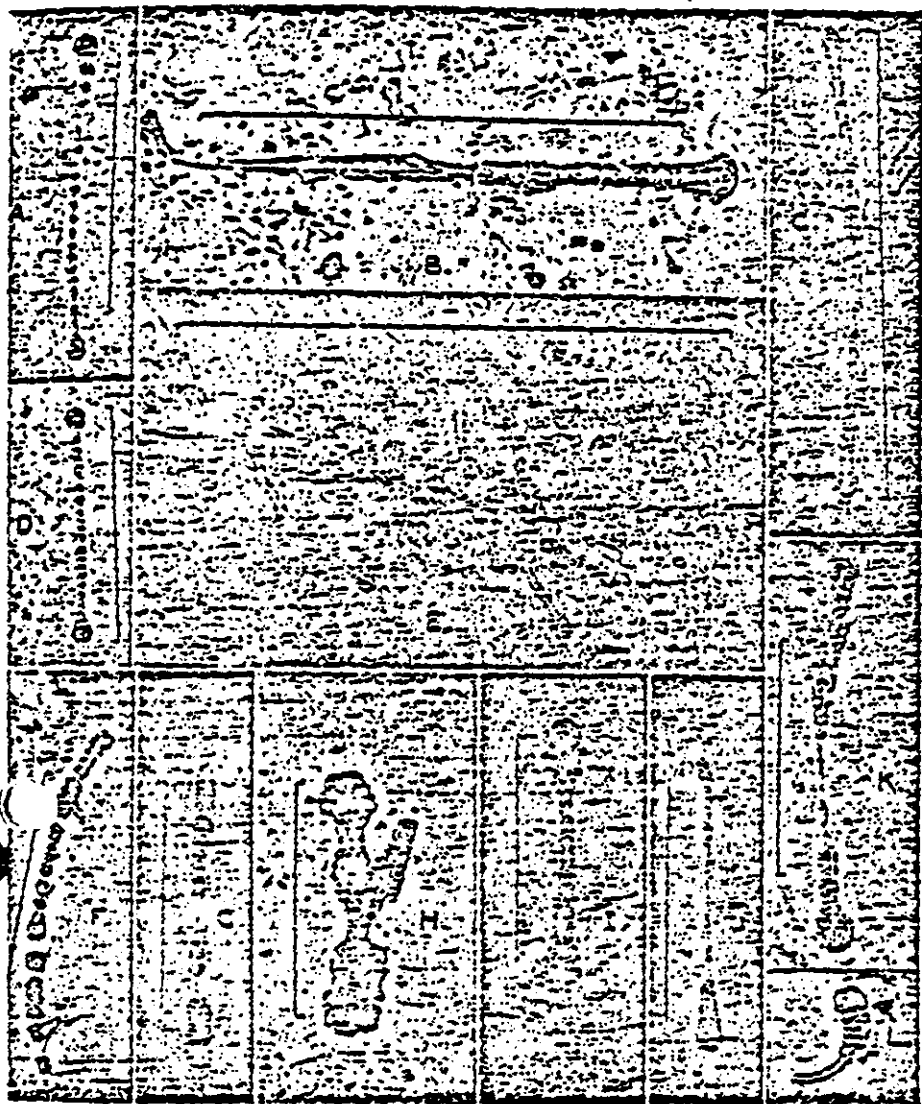


FIGURE II—Photomicrographs of Asbestos Bodies Found in Sputum. Enlarged 530 Times (Except E, Which Is Enlarged 310 Times). A Scale, Ruled in Units of 50 Microns, Has Been Drawn Beside Each Asbestos Body.

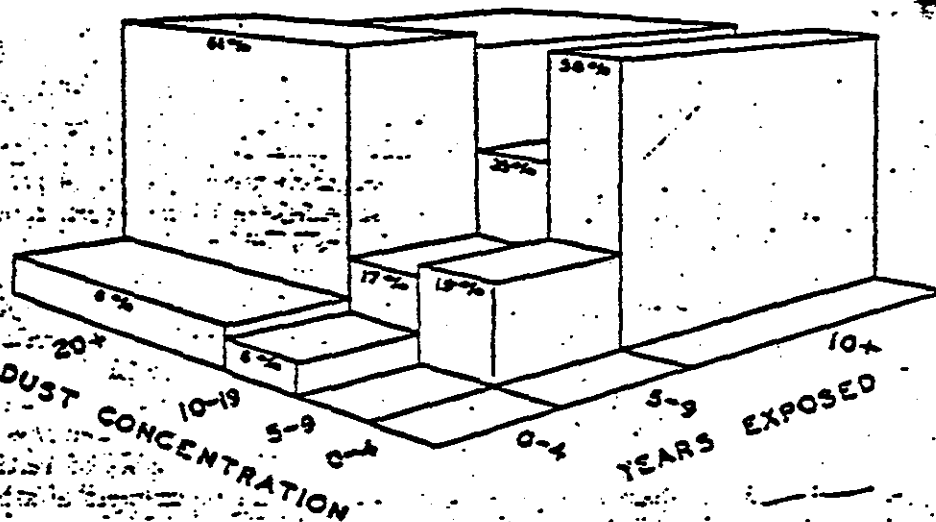
ck shape (Figure II). Occasionally forked or Y-shaped body is observed. They range from 10 to 130 μ in length, averaging about 35 μ .

It has been suggested that damage to the lungs occurs while the body is being formed, but once the body is formed the fiber in the core is rendered inert by its coating of iron.¹⁰ The finding in the sputum of clumped asbestos bodies in radial pattern or rosette

(Figure II E), which is common in lung sections, has been suggested by Stewart, Tattersall, and Haddow¹¹ as a clear indication of disintegration of lung tissue whether by a process of simple suppurative broncho-pneumonia, or as a result of secondary tuberculous infection. In either case, they feel that it strongly indicates a definite underlying asbestosis.

On single sputum specimen analysis

FIGURE IV—Percentage of Asbestos Textile Workers, Classified by Average Dust Concentration (Measured in Million Particles per cu. ft.) and Duration of Exposure, Found to Have Ground-Glass Lung-Field Markings.



is probably due to total amount of asbestos dust inhaled. Difference in amount of physical exertion may play an important rôle—a person in a sedentary job having a smaller need for oxygen inhales less air and consequently fewer particles of asbestos.

It was noted that the relation between dust exposure and the incidence of ground glass lung field markings was not a simple one. Even when length of employment was disregarded, it was found that incidence of these markings

was proportionately lower at 10 to 19.9 m.p.p.c.f. than it was at the next higher or next lower concentrations. Although it is probable that these differences represented sampling errors due to small numbers of exposed persons, it may be that asbestos fiber excites a different and possibly more severe reaction than asbestos particles.

On account of large labor turnover, as well as the short time that the plants had been in operation, no exact estimate of incidence of asbestosis can

TABLE IV

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	34	19	5
	Percentage	2%	5%	0%
5 to 9.9.....	Affected	0	6	13
	Exposed	70	37	19
	Percentage	0%	16%	68%
Over 10.....	Affected	8	22	21
	Exposed	134	43	36
	Percentage	6%	51%	58%

be made that the asbestosis for McConr similar creases tion. but few exposure are not prevalent several included

SAVE LI

For to have conditio centrati dust c produce healthy working interpre difficult. exposed bestosis played doubtful 2.5 to Becm

Percenta: Concer Textile Measur and i Contro

Dust Co: Million per Cu Over 10 5 to 9.9 2.5 to 4 Under 2

* Data ? Data

be made. Table IV shows, however, that the percentage of persons with asbestosis increases greatly with increasing length of employment. Lanza, McConnell, and Fehnel¹² reported a similar trend. The incidence also increases with increasing dust concentration. For the reason that there are but few cases with more than 15 years' exposure, the percentages in Table IV are necessarily minimal estimates of prevalence of asbestosis even though several of the former employees are included.

SAFE LIMITS OF EXPOSURE TO ASBESTOS DUST

For practical purposes it is useful to have a definition of safe working conditions. 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. Below 2.5 m.p.p.c.f. the interpretation of Table IV offered no difficulties, since none of 39 persons exposed to that concentration had asbestosis, although only 6 had been employed more than 5 years. Three doubtful cases of asbestosis fell in range 2.5 to 4.9 m.p.p.c.f.

~~Asbestosis Clean-cut cases of asbestosis~~

TABLE V

Prevalence 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 *	Extensive Use of Dust- Control Measures †
Over 10	48	7
5 to 9.9	29	17
2.5 to 4.9	15	32
Under 2.5	9	44

* Data from Table IV.

† Data from reference 13.

mentioned in dust concentrations exceeding 5 m.p.p.c.f., and because they were not found at lower concentrations, 5 m.p.p.c.f. may be regarded tentatively as the threshold value for asbestos dust exposure.

A supplemental engineering study¹³ was made in an asbestos textile factory in which dust control equipment had recently been installed. This showed that means are already available for reducing the dust exposure of a majority of asbestos textile workers to less than 5 m.p.p.c.f. The basis of this control was on exhaust ventilation near source of dust.

CONCLUSIONS

As in other forms of pneumoconiosis, occupational history, and clinical and X-ray (or pathologic) findings must be in harmony before a sound diagnosis can be made. The occupational history should be a reflection of manufacturing processes since the job designation refers to a stage in manufacture. The occupations which were found particularly liable to induce severe forms of asbestosis were willow, pick, card, spin, twist, and cloth weave. Confirmation of the hazardous nature of some of these occupations is the frequency with which the occupational designation of carder, weaver, or spinner is encountered in autopsy reports of cases.¹⁴⁻¹⁸ The following are the outstanding findings determined in this study:

1. In a study of 541 employees of North Carolina textile mills, pulmonary asbestosis was the principal physical defect found.
2. The most serious forms of the disease were observed in carders, spinners, weavers, twisters, willowers, and pickers.
3. Exposures ranged from 0.10 to 76 m.p.p.c.f.
4. Dust contaminants of air in asbestos textile plants are both particulate and fibrous.
5. It is imperative that findings of occupational history, clinical examination, and X-ray film all be considered in making diagnosis of a case.
6. Definite clinical and roentgenographic

evidence of asbestosis is observed in exposed persons after 5 to 10 years of work in exposures exceeding 5 m.p.p.c.f.

7. It appears that if asbestos dust concentrations in the air breathed are kept below 5 m.p.p.c.f. new cases of asbestosis will not appear.

8. Methods for controlling the dust below this tentative threshold are already available for most of the processes in the industry.

REFERENCES

1. Gloyne, S. R., and Merewether, F. R. A. *Asbestos. Occupation and Health* (Suppl.), International Labour Office, Geneva, 1938.
2. Cooke, W. E. Pulmonary Asbestosis. *Brit. M. J.*, 2:1024-1025, 1927.
3. McDonald, Stuart. Histology of Pulmonary Asbestosis. *Brit. M. J.*, 2:1025-1026, 1927.
4. Hoffman, F. L. Mortality from Respiratory Diseases in Dusty Trades (Inorganic Dusts). *Bull. U. S. Bur. Lab. Stat.*, No. 231, 1912, pp. 176-180.
5. Fraumeni, H. K., Miller, T. G., and Landis, H. R. M. A Roentgenologic Study of the Effects of Dust Inhalation Upon the Lungs. *Tr. A. Am. Physicians*, 32:97-108, 1917.
6. Miller, R. G. Pulmonary Asbestosis: Report of a Case. *Minnesota Med.*, 13:495-499, 1930.
7. Lynch, K. M., and Smith, W. A. Asbestosis Bodies in Sputum and Lung. *J. A. M. A.*, 95:659-661, 1930.
8. A Study of Asbestosis in the Asbestos Textile Industry. *Pub. Health Bull. No. 241*, Aug., 1932.
9. Gloyne, S. R. The Asbestosis Body. *Lancet*, 1:1351-1356 (June 23), 1942; *Quart. N.*, and *Byrdin, A. Die Staubkrankheit eines Asbestearbeiters und die Beschaffenheit der im Sputum und in Gewebeschneidungen. Archiv. f. Geschichte d. Naturgesch.*, 5:24-50, 1937; Gloyne, S. R. The Morbid Anatomy and Histology of Asbestosis. *Tubercle*, 14:441-451, 1933-1937; 530-533, 1937.
10. Kettle, E. H. The Interstitial Nodules Caused by Various Dusts and Their Influence on Tuberculous Infections. *J. Path. & Bact.*, 15:385-403, 1932.
11. Stewart, M. J., Tatterall, N., and Hutton, A. C. On the Occurrence of Clusters of Asbestosis Bodies in the Sputum of Asbestos Workers. *J. Path. & Bact.*, 35:737-741, 1932.
12. Lauer, A. J., McConnell, W. J., and Tolbert, J. W. Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers. *Pub. Health Rep.*, 50:1-12, 1935. *Reprint No. 1645*.
13. Page, R. T., and Bloomfield, J. J. A Study of Dust Control Methods in an Asbestos Fabricating Plant. *Pub. Health Rep.*, 52 (Nov. 26), 1937. *Reprint No. 1523*.
14. Lynch, K. M., and Smith, W. A. Pulmonary Asbestosis II. *Am. Rev. Tuberc.*, 23:443-460, 1911.
15. Eghter, D. S. Pulmonary Asbestosis. *Am. Rev. Tuberc.*, 31:25-34, 1935.
16. Stock, G. A. Pulmonary Asbestosis. *M. Ed. Soc. Admin.*, 10:126-129, 1933.
17. White, T. P. Pulmonary Asbestosis. *Tr. Med. Soc. North Carolina*, 1935, pp. 259-262.
18. Lynch, K. M., and Smith, W. A. Carcinoma of Lung in Asbestosis. *Am. J. Cancer*, 14: 56-64, 1935.