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A STUDY OF ASBESTOSIS IN THE ASBESTOS TEXTILE INDUSTRY

By

WALDEMAR C. DREESSEN, Passed Assistant Surgeon
J. M. DALLAVALLE, Passed Assistant Sanitary Engineer
THOMAS I. EDWARDS, Technical Editor
J. W. MILLER, Pathologist
R. R. SAYERS, Senior Surgeon

With the assistance of
H. F. EASOM, M. D., Director
M. P. TRICE, Engineer
Division of Industrial Hygiene
North Carolina State Board of Health

From the Division of Industrial Hygiene
National Institute of Health

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ABSTRACT

Asbestosis, a lung disease caused by long-continued inhalation of asbestos dust, was the principal physical defect found on medical examination of 541 men and women employed (or recently employed) in three asbestos textile factories. The primary effect of asbestos dust seems to be initiation of fine, interstitial, pulmonary fibrosis which was not observed to progress as far as the nodular stage. This fibrosis was observed in post-mortem examinations of three former asbestos workers. During life, lung fibrosis can be detected by X-ray examination. The more important symptoms of asbestosis are progressive dyspnoea, variable cough which sometimes raises blood-streaked sputum, and loss of weight. The processes that produce dust in asbestos textile factories were studied and recommendations for dust control are incorporated in this report. Enough dust counts (242) were made in all parts of these factories so that the dust exposure of each worker can be estimated. The percentage of persons in different occupational groups who were affected by asbestosis or any of its symptoms varied with the average dust concentration to which they were subjected and with their length of employment. The only cases of asbestosis, three in number, found below 5 million particles per cubic foot were diagnosed as doubtful; well-established cases occurred at higher concentrations. It appears from these data that if asbestos dust concentrations in the air breathed are kept below this limit new cases of asbestosis would not appear.

A STUDY OF ASBESTOSIS IN THE ASBESTOS TEXTILE INDUSTRY

INTRODUCTION

Several important properties of asbestos have led to its widespread use in industry. As everyone knows, it is noncombustible and an excellent insulator against heat and electricity. Fewer people seem to be aware that its fibers are sufficiently long and flexible to be carded, spun, and woven like wool, flax, or cotton. Utilizing these properties of asbestos, industries have been developed in this country which employed more than 6,000 men and women in slightly more than 60 establishments in 1935.

Another property of asbestos has been discovered more recently. When asbestos dust is inhaled daily, year in and year out, a lung disease termed asbestosis may develop. In many respects asbestosis resembles silicosis, but it differs from silicosis in several important particulars which will be discussed in the medical section of this report.

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 Cooke (9) and McDonald (22) reported two cases of asbestosis and listed their reasons for believing that asbestosis bodies originate from asbestos fibers that reach the lungs. The publication of their papers aroused general interest in the subject and numerous papers appeared soon afterward. Hoffman (46) 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 Panconst, Miller, and Landis (29) reported on 17 cases of asbestosis. Mill's (26) paper was the first pathological report on asbestosis published in the United States, and in the same year, 1930, Lynch and Smith (38) reported on asbestosis bodies found in the sputum of asbestos workers.

Because several satisfactory literature reviews of asbestosis have appeared in recent years there seems to be no need to present a complete literature review in this report. Some of the more useful literature reviews have been listed in the annotated bibliography. In Merewether's (25) review emphasis is laid on the relation of asbestosis

to working conditions. The clinical aspects of the subject are well treated. Gloyne's (16) discussion of the pathology of asbestosis is particularly helpful. Middleton's (47) Milroy lectures on pneumoconiosis contain a section on asbestosis. Egbert (13) has summarized the literature reports on 28 fatal cases of asbestosis. "Active pulmonary tuberculosis was present in 6 instances of the total 28 cases; the fatal outcome was primarily the result of tuberculosis in 3 instances."

OBJECTIVES OF THE STUDY

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 health hazards in the asbestos textile industry.[1] The objectives of this study were threefold: (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 and, when necessary, equipment that will reduce the dust exposure of workers; and (3) to find out what concentration of asbestos dust can be tolerated without injury to health.

[1] This represents one of the first uses of Social Security funds for a cooperative project carried on by the Public Health Service and a State health department.

THE WORKING ENVIRONMENT

METHODS AND INSTRUMENTS USED IN THE STUDY OF THE WORKING ENVIRONMENT

The evaluation of the working environment and its relation to the health of workers forms the basis of all field investigations conducted by the Division of Industrial Hygiene. The procedure followed by this Division is described in Public Health Bulletin No. 217 (2). Therefore, only a brief discussion of the subject is presented in this report.

The study of an occupational dust problem begins with a preliminary survey designed to obtain data on all the occupations in a given plant. It includes the number of workers employed in each occupation and the materials used in each process. The survey also reveals the number of workers exposed to a given hazard. Following the preliminary survey an evaluation of the occupational environment is made. In the dusty trades this includes a determination of the amount of dust present in the air, its composition and particle size and the measurement of the effectiveness of exhaust systems where they are used to control dust hazards. The latter, in this instance, involved airflow measurements of several exhaust systems by means of a pitot static tube. The effectiveness of these systems was determined by dust counts.

THE MANUFACTURE OF ASBESTOS TEXTILES

Approximately 90 percent of the asbestos used in these plants is obtained from Canada. The remaining 10 percent comes from Arizona or South Africa, and, infrequently, from Russian and Australian sources.

Much of the machinery that is employed for the manufacture of asbestos textiles is identical with the machinery used for the production of cotton and woolen goods. Crude asbestos fiber is sent first to the preparation department and then, in the order named, to the carding machines, spinning frames, winding bobbins, twisting machines, spooling frames, and finally to the looms and miscellaneous fabricating devices.

The four plants investigated in the present study fall naturally into two classifications. Two plants convert crude asbestos and raw cotton into asbestos yarn and woven goods, but do not prepare the

products for the ultimate consumer. The other two plants purchase both asbestos yarn and unfinished woven asbestos tape. These establishments weave brake bands and convert their own woven product, as well as purchased material, into finished goods. The practices followed in the plants studied are schematically presented in the flow sheet shown in figure 2.

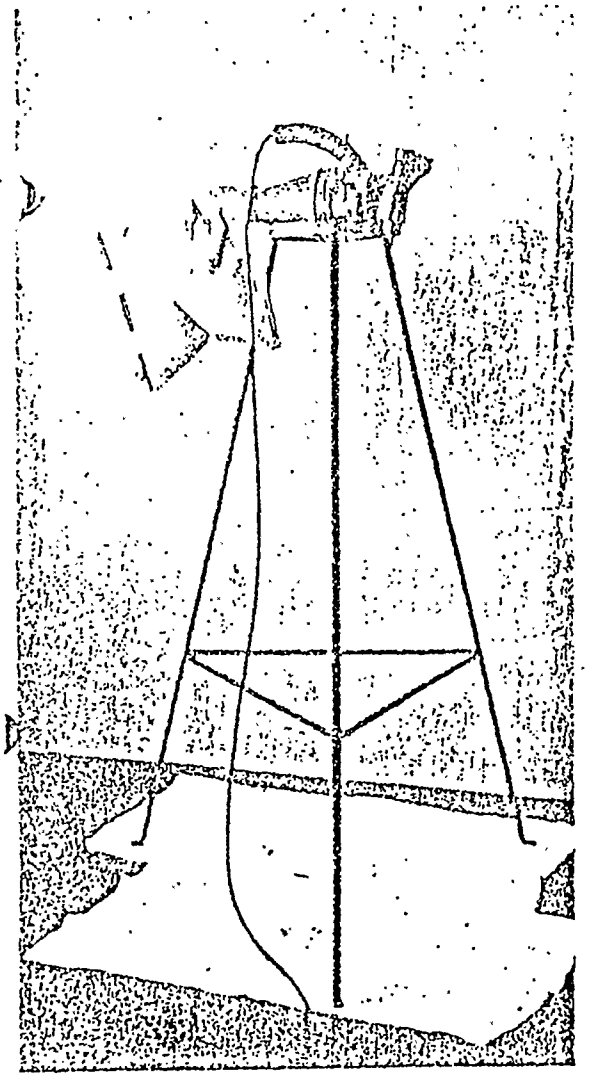


FIGURE 1.—VACUUM CLEANER BAG ARRANGEMENT FOR COLLECTING SUSPENDED DUST SAMPLES.

PREPARATION

Crushing.—The first step in the manufacture of asbestos textiles is the crushing of the crude stock in a milling machine. This is accomplished by large iron rollers which revolve in a steel pan in which the crude material is placed. (See fig. 3.) This operation separates the fibers and loosens the impurities that survived the cobbing of the ore at the mines. The crushing operation does not produce much dust, the fibers being disintegrated by a mashing and twisting action.

Most crude ore must be crushed for 20 minutes, although in some cases it may be satisfactorily disintegrated in 5 minutes. The crushed asbestos has a matted appearance. Following the crushing, the asbestos is replaced by hand in the bags for delivery to the willower.

Willowing.—The willowing or opening of the asbestos is essentially a cleaning operation. The willower beats and shreds the matted

FLOW SHEET

AMERICAN PRACTICE

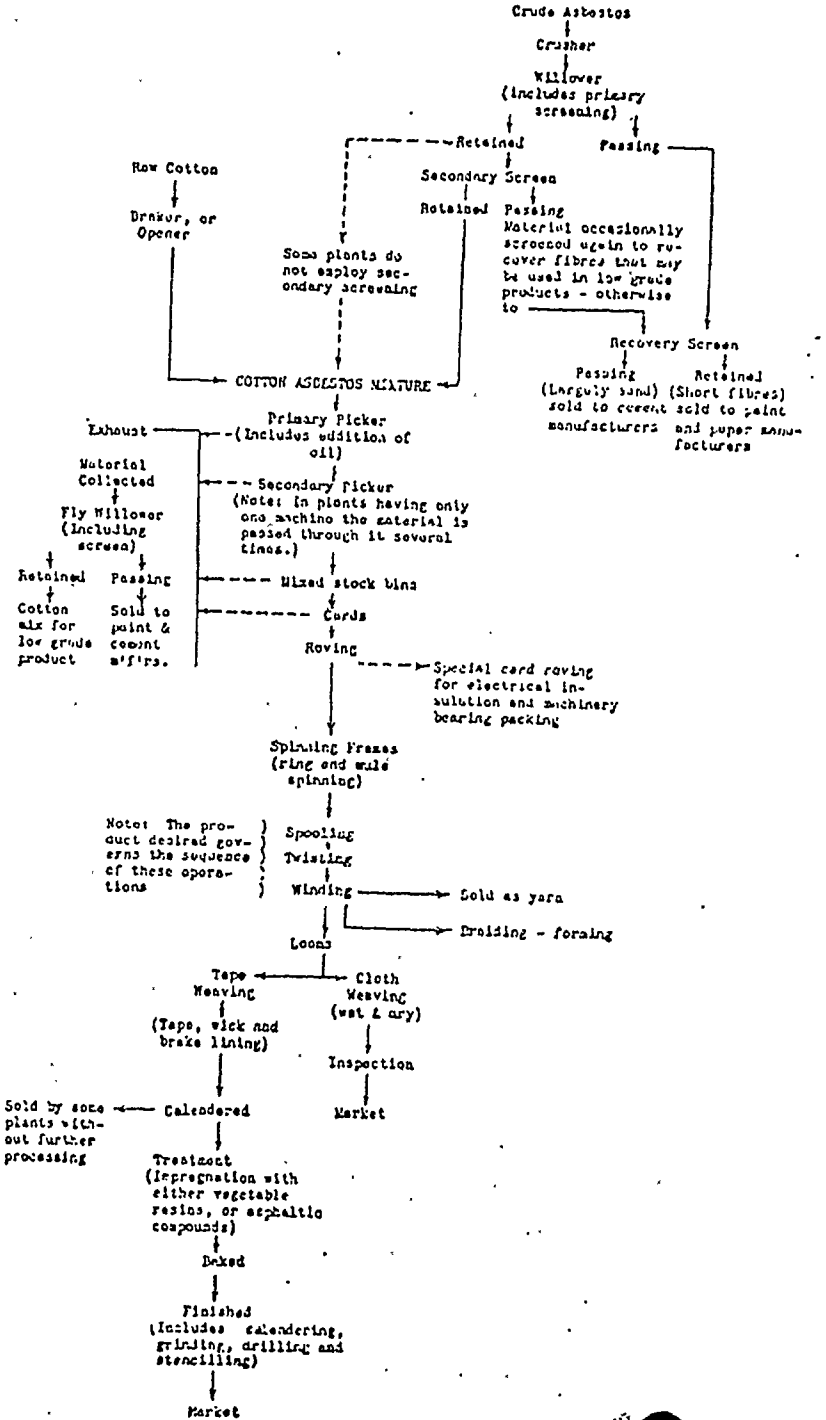


FIGURE 2.—FLOW SHEET OF AMERICAN PRAC

asbestos masses into individual fibers and removes some of the rock. The asbestos fibers that are retained are delivered directly to a storage bin or conveyed to one by pneumatic duct. (See fig. 4.) The material falling to the floor is screened in order to separate the grit from the short fibers. The grit is sold to cement manufacturers and the short asbestos fibers to paint, paper, and roofing manufacturers.

The willower is fed manually by a laborer who lifts the crushed asbestos into the willower hopper by means of a pitchfork. The beating and combing action which takes place within the machine produces a large amount of dust which escapes from the loose-fitting enclosure. The production of dust is increased when waste asbestos, recovered from the cyclone separators which form a part of the dust-

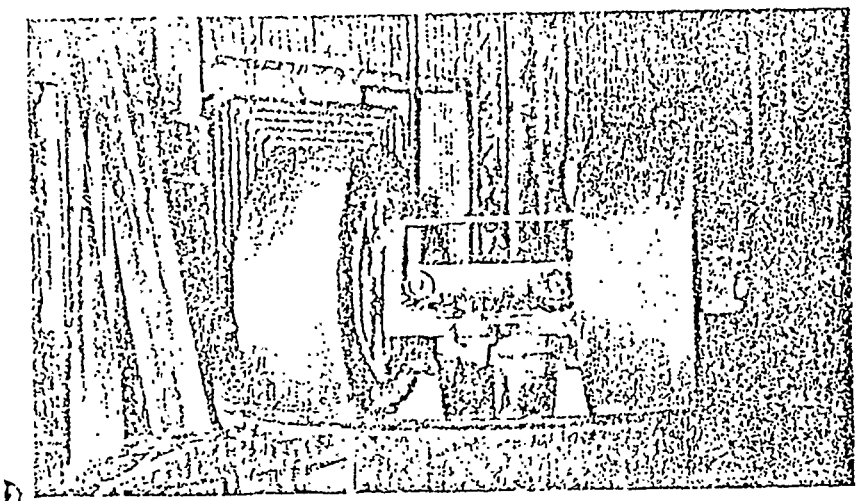


FIGURE 3.—MILL FOR CRUSHING ASBESTOS FIBER.

removal system, is willowed for reclaiming purposes. This latter operation is called fly willowing.

At one of the plants studied, the willowed asbestos is passed over a vibrating screen. This operation insures a cleaner product, since additional grit and short fibers are removed. Where a second screen is not available the asbestos may be passed through the willower a second time, a practice which is followed at two of the plants studied. The screening of asbestos is a decidedly dusty operation.

Mixing.—The asbestos fibers consist of blends of various grades and often include a certain amount of waste recovered by the fly willower. This process is followed in order to obtain end products with the characteristics demanded by the market. When a batch of asbestos has been made, it is then mixed with cotton. Asbestos fibers must be mixed with cotton before they can be spun and woven. The amount of cotton used depends upon the quality of the willowed asbestos fibers used. As a rule, from 5 to 15 percent by weight of all the asbestos textiles produced in this country is cotton.

Piling.—At the plants studied, the mixing is accomplished in two steps, the first consisting of placing the cotton and asbestos in alternate layers in a pile upon the floor, and the second step of feeding the mixture into a picker machine. The batch making, or the placing of the cotton and asbestos, is known as piling. It is productive of much dust since it is necessary to spread out both the cotton and asbestos into uniform layers, which involves the use of a rake and much trampling of the materials.

Picking.—After the mixture is made, it is lifted into the breast-high hopper of the picker machine. The material emptied into the

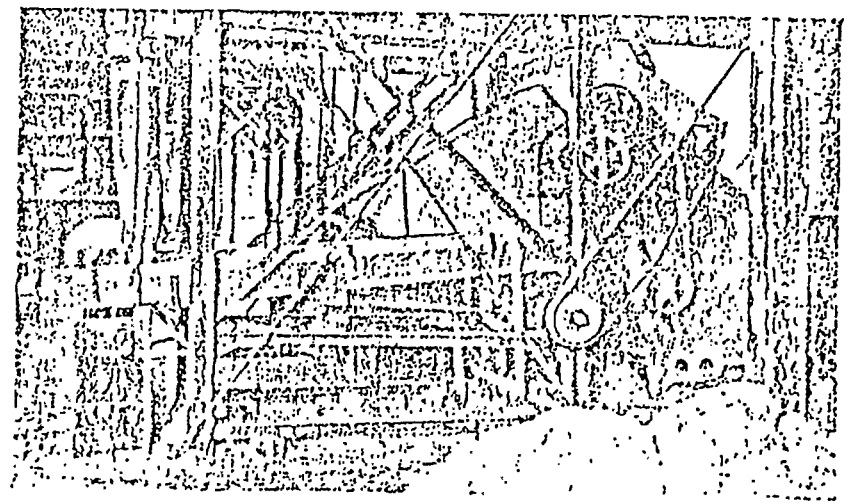


FIGURE 4.—FLY OPENER OR WILLOWER (NOTE EXHAUST).

hopper displaces air in the hopper which causes dust and fibers to escape upward into the face of the worker. In addition, the cylinder and accessory rollers of the picker machine operate at a high speed and much fine material escapes from the many small openings in the frame. The dust that is fanned out of the machine, together with that produced in the feeding operation, makes the pickerman's occupation comparatively dusty. In some plants two pickers are operated in series and in others only one, through which the material may be passed several times. In either case the picker discharges directly or by pneumatic duct into a collector bin. Where only one machine is available, however, the pickerman must move the material by pitchfork from the collector bin to the floor of the picker room for its recirculation through the picker. At one of the plants studied there are two pickers operated in series and connected by pneumatic

ducts. Here the collector bin is also the storage bin. In order to prevent the mixed material from accumulating in a steep pile a man is stationed in the stock bin at all times. This occupation creates considerable dust, since the cotton-asbestos mixture is delivered into the bin by overhead duct discharge. In order to reduce the dust exposure of the stock-bin man, his position is made interchangeable with the pickerman, each of whom spends a half of every day in the stock bin and a half day at the picker. In the plant having only one picker, the mixture is trucked from the collector bin to the storage bin. At one of the plants studied, exhaust ventilation is provided for the picker machines and the mixed stock storage bins. Oil sprays are

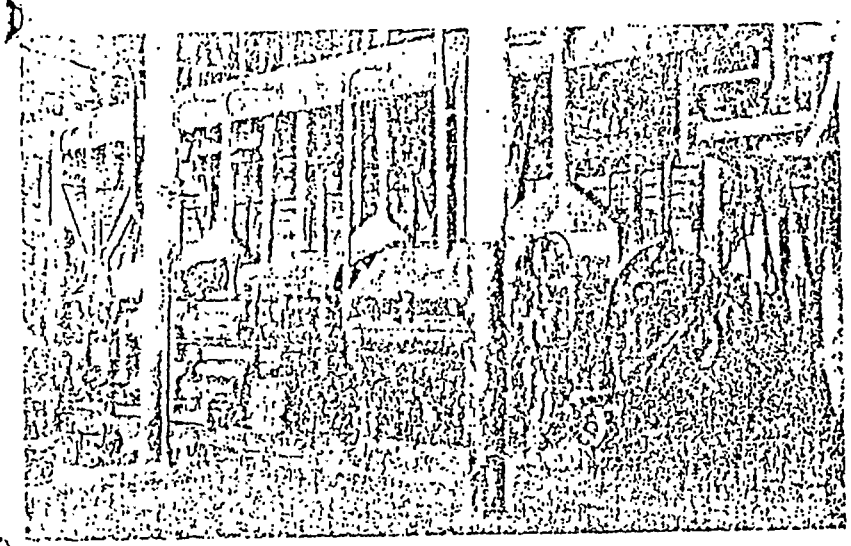


FIGURE 5.—EXHAUSTED CARDING MACHINES.

frequently used within the picking machines in an attempt to prevent the formation of dust.

CARDING

From the storage bins the stock, as it is called, is hand-trucked to the carding machines where it may be piled upon the floor or fed into the hopper of the breaker card.

The cards perform several duties. They give the fibers an additional mixing, arrange them in parallel positions, and form them into roving. The term roving is applied to the strands of parallel fibers that are produced. A carding unit consists of two almost identical machines. The one through which the cotton-asbestos mixture passes first is termed the breaker card, and the other, the finishing card. The two machines are quite similar in that a large revolving cylinder with several accessory rollers performs the major portion of the work. (See [illegible].) The carding operation is quite similar to that of the picker, the essential difference being that the roller and cylinder surfaces are studded with fine steel bristles rather than large hooks. Some of the rollers straighten the fibers by combing out the tangles, while others doff, or remove, them from the combing rollers and the cylinder. Aside from these main features, the two units differ slightly. The breaker card is equipped with a feeding hopper similar to that used on the picking machine, while the finishing card is provided with a device for forming the carded fibers into strands. In the plants studied a system of conveyor lattices transfer the carded fibers from one machine to the other. The outside strands of roving are returned

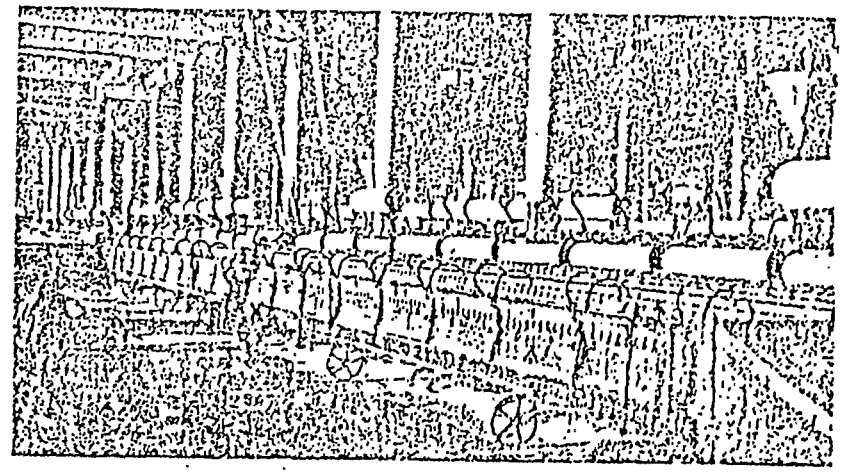


FIGURE 6.—SPINNING MULES.

to the picker for reworking. The remainder of the roving is wound on long rollers with flanged ends, called jack spools.

The bristle-studded cylinders of the carding machines revolve at high speeds and much lint is thrown into the atmosphere. The effect produced by the revolving cylinders and wheels is similar to that of a fan, and as a result much of the dust remaining on the stock at this point, as well as asbestos and cotton fibers, is blown into the air. All carding machines studied were provided with exhaust ventilation.

While practically all of the roving is spun into yarn, some of it is marketed without further processing other than winding on spools or into coils. In this form it is used for electrical insulation and for conversion into packing for machine bearings.

SPINNING

The jack spools containing the roving may be conveyed either to the mule spinning machines shown in figure 6 or to the ring spinning frames. The spinning, on whatever machine performed, is a twisting operation which imparts strength to the strand of fibers by inter-

locking or meshing them together. The ring spinner twists the roving as it is wound on a spindle, while the mule spinner performs the same treatment in two operations; first, spinning it in 5- or 6-foot lengths and then winding it on a spindle. The ring spinner makes a yarn that is stronger and better in many respects than that produced on the mule spinner.

WINDING, TWISTING, AND SPOOLING

The spun yarn is converted into stronger thread in two operations. It is taken first to the winding machines where it is transferred from spindles to tubes. After being wound on tubes, the yarn goes to the twisting machines where the thread from two or more tubes is brought together and twisted into one thread. In the twisting operation the thread is frequently strengthened with brass, zinc, or copper wire, or

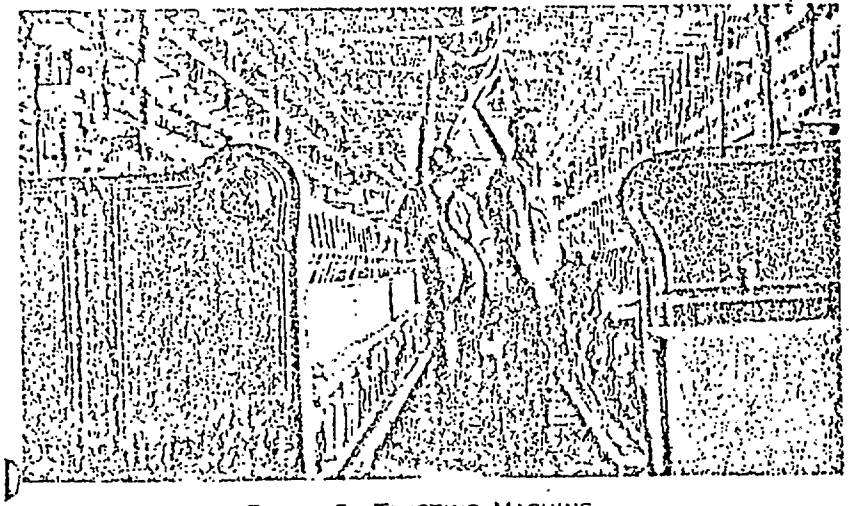


FIGURE 7.—TWISTING MACHINE.

with cotton twine. Following the initial twisting operation the yarn may return to the winders for placing on special tubes, in which form it is marketed. Then again, it may be conveyed to the cop or twill winders where it is wound into spindles for use in the shuttles of the looms. There are a variety of winding operations that prepare the yarn in some particular manner for the market or for use on certain types of machines. Likewise, there are a variety of twisting and spooling operations. (See fig. 7.)

The winding and twisting of the yarn is followed by a number of more or less related operations. The thread may be converted into cloth, tape, wick, brake lining, tubes, braids, or formed into rope. (See figs. 8 and 9.) A small amount of the product of the winders and twisters is treated by passing through a sizing compound, and subsequ[illegible] dried on reels before being wound again on tubes for the market.

WEAVING

The weaving of asbestos is done on looms that operate in the conventional manner. Both cloth looms and tape looms are used. (See figs. 10 and 11.) On the latter, brake lining, asbestos tape, and wick for oil stoves are woven. The tape looms differ from the cloth looms in that the warp is segregated into several strips of material and that there is a shuttle for each strip of warp. The weaving of all strips proceeds simultaneously; one mechanism operates all shuttles and a single reed suffices for all warps. Looms for tape weaving are made in multiples of 6 spaces up to as many as 30. Both dry and wet weaving are employed for the production of both cloth and tape. In wet weaving the warp is passed through a shallow pan of water and the cop (for use in the shuttle) is dipped into water.



FIGURE 8.—EXHAUSTED BRAIDER.

Dry weaving of broadcloth produces much lint and dust. The threads of the warp are continually rubbed past each other as they are moved from positions above or below the line of the shuttle which is shot from one side of the loom to the other at each change in position of the warp. Fiber fragments are rubbed from the threads at each change in their position and the fanning of the air by the moving machinery carries them into the atmosphere in and about the loom. The same machines are used for both wet and dry weaving.

In the manufacture of asbestos woven products at some of the plants studied, the warp is formed on individual looms by harnessing the thread from many spools which are hung on creel racks. At others

the warp is first wound on a beam from the individual spools and then transferred to the looms. The latter practice makes a more compact weaving unit as creel racks occupy several times as much floor space as the loom. The creel racks are attended by a worker who is kept

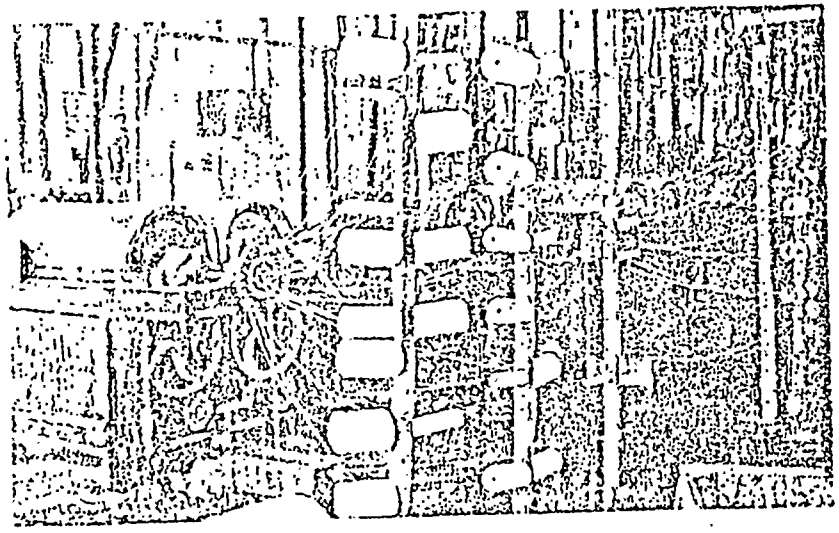


FIGURE 9.—ROPE-MAKING MACHINE.

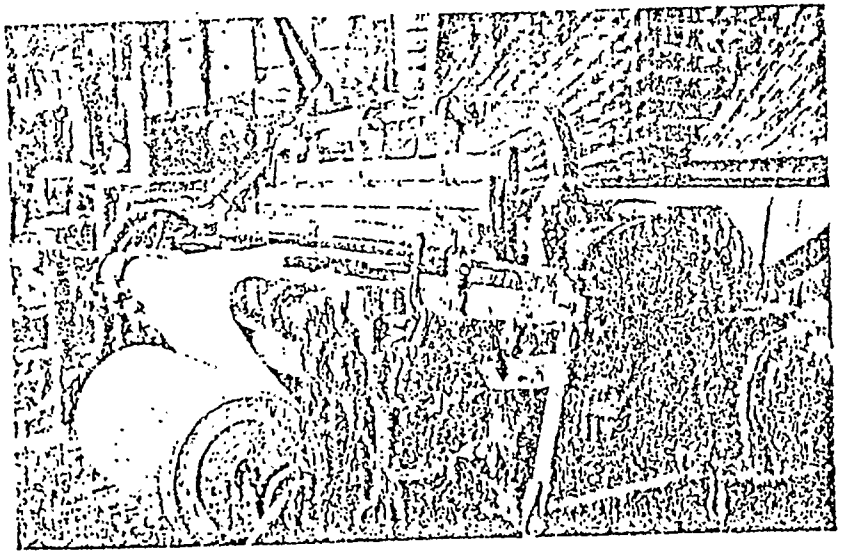


FIGURE 10.—BROADCLOTH WEAVING LOOM.

busy tying broken threads and replacing empty spools with full ones. The occupation involves an exposure to asbestos dust for which the looms are largely responsible.

The asbestos cloth is usually shipped to firms that convert it into gaskets, [illegible] of clothing, and other products that require a non-combustible fabric. Some asbestos wick is packed for the market without further treatment. Asbestos tape is usually sold to other industries. Some plants that weave brake bands sell it as a crude woven ribbon of fabric to finishing factories, while other manufacturers of the material convert it into finished products.

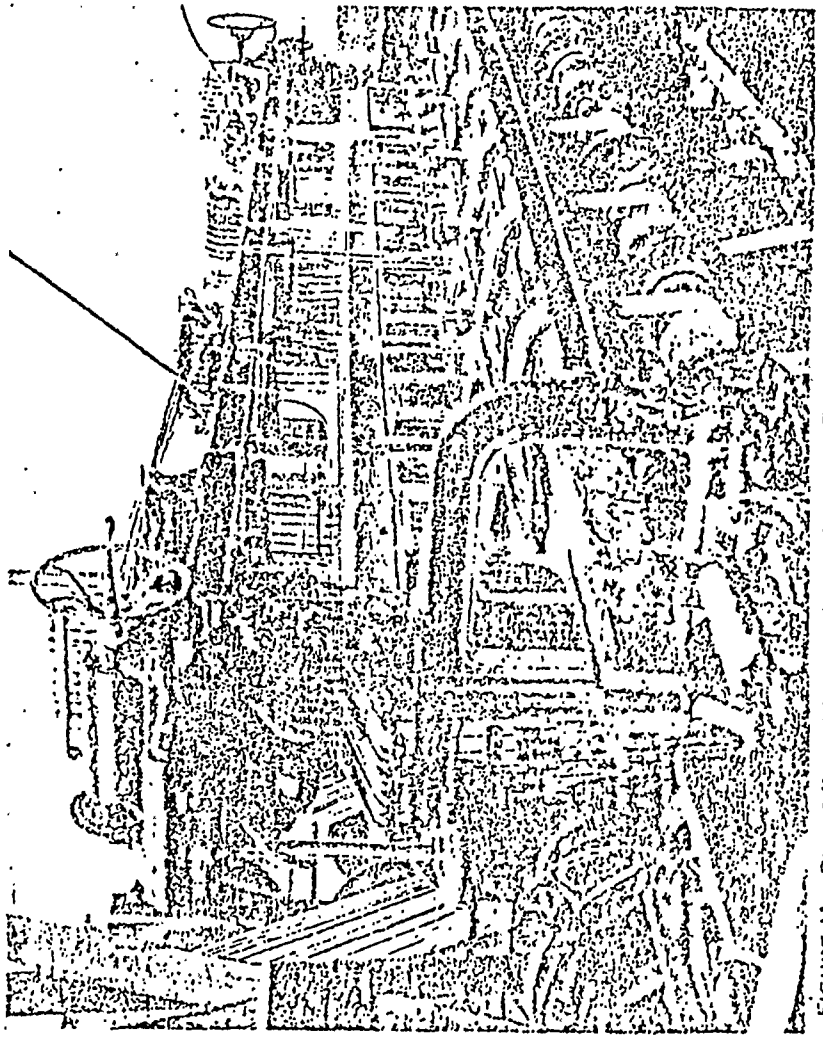


FIGURE 11.—BRAKE-BAND WEAVING LOOM SHOWING THREE OF THE SIX SECTIONS WITH WOVEN BANDS.

BRAKE-BAND MANUFACTURE

The manufacture of brake bands proceeds in several steps, the first being the impregnation of the woven fabric with either an asphaltic compound or a vegetable resin. The latter is the newer form of treatment and is rapidly supplanting the former. Following this operation a heat treatment is applied in a specially constructed oven. In practice the ribbon of woven asbestos passes first into [illegible] of the

treating substances and thence immediately into a baking oven. These operations are carried on in a building that is well ventilated and which is removed from other structures sufficiently to reduce the fire hazard. The treated asbestos fabric is now calendered into uniform width and thickness and one surface is ground smooth. The brake-band material is next cut into suitable lengths and holes drilled into them according to pattern. Finally, the finished brake bands are branded and packed for the market.

In some plants molded brake bands are made of asbestos yarn and

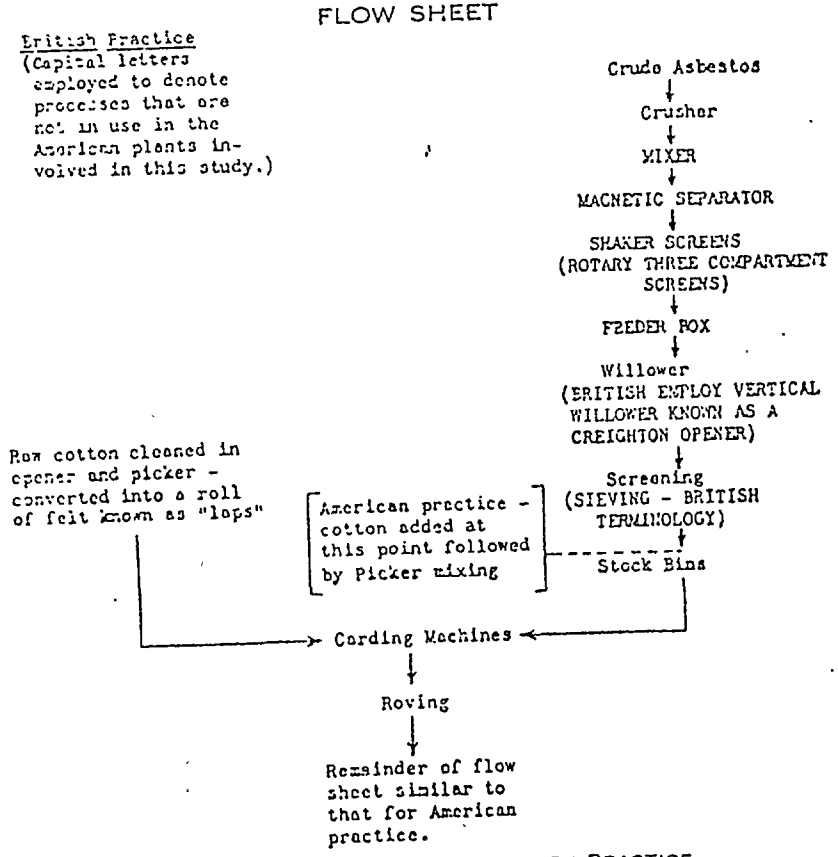


FIGURE 12.—FLOW SHEET OF BRITISH PRACTICE.

a synthetic resin. The process consists of passing asbestos thread through a bath of resinous material and immediately winding it upon a spool or cylinder. Enough of the resin will adhere to the asbestos to form a matrix into which the asbestos thread will be imbedded. When the desired thickness is obtained, the spool or cylinder is given a heat treatment which hardens the resin. The baked cylinder of material is removed from the spool and cut into narrow sections. The circular [illegible] thus formed are ground, machined, and otherwise made ready for the market.

The grinding or surfacing of the brake bands produces considerable dust. In the plants studied exhaust ventilation was used to control the exposure.

BRITISH MANUFACTURING METHODS

British practice differs in many respects from American practice. The same type of machinery is used in both countries but in British factories, dust-control equipment seems to be more generally used than in American factories. British factories specialize in the production of fine yarn and this involves numerous differences in practice.

In England the processing of the asbestos begins, as it does in the United States, with the crushing of the ore, but when this operation has been completed the manufacturing process does not parallel American practice until the fiber has been carded and is ready for spinning into yarn. (See fig. 12.)

After crushing, various grades of asbestos are blended in a machine quite similar to the conventional type of rotary concrete mixer. It is of passing interest to note that the same device is used for blending tea. The blend of asbestos is passed through a magnetic separator where certain mineral impurities are removed. It is cleaned further on rotary shaker screens where some grit and fiber fragments are eliminated. After screening, the material is fed into a willower. The British employ a vertical willower that is known as a Creighton opener. Its action is similar to that of the American willower, and the internal revolving mechanism is retained. The willowed material is subjected to a final cleaning on a second set of screens; such an operation is termed "sieving." Willowing is the last of the cleaning processes, after which the asbestos is placed into bins and is thereafter known as stock. It is now fully prepared for carding.

The practice at the plants included in this investigation is to add cotton to the asbestos and then subject the combination to a thorough mixing in a picker machine. In England cotton is added to the asbestos on the carding machine, both fibers being fed separately into the breaker card. The cotton in all probability is prepared for the carding operation on a conventional type of combination cotton textile picker machine which opens the fibers, cleans them, and forms the material into laps. The latter term is applied to the cotton which is formed into a broad ribbon of felt and then wound into a roll.

English carding machines are equipped with a feeding conveyor, or lattice, upon which first the asbestos and then the cotton are placed. The asbestos is added from a weighing box that is supported over and near the outside end of the conveyor. This box feeds asbestos in accordance with a predetermined ratio of the fiber to the cotton. When a certain weight of asbestos has accumulated in the box, a mechanism opens the bottom and allows the material to fall upon the

lattice. Further along a roll of cotton felt rests upon the lattice and unwinds as the conveyor moves forward. Thus, asbestos covered with a layer of cotton felt is fed into the first machine of the carding unit. The carding operation has already been described.

The roving produced by the carding machines may be converted into yarn on either one of the two machines, both of which have been discussed. As was pointed out, the ring spinning machine produces a much finer yarn than the mule spinning device. Since the British specialize in fine yarn, the ring spinning frames are used virtually to the exclusion of the mule spinner. The preparation of yarn for the market and its conversion into woven products will not be discussed since they are similar to the processes used in this country.

OCCUPATIONAL ANALYSIS AND A DESCRIPTION OF THE CHIEF OCCUPATIONS

The occupational analysis of the four asbestos plants covered in the present investigation is shown in table 1.

TABLE 1.—*Occupational analysis of workers in the asbestos textile manufacturing industry*

Section and activity	Number of workers in—								Total	
	Plant A		Plant B		Plant C		Plant D			
	M	F	M	F	M	F	M	F	M	F
Preparation:										
Foremen			2						2	
Crushermen	3		6						9	
Willowers	1		6						7	
Pickermen	3		6						9	
Total	7		20						27	
Carding:										
Foremen	2		3						5	
Second hand	3		3						6	
Carders	9		21						30	
Stockmen	3		15						18	
Tenders	3		3						6	
Total	20		45						65	
Spinning:										
Foremen			2						2	
Mule spinners	4		10						14	
Ring spinners	5		15						20	
Helpers	3		10						13	
Total	12		37						49	
Spooling: Spoolers		9		13						22
Twisting:										
Foremen	2		2						4	
Second hands	3		2						5	
Twisters	15		15				1		30	1
Helpers	3		4						7	
Total	23		23				1		46	1

TABLE 1.—*Occupational analysis of workers in the asbestos textile manufacturing industry*—Continued

Section and activity	Number of workers in—								Total	
	Plant A		Plant B		Plant C		Plant D			
	M	F	M	F	M	F	M	F	M	F
Winding:										
Foremen	1		2						3	
Winders		13		11		1				24
Yarn packers	8		5						13	
Total	9	13	7	11		1			16	25
Treating: Tenters	1		2	2	4		5		10	2
Cloth weaving:										
Foremen	1		1						2	
Second hands	2		1						3	
Weavers	6		25						31	
Inspectors	1		2						3	
Calenderers	3								3	
Creelers		4		5						9
Cop winders		2								2
Helpers			1						1	
Total	13	6	30	5					43	11
Tape and brake-band weaving:										
Foremen			1						1	
Weavers (tape)	6		18	4	2	1			26	5
Weavers (brake band)			2		2		9		13	
Calenderers				2	1				1	2
Creelers			7		1				8	
Clerk			1						1	
Total	6		29	6	6	1	9		50	7
Cord, rope, and braid workers			9	12					9	12
Finishing:										
Treaters and driers							3		3	
Calenderers					1				1	
Grinders					1				1	
Cutters and drillers					3		3		6	
Branders					1				1	
Coilers					1				1	
Total					7		6		13	
General labor:										
Engineers	1		2		1		3		7	
Laborers	6		6				2		14	
Watchmen	2		3				1		6	
Shipping clerks			1	1	3		2		6	1
Yardmen			2						2	
Total	9		14	1	4		8		35	1
Office:										
Superintendents	1		3		1		1		6	
Others	9		8	3	3	3	2		22	6
Total	10		11	3	4	3	3		28	6
Grand total	110	28	225	66	25	5	31	1	391	99

Reference to table 1 shows that a number of men have been listed as foremen and second hands. These terms have been adopted from the cotton textile industry. They do not always mean full-time supervisory duties, as might be presumed; in most cases foremen and second hands are engaged in all the activities of the department to which they are assigned. Further mention of these occupations is not made in the brief occupational descriptions which follow.

PREPARATION

Crushermen empty the raw asbestos, contained in 100-pound bags, into the pan of the crusher. They also operate the crusher and remove the asbestos fiber after crushing.

Willowers or openers handle crushed fiber and waste roving. The crushed ore is fed into the willowing machine by means of pitchforks. The willowed material is usually blown into a special bin. The waste accumulated by the machine is removed several times daily by the willower. Willowers also operate screens for separating coarse granular material (cobs) from the fiber.

Pickermen make up the asbestos-cotton batches (piling) according to specifications and pitch the material into the feeder hopper and the picker machine. The pickermen in some plants may hand truck to special bins the material which is blown from the machines.

CARDING

Carders operate the carding machines and clean them when necessary.

Stockmen handle the stock prepared by the pickermen. The material is conveyed by hand trucks from the stock bins to the card feeders.

Tenders look after the stock feeders and roving jack spools. The latter are removed when full, and replaced with empty jack spools. The occupations of tender and carder are in some plants interchangeable.

SPINNING

Mule spinners operate the mule spinning frames.

Ring spinners operate ring spinning machines.

Helpers (bobbin boys) assist the mule spinners and ring spinners conveying full jack spools of roving to the spinners and carrying empty spools back to the cards. They also transport the spun yarn and attend to the roving jacks and bobbins whenever required.

SPOOLING, TWISTING, WINDING, AND TREATING

Spoolers tend the spooling machines and handle spools on which the spun yarn is wound.

Twisters and helpers operate machines that twist spun material into heavier and stronger yarn.

Winders operate yarn winding machines.

Treaters pass yarn through special compounds (usually predominantly starch) and wind it on large reels for drying. In brake lining manufacture, treaters handle asphalt and resinous materials with which the product is impregnated.

Yarn packers wrap and box finished yarn.

CLOTH AND TAPE WEAVING

Broadcloth weavers operate both dry and wet weaving looms.

Tape weavers operate looms for weaving tape and brake lining materials.

Inspectors and calenderers inspect and examine all woven material for defects and pass tape and brake bands through calendering machines to achieve desired width and thickness.

Creelers attend to the creel racks on which the warp thread spools are mounted.

Cop winders operate cop-winding machines which wind thread on metal spindles for use in the loom shuttles.

We[illegible]ers usually attend to the drying of broadcloth which has been woven wet. [illegible]o handle material for the creelers and weavers.

ALL OTHER

Cord, rope, and wick workers operate special cord and rope-making machines and braiding machines.

Brake-lining finishers include treaters and dryer men, calenderers who operate calendering machines for squaring uneven edges, grinders who smooth off uneven surfaces, and cutters and drillers who cut the finished band to required sizes and drill it for riveting to brake shoes for automobiles and other machines. In the plants studied, these occupations were interchangeable.

General labor includes those occupations associated with maintenance of equipment and generation of steam. They also include janitors, watchmen, and shipping clerks.

Superintendence is concerned with the supervision of all plant activities.

RESULTS OF THE ENGINEERING STUDY

DUST STUDIES

In the present study, dust counts were made by the impinger method (2), using ethyl alcohol and distilled water mixtures containing from 25- to 50-percent alcohol as a collecting fluid. Two sampling methods were used for collecting dust for chemical analysis. (1) Settled dust samples were collected at the breathing level and (2) air-suspended dust was collected with a small, hand-type, vacuum cleaner fitted with a removable, paper-bag type filter. The latter method is similar to a procedure developed by Hatch (5). (See fig. 1.) In addition to chemical analyses, petrographic analyses were made on several samples. In order to determine the particle size of asbestos dust, an Owens jet apparatus was employed (2). The samples collected were measured by means of a filar micrometer at a magnification of approximately 1,000 diameters (oil immersion). Photomicrographs were also made of dust obtained by the vacuum-cleaner device described above.

NATURE OF THE DUST FOUND IN ASBESTOS TEXTILE FACTORIES

Composition.—Table 2 shows the results of chemical analysis of settled dust samples obtained by the Hatch method (5). Examination of these tables does not reveal significant differences among the various samples.[2] It will be noted, with one or two exceptions, that the total ash (no water of crystallization present) does not vary greatly. The value of 54.5 obtained for wet weaving (sample 8) is due to the moisture content of the dust sample. Calculations of composition on an ash basis bring the values into closer agreement than indicated in either table. The results given for sample 11 in table 2 are for brake lining which has been impregnated with resinous compounds.

[2] Canadian asbestos (crysotile), the asbestos used in these factories, has the following composition: Ash, 85.70; SiO_2, 40.49; R_2O_3, 1.27; MgO, 41.41; Fe_2O_3, 2.53 percent. (Asbestos: Vol. 3, No. 7, January 1922.)

TABLE 2.—*Chemical analyses of settled dust samples obtained in asbestos textile plants*

Department in which samples were collected	Average chemical composition of samples (percentage)						Remarks
	Ash	SiO_2	R_2O_3[1]	CaO	MgO	Fe_2O_3	
1. Preparation (plant A)	83.6	37.0	10.5	0.7	35.5	8.1	
2. Preparation (plant B)	87.5	35.5	17.1	.3	34.7	16.2	Near willower.
3. Carding (plant A)	85.2	36.1	13.5	1.5	31.0	10.3	Top of card hoods.
4. Carding (plant B)	82.8	36.3	11.5	1.2	31.7	6.9	Do.
5. Carding (plant B)	79.6	35.3	9.3	.6	31.5	6.8	Bottom of cards.
6. Mule spinning (plant B)	81.2	31.1	11.1	.9	32.3	11.2	
7. Weaving (plant A)	84.5	35.6	10.9	1.0	31.0	8.7	Tape weaving loom.
8. Weaving (plant B)	51.5	21.2	5.8	.2	21.5	4.8	Wet weaving.
9. Weaving (plant B)	75.3	34.0	11.0	.8	30.1	7.8	Between creel cracks.
10. Weaving (plant B)	72.8	32.0	10.9	1.0	28.5	7.7	On I-beam.
11. Cyclone grinding (plant C)	23.2	16.2	8.1	1.9	44.2	5.6	Garnet and metal separated with bromoform.
12. Dust from cyclone connected to exhaust system (plant B)	81.8	34.7	15.2	.6	31.2	10.6	Mostly card room dust.

[1] Combined oxides.

TABLE 3.—*Chemical analyses of suspended dust samples collected in an asbestos textile plant*

Department in which samples were collected	Average chemical composition of samples (percentage)						Remarks
	Ash	SiO_2	R_2O_3[1]	CaO	MgO	Fe_2O_3	
13. Preparation	89.8	35.4	9.3	1.2	31.9	6.8	General air sample.
14. Carding	83.7	37.5	9.5	1.0	36.0	6.3	Do.
15. Mule spinning	85.6	33.9	16.4	.9	34.3	7.1	Between 2 mule spinners.
16. Ring spinning	75.2	34.2	9.3	.8	31.6	7.0	Near ring spinning machines.
17. Dry weaving[2]	76.5	35.7	8.9	.1	31.3	7.0	Broadcloth weaving.
18. Wet weaving[2]	71.6	31.9	8.7	.9	30.4	6.7	Do.
19. Twisting[2]	75.9	32.7	10.4	.6	32.1	8.3	Near twisting machines.
20. Spooling[2]	81.2	34.7	12.0	.9	33.6	9.6	Near spooling machines.
21. Winding[2]	69.5	30.6	8.9	.7	29.6	6.6	Near winding machines.

[1] Combined oxides.
[2] Operations conducted in same room.

The losses on ignition of all samples analyzed are due to the presence of cotton fiber and moisture. The 30.5 percent loss on ignition of sample 21 in table 3 is probably due to the presence of cotton fibers.

In passing, it may be pointed out that no carbonate of the order reported by Hurlbut and Williams was found in any of the samples analyzed (6).

No quartz was found in any of the samples examined petrographically.

The consistency of the results indicated in tables 2 and 3 is more remarkable when the physical characteristics of the dusts are taken into consideration. Figure 13 illustrates the gross appearance of the dust samples taken with the vacuum cleaner device above mentioned. Each sample shown in the figure represents two grams of dust. There are conspicuous differences among the samples, both in color and bulk. Preparation and carding produce granular material. Weaving

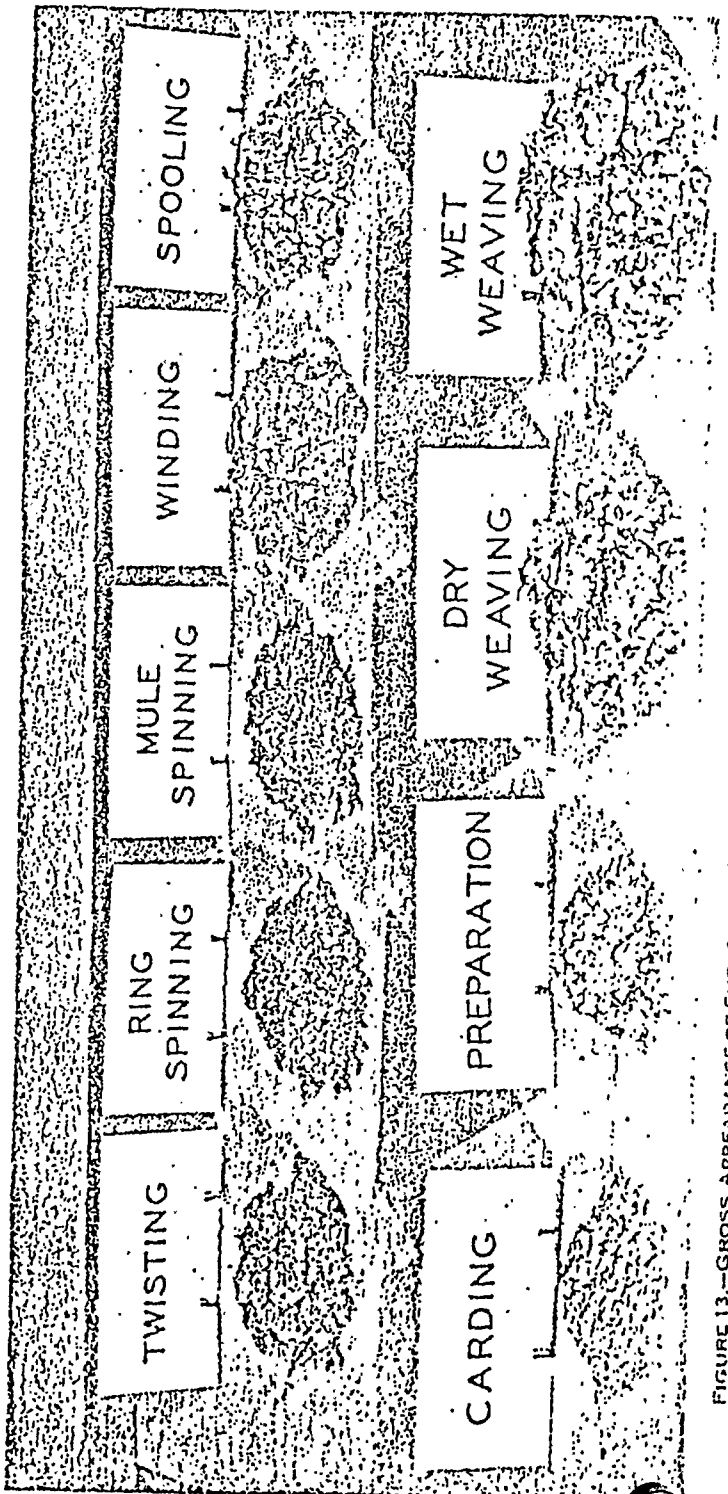


FIGURE 13.—GROSS APPEARANCE OF SUSPENDED DUST COLLECTED WITH VACUUM CLEANER DEVICE. (SEE FIGURE 1.)

samples, on the other hand, are bulky, the difference probably being due to the presence of long asbestos and cotton fibers in these samples. Carding and preparation samples contain cobs which are present in the raw asbestos. The darker appearance of the samples shown in the top row of figure 13 is difficult to explain. It is probably due to the

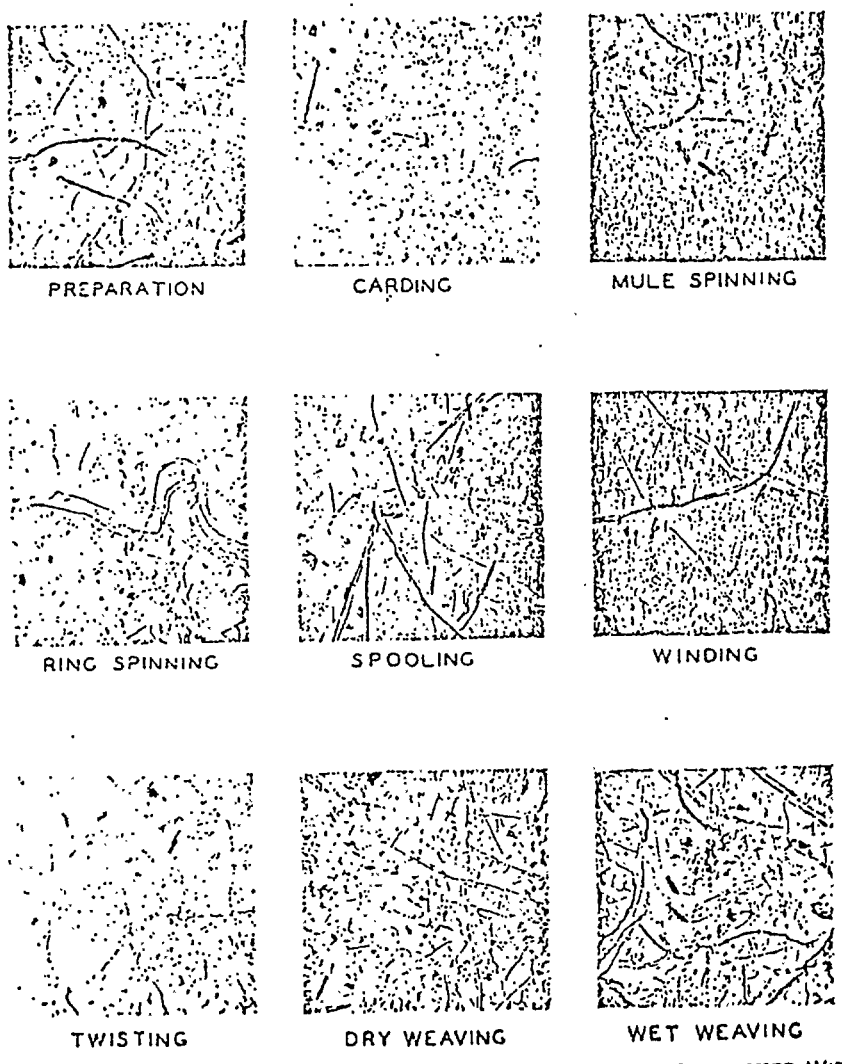


FIGURE 14.—MICROSCOPIC APPEARANCE OF SUSPENDED DUST COLLECTED WITH VACUUM CLEANER DEVICE. 200X.

contact of the spun asbestos-cotton mixture with oily metal surfaces.

The microscopic appearance of the vacuum cleaner samples is illustrated in figure 14. The presence of both cotton and asbestos fibers and of small particulate matter is readily noted. The dust from the [illegible] room is most striking. It has few fibers and it has consist[illegible] particulate matter.

Particle size.—The measurement of dust particles suspended in the air of asbestos textile plants is difficult because of the presence of both asbestos and cotton fibers. The differentiation of these fibers under the microscope is not always possible, especially when the fibers are short and fine. Several samples obtained with the Owens jet apparatus were examined with a petrographic microscope in order to estimate the proportion of asbestos and cotton fibers, but no satisfactory technique could be developed for this purpose. In this study, particle size determinations based on Owens jet samples were made by three methods: (1) eusope projection device; (2) a filar micrometer attachment to a microscope (using oil immersion, 1,000X), and (3) by means of photomicrographs.

Data obtained with the eusope showing the percentage of fibers (irrespective of whether they are cotton or asbestos) are presented in table 4. These are based on Owens samples and not on impinger samples. With regard to the selectivity of the device used, little information can be given, although it is known that for discrete particles, the selectivity of the Owens apparatus and the impinger are the same (2).

TABLE 4.—*Relation of fibers to particles in various Owens jet samples*

Activity	Number of Fibers counted	Number of Particles counted	Total count	Percent fibers
Weaving [1]	83	233	316	26
Weaving [2]	35	251	286	12
Picking [2]	21	279	300	7
Carding [1]	22	293	315	7
Twisting [1]	11	275	286	4
Twisting [1]	11	266	280	5
Crushing [2]	5	402	107	1

[1] Plant B.
[2] Plant A.

The results given in table 4 indicate that weavers are exposed to the highest percentage of fibers. This is to be expected because the warp threads fray as the shuttle crosses them. Samples collected near the pickers and carders have fewer fibers, while the dusts to which crushermen are exposed have the least proportion of fibers.

The relatively low percentage of fibers in suspended dust explains the small number of fibers observed while making dust counts of impinger samples. The latter were usually diluted to give 20 to 50 particles per quarter field. Hence, except for weaver's samples in which considerable fibers were observed, fibers are only rarely found in making counts of other samples. (Dust counts included both fibrous and nonfibrous particles, irrespective of size.)

Table 5 illustrates the size range of particulate matter. Two hundred particles in each sample have been measured by means of the

filar micrometer. As may be seen, the median size varies from 1.22 microns to twice this value. Fulton and his coworkers used an electrostatic precipitator to collect dust samples and calculated an average value for the total distribution of fibrous and nonfibrous of 1.33 microns (4). The median size for particles less than 5 microns, as deduced from data given by these investigators, is approximately 0.47 microns, which is much lower than the values given in table 5 of this report. This difference can probably be attributed largely to the type of sampling devices used in the two investigations.

TABLE 5.—*Size frequency distribution of particulate dust suspended in the air of asbestos textile plants*

Nature of process where sample was taken	Median size (in microns)	Geometric standard deviation	Percentage frequency of each particle size group (in microns)											
			0 to 0.49	0.5 to 0.99	1 to 1.49	1.5 to 1.99	2 to 2.49	2.5 to 2.99	3 to 3.49	3.5 to 3.99	4 to 4.49	4.5 to 4.99	5 or more	Total
Preparation	1.85	1.56	3	21	37	22	10	4	2	1	0	0	0	100
Carding	1.45	1.57	1	9	24	25	9	16	4	5	1	3	3	100
Mule spinning	1.80	1.34	0	1	25	38	28	4	1	1	1	0	1	100
Twisting	1.22	1.71	5	30	21	14	9	5	4	5	0	1	3	100
Weaving (broadcloth)	1.55	1.31	1	4	43	25	10	3	2	3	4	0	3	100
Weaving (tape)	2.40	1.64	0	3	11	21	11	12	7	13	7	3	8	100

In determining the length of fibers present in Owens samples, photomicrographs were used. It was necessary to use this method of measurement because of the curvature and matting of fibers in the microscopic field. This difficulty was overcome by using compass pointers and measuring each fiber visible across a given section of the photomicrograph. The size distribution of fibers, classified by occupation, is presented in table 6. The fibers range from as little as 2 microns (450× magnification) to as high as 400 microns. The median sizes, as may be seen by reference to the table, range from 7 to 16.3. Fulton's data indicates (assuming all particles greater than 5 microns to be fibers) a median size of 15 microns.

TABLE 6.—*Median length of fibers sampled with an Owens jet apparatus*

Activity	Median length of fibers in microns
Willowing	7.0
Picking	9.5
Carding	8.8
Twisting	12.6
Weaving (broadloom)	16.3

No size frequency distribution with regard to the width of fibers present in the air has been made in this study. However, it has been noted, [illegible] few exceptions, that the width of fibers collected by the

Owens jet apparatus was less than 0.5 micron. This is in agreement with the results given by Fulton, who shows more than 78 percent of the fibers measured by his projection method to be less than this size.

DUST CONCENTRATION IN DIFFERENT OCCUPATIONS

In earlier studies of the dusty trades carried on by the Public Health Service, determinations of the average dust concentration have been of great value (1) in describing the working environment, (2) in making comparisons of the effectiveness of different methods of dust control, and (3) in relating health conditions to dust exposure. In this study 242 impinger samples were taken and counted, and the results have been classified by occupation in tables 8 to 18. Each of the occupations carried on in asbestos textile factories has been discussed separately in the following section and data for the entire industry have been brought together in table 10. In this industry there are relatively few occupations in which workers do two or more kinds of work which expose them to totally different dust concentrations. In such cases the several exposures have been weighted according to the average time of exposure.

For example, in plant A, willowers are engaged in the following activities: willowing, 5 hours; picking, 1½ hours; storage-room work, 1½ hours. These activities are associated with exposures of 2.3, 36.3, and 16.4 millions of particles per cubic foot, respectively. Hence, we may tabulate exposures as follows and calculate a weighted average:

TABLE 7.—*Dust exposure of willower, plant A*

Activity	Time of exposure (hours per day) *a*	Average dust exposure (MPPCF) *b*	Millions of particle-hours per cubic foot *a*×*b*
Willowing	5	2.3	11.5
Picking	1½	36.3	54.4
Storage-room work	1½	16.4	24.6
Total	8		90.5

$$\frac{90.5 \text{ million particle-hours per cubic foot}}{8 \text{ hours}} = 11.3 \text{ million particles per cubic foot}$$

The average dust count of the 10 samples in the above table, calculated without regard to the duration of exposure, would be 18.7 million particles per cubic foot.

Preparation.—Preparation includes the occupations of crushermen, willowers, and pickermen. In plant B, these three occupations were conducted in the same room, while in plant A only crushers and willowers worked together. Picking (including piling), in the latter plant, was done in a separate room. The dust exposures are indicated in table 8.

TABLE 8.—*Dust exposure of workers in the preparation department*

Occupation	Number of samples taken	Dust concentration (MPPCF)
Crushermen	9	2.3–6.5
Willowers [1]	20	11.1–46.0
Pickermen [1]	19	31.3–71.3

[1] Weighted average.

It will be noted in table 8 that the crushermen's exposure is relatively low. In the plants studied, this work was carried on near an open door. However, it is doubtful that this occupation generates the quantity of dust shown in the above table. It is more than likely that willowing and picking (and even carding) contribute to the dust exposure of crushermen.

Willowing of crushed asbestos is not in itself productive of much dust (11.1 million particles per cubic foot). Only when waste roving and fly from cyclone collectors are willowed does the dust count increase. Dust counts as high as 99 million particles per cubic foot have been obtained with fly. A continuous source of dust production results from the manner in which willowing machines are fed. The material to be willowed is raised waist high by pitchfork, causing considerable spilling. Willowers also operate shaker screens.

A pickerman performs several kinds of work. This is especially true in plant B, where pickermen not only assist in piling (mixing of cotton and asbestos), but also aid in stacking the material blown from the picker machine into storage bins. This last-mentioned activity produces dust concentrations ranging as high as 211 million particles per cubic foot. Pickermen usually work in pairs and alternate in feeding the picker machine and tending to the storage bins. Piling is done by both workers together and contributes to dust counts averaging 16.2 million particles.

Picker machines, like willowers, are fed by pitchfork, a method which produces undesirable dust. This is true even where picker machines have hooded feeder boxes.

The picker machines studied were equipped with oil sprays, presumably to hold down dust. The ineffectiveness of this method of dust control is shown by the dust counts which exceed 50 million particles per cubic foot of air.

Carding.—The brushing and combing of asbestos and cotton fiber mixtures by carding machines creates considerable dust. The carding action is such that small cobs and fine tale adherent to the asbestos fiber are thrown into the air. This effect is accentuated by the fan action of the brushes. The granular and nonfibrous appearance of the dust present in the air of carding rooms has been shown in figure

The exposure of workers in carding rooms is given in table 9.

TABLE 9.—*Dust exposure of workers in carding department*

Occupation	Number of samples taken	Dust concentration (MPPCF)
Carders and tenders	38	29.1
Stockmen [1]	11	22.9

[1] Weighted average.

The average dust concentrations in both plants A and B were similar. Counts as low as 6.4 million particles per cubic foot were obtained for some carding operations where machines were operated at a relatively low speed and as high as 130 million particles for high-speed operations.

In the two plants studied which carded asbestos-cotton mixtures, all equipment was hooded and exhausted. Under the methods of operation, which required accessibility to the machines, complete enclosure was impossible. Consequently, exposed brushes, revolving at high speeds, often nullified the effect of the control system. Nor could air flows in the exhaust system be increased since a considerable amount of valuable fiber would be drawn from the brushes and this would reduce the weight of roving to an undesirable extent.

Spinning.—Both mule spinners and ring spinners are exposed to similar dust concentrations. The range shown in the following table (table 10) for each occupation is probably due to the presence of more spinning and weaving equipment where the dust concentration is higher.

TABLE 10.—*Dust exposure of workers in mule- and ring-spinning departments*

Occupation	Number of samples taken	Dust concentration (MPPCF)
Mule spinning	15	2.6–7.9
Ring spinning	13	3.2–8.3

Mule-spinning operations require considerable space and are generally separated from other activities. On the other hand, ring spinning is done in close proximity to spooling and twisting and it is possible that the dust counts are influenced by these operations. The results obtained for mule spinning check with those given by Fulton and his coworkers (4).

Spooling, twisting, and winding.—The equipment for these operations is generally grouped together and therefore it is difficult to obtain a clear-cut estimate of the amount of dust produced by each.

The average range of dust concentrations in plants A and B are shown in the accompanying table (table 11).

TABLE 11.—*Dust exposure of workers in spooling, twisting, and winding departments*

Occupation	Number of samples taken	Dust concentration (MPPCF)	Occupation	Number of samples taken	Dust concentration (MPPCF)
Spoolers	6	3.2–13.1	Roving winders	1	6.5
Twisters and helpers	20	3.2–13.2	Yarn packers	2	1.2
Universal winders	5	1.2–8.0	Yarn treaters	3	1.7–3.6

Of the occupations listed in the table, twisting appeared to contribute most of the dust in plant B and to influence the exposure of spoolers and ring spinners close by. Twisting frequently produced concentrations of over 20 million particles per cubic foot of air, yet counts below 10 million particles were not uncommon. Twisting involves a considerable amount of fraying and when low-grade materials are used, the dust production is increased.

Winders and yarn packers are exposed to lower dust concentrations than spoolers and twisters. Here again, however, because of the proximity of the latter dust-producing occupations, the amount of dust actually generated cannot be estimated. It is hard to conceive of such operations as roving winding and yarn packing producing much dust.

Broadcloth weaving.—The dust created during the weaving of broadcloth depends upon the number of warp threads per unit of width, the quality and width of the material woven, and on whether the operations are conducted dry or wet. If the warp threads are dry, the fraying action which takes place during weaving produces a considerable amount of dust. With some looms the dust concentration was found to exceed 140 million particles per cubic foot. Average dust counts obtained during weaving operations are shown in table 12.

TABLE 12.—*Dust exposure of workers associated with broadcloth weaving*

Occupation	Number of samples taken	Dust concentration (MPPCF)
Weavers and helpers:		
Dry	22	4.7–19.7
Wet	7	4.7–11.1
Creelers and helpers	4	3.6–9.8
Inspectors and calenderers	3	3.6–10.5
Cop winders	6	3.6–8.0

Weaving operations are intermittent owing to frequent breakage of warp threads. Consequently, workers are exposed to alternate periods of high and low dust concentration. On the other hand, weavers must pay close attention to work and are subjected to heavy exposures while the looms are in operation.

The dust counts shown in table 12 for weavers and helpers illustrate the differences between dry and wet weaving. Both dry and wet weaving are carried out on similar looms in close proximity. However, operators are interchangeable and are not specifically designated as dry or wet weavers. Under such conditions, it is impossible to gauge the actual exposure of a weaver. The low count of 4.7 million particles per cubic foot given in table 12 for both dry and wet weaving is for a small plant with one dry and one wet loom in operation side by side. The higher counts are for a larger plant manufacturing all types of broadcloth and at the time of the survey having from 8 to 12 looms in operation.

In view of the high counts obtained during broadcloth weaving, it would be expected that creelers are exposed to comparable concentrations. That this is not the case, however, is indicated in table 12. With all looms in operation, as is frequently the case, the counts might conceivably be higher. The same is also true of inspectors, calenderers, and cop winders who work in the weaving department. These occupations do not produce large amounts of dust.

Tape, wick, and brake-band weaving.—The dust exposures for tape, wick, and brake-band weavers are indicated in table 13.

TABLE 13.—*Dust exposure of workers associated with tape, wick, and brake-band weaving*

Occupation	Number of samples taken	Dust concentration (MPPCF)
Weavers (tape and wick)	12	2.4–5.1
Weavers (brake band)	14	4.7–20.3
Creelers	3	2.4–20.3
Calenderers	1	3.6

It may be seen from this table that tape, wick, and brake-lining weavers are exposed to lower dust concentrations than broadcloth weavers. Excepting certain types of brake bands, the fraying of the warp threads during weaving is not as great as in broadcloth weaving. However, with heavy brake bands, such as are used on trucks and hoisting machinery, the dust concentrations become high and comparable to those obtained in broadcloth weaving. It is in the manufacture of such brake bands that creelers are exposed to counts of 20.3 millions of particles per cubic foot of air. Calenderers, being at some distance from the looms, have a low exposure. The dust counts obtained for the type of weaving here discussed were consistent in all four plants studied.

Cord, rope, and braid working.—Other activities found in the asbestos textiles plants are concerned with the manufacture of special products, such as cord, rope, and braid, and in treating and finishing yarn. These operations produce little dust as may be seen by reference to table 14.

TABLE 14.—*Dust exposure of workers manufacturing and finishing special products*

Occupation	Number of samples taken	Dust concentration (MPPCF)
Cord, rope, and braid workers	8	1.2–10.2
Yarn treaters	3	1.7–3.6

The equipment used in manufacturing these products does not operate continuously. None of the operations of cord making, rope making, or braiding requires full-time personnel, and a small group of workers perform all these jobs. The highest dust count (10.2) in table 14 is for braiding. The counts obtained for yarn treaters are influenced by the operations undertaken nearby.

Brake-band finishing.—This generates little dust. In the plants studied, finishing operations were conducted in separate rooms or buildings. Dust counts are shown in table 15.

TABLE 15.—*Dust exposure of workers manufacturing finished brake-band lining*

Occupation	Number of samples taken	Dust concentration (MPPCF)	Occupation	Number of samples taken	Dust concentration (MPPCF)
Treaters and dryers	1	1.5	Cutters and drillers	1	1.8
Grinders	1	1.5	Branders	1	1.5
Finishers	1	1.2	Coilers	1	1.5

Workers employed in the occupations listed in this table are exposed to dust which differs in composition from the dust found elsewhere in asbestos textile plants. This is because the dust generated by grinders, cutters, and drillers of treated stock is made up of resin, asphaltum, asbestos, and metal. (See analytical results for sample 11, table 2.)

Miscellaneous occupations.—In addition to the groups directly concerned with the manufacture of textiles, a considerable number of workers (engineers, general laborers, clerks, and superintendents) are engaged in other activities. With the exception of certain foremen, these workers are exposed to low dust concentrations. The data presented in table 16 require no comment.

TABLE 16.—*Dust exposure of miscellaneous groups*

Occupation	Number of samples taken	Dust concentration (MPPCF)	Occupation	Number of samples taken	Dust concentration (MPPCF)
Engineers	2	1.2	Yardmen	2	0.10
Laborers	2	.36	Foremen (plant)	(1)	(1)
Watchmen	1	1.2	Superintendents (office)	2	.12
Shipping clerks	1	.12	Clerks	2	.12

[1] Samples and exposures depend on the activities with which they are associated.

PREVIOUS DUST EXPOSURE

The dust concentrations discussed in the preceding paragraphs represent present-day conditions. The officials of asbestos textile plants have in recent years attempted to control the dust hazard. In the plants studied, efforts have been made to exhaust picking, carding, and brake-band grinding machines. (Another plant, which will be discussed later, has extended the use of control equipment to nearly every dusty operation concerned with the manufacture of asbestos textiles.) A knowledge of the exposure of workers prior to the introduction of control equipment is important in correlating clinical findings with dust exposure.

In order to estimate past exposure, the air flow through various hoods attached to carding machines in plant A was shut off for 2 hours. Dust was allowed to accumulate for 1 hour prior to sampling. The dust thus generated caused much inconvenience to employees in the course of their work. For this reason, permission to shut off exhaust equipment was not granted in other plants studied. Carding machines are sensitive to slight air movements and are consequently difficult to regulate. Once they have been adjusted, any external disturbance affects the weight of the roving produced.

The dust counts obtained under conditions simulating those prior to the installation of the exhaust system are shown in table 17. For contrast, present exposures are included.

TABLE 17.—*Dust counts when control measures are used and when they are not in use*

Occupation	Average dust count—past (MPPCF)	Dust concentration—present (MPPCF)	Occupation	Average dust count—past (MPPCF)	Dust concentration—present (MPPCF)
Crushermen	50.2	2.3	Mule spinners	15.5	2.
Willowers	50.2	11.1	Ring spinners, twisters, spoolers, and winders	3.0	3.2
Pickermen	61.0	44.3	Weavers	7.1	1.7
Carders	72.3	20.1			

The low counts obtained for the occupations listed below carders is due to a partition which separates them from the carding machines.

Mule spinners are located immediately beyond this partition, and the dust count shown is due to dust which has passed through an open door.

Three dust samples obtained in the carding room 1 hour after the exhaust fans were shut off gave counts of 77, 73, and 78 million particles per cubic foot. At the end of 2 hours a sample taken in the same room gave a count of 61 million particles.

No major changes have been made in plants C and D since the start of operations. The dust counts made are essentially the same as those which have always existed. On the other hand, no data are available for past exposures in plant B. This failure can be attributed to several factors, but principally to the gradual expansion of the plant with the growth of the industry, which increased the amount of equipment, and the changes made in recent years with the exhaust system. The first of these factors appears to have affected all operations conducted in the plant, while the latter is restricted to the carding and preparation departments. In addition, it may be pointed out that plant expansion has tended to increase dust productivity, while the introduction of exhaust systems has reduced it. Hence, it may be assumed that the exposures of all occupations in plant B following carding were probably higher during the study than prior to expansion. It must also be remembered that more men have been working since the introduction of added equipment than at previous times.

INTERPRETATION OF DUST COUNTS

The average dust concentrations just discussed make a satisfactory measure of dust exposure for most purposes. In correlating medical findings with dust exposure, however, it is frequently advantageous to express the dust exposure in units which combine duration of exposure with average dust concentration. An easily calculated descriptive value was developed during statistical analysis of the results of an engineering and medical study of anthraco-silicosis (51). If a worker's dust exposure has been unchanged for all practical purposes during his period of employment the average dust count (in million particles per cubic foot) is multiplied by the duration of exposure in years. The product is used as a numerical measure of his total dust exposure. If a worker has changed his occupation (and in so doing has changed his dust exposure appreciably) a calculation such as the one shown in table 18 is made.

TABLE 18.—*Example showing the method of computing the dust exposure of asbestos textile workers* [1]

Occupation of worker	Number of years in occupation	Dust count (MPPCF)	Millions of particle-years per cubic foot of air
Carder [2]	3	72.3	216.9
Carder	2	29.1	58.2
Mule spinner	2	2.6	5.2
Total	7		280.3

[1] Plant A.
[2] Prior to installation of exhaust system.

$$\text{Weighted dust exposure} = \frac{280.3 \text{ millions of particle-years per cubic foot}}{7 \text{ years}} = 40 \text{ million particles per cubic foot}$$

The worker whose dust exposure has been calculated in table 18 would be assigned a value of 280 million particle-years in tabulations in which, for the sake of simplicity, it was desirable to have a one-term measure of dust exposure; table 42, for example. When average dust concentration and duration of exposure are to be tabulated separately, as in table 43, the individual described in table 18 would be assigned a dust concentration of 40 million particles per cubic foot.

THE CONTROL OF ASBESTOS DUST

The control of dust in industry often entails many practical difficulties. These difficulties pertain chiefly to the handicaps imposed by control equipment upon the performance of the work being done, and in estimating a priori the air volumes required. Both of these difficulties are inherent in the asbestos industry. The arrangement necessary for removing dust from carding machines and from looms, which demand accessibility to all parts, is self-evident. Added to this requirement is the limited information available with regard to suitable design of equipment for each dust-producing source and the requisite air volumes. The latter item is most important in the design of hoods for carding machines, inasmuch as air velocities must be kept relatively low in order to prevent the asbestos-cotton mixtures from being subjected to air currents which convey them to the exhaust system. At the same time, however, the velocities chosen must be adequate to maintain the dust below the safe or threshold limit.

The dust-control data contained in the following paragraphs are based on observations made in two plants included in the present study and on data obtained elsewhere by Assistant Public Health Engineer Richard Page (7). The data presented include operations for which control methods have been developed. In addition, the type of control equipment used in England is discussed, although no values pertaining to air requirements are available in this instance.

PREPARATION DEPARTMENT

The dust counts presented earlier in this bulletin for the preparation department apply to uncontrolled conditions. A high degree

of control can, however, be effected under certain conditions, especially as regards willowing (opening), piling operations, and picking. The crushing machines in the plants studied were not exhausted or segregated. In England, on the other hand, it is customary to enclose and segregate crushing equipment while it is in operation.

Willowing (opening).—Willowing machines are often productive of high dust concentrations (36 MPPCF). As has been previously mentioned, this dust is caused both by the manual methods used in feeding these machines and by the action of the machines themselves. Consequently, a reduction of dust concentration may be obtained by using a conveyor feeder which eliminates raising materials to waist

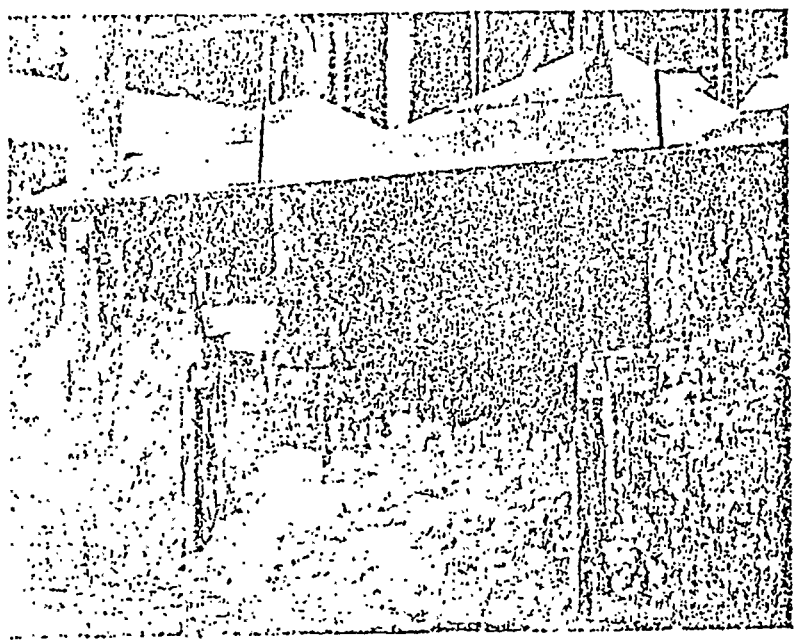


FIGURE 15.—EXHAUSTED PILING BINS.

levels with forks and using an exhaust above the feeder box and below the machine housing. The willowed material should, of course, be pneumatically conveyed to a suitable bin which is segregated from the preparation room.

In the case of two machines equipped with exhaust, it was found possible to reduce dust concentrations to 3.6 MPPCF. The exhaust hoods located above the feeders drew 400 CFM, while the downward exhaust below the willower housing drew amounts of air ranging from 225 CFM to 600 CFM, the latter air flow being used on a fly willower. (See fig. 4.) The air handled by the pneumatic conveyor was 1,800 CFM. For the 11-inch diameter ducts used, this corresponds to an air velocity of 2,730 feet per minute.

Piling.—Piling operations are usually conducted close to picking machines. The dust generated by this operation may be effectively controlled by using small exhausted compartments or bins. Compartments constructed as shown in figure 15 and handling approximately 1,025 CFM of air reduce the dust concentration from an average of 20 to 5.4 MPPCF.

English practice in piling consists in using exhaust manifolds close to the mixture being made (fig. 16). Air volumes are not available for the type of exhaust shown in the figure. The method appears to be cumbersome and to interfere with the process being carried on.

Picking.—The exhaust control used on picker machines is partly

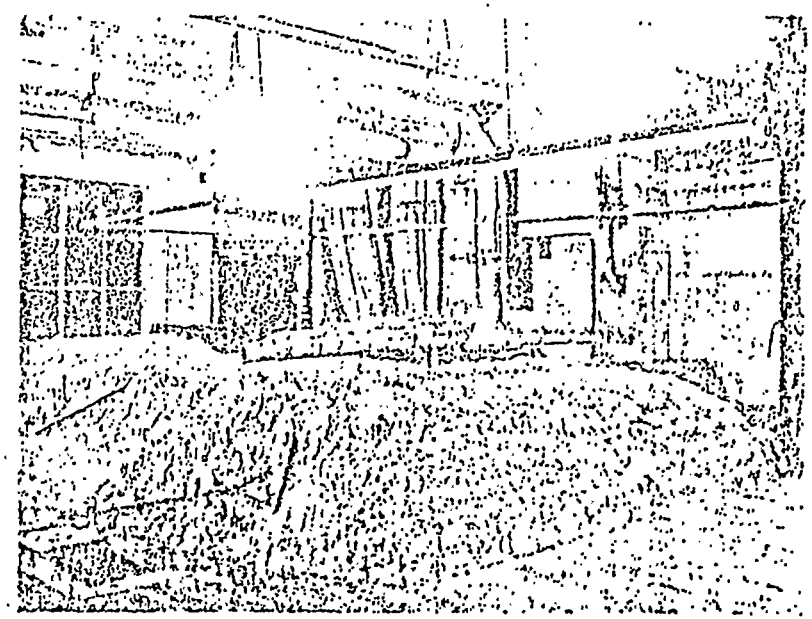


FIGURE 16.—EXHAUST VENTILATOR USED FOR PILING OPERATIONS (BRITISH PRACTICE).

shown in figure 17. One hood is connected at the feeder end and one at the discharge end. The center portion of the picker is also exhausted (not shown in figure). With this type of arrangement the dust concentration averages 6.7 MPPCF. This value is considerably lower than found among uncontrolled equipment which exceeds in many instances 74 MPPCF.

It was found that hoods above the feeder handled 500 CFM; the downward exhaust at the center measured 1,620 CFM; at the discharge end the air flow was 450 CFM. Pneumatic conveyors attached to the discharge end of some of the pickers handled 2,000 CFM. These ducts were approximately 12 inches in diameter, thus providing for a conveying velocity of 2,550 feet per minute.

It is possible with special conveyor belts operating between the asbestos-cotton piles and the feeder mixer that the dust concentrations might be reduced below the value of 6.4 MPPCF, indicated above.

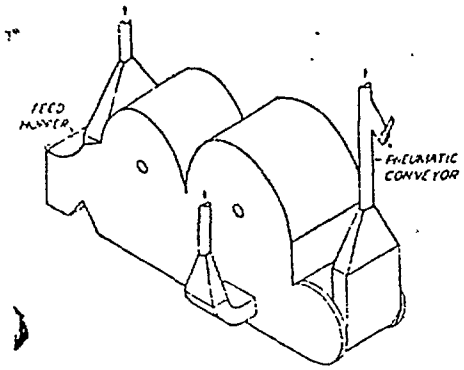


FIGURE 17.—SCHEMATIC DRAWING OF EXHAUST SYSTEM USED WITH PICKER MACHINE.

CARDING

Two types of dust-control equipment are at present used on carding machines. The first type consists of a hood which is located directly above the exposed brushes of the machine (fig. 18), while the second type is totally enclosed and exhausted at various points (fig. 5). Studies made of the first type of exhaust systems showed a reduction in dust concentration ranging from 72.3 MPPCF when no ventilation was applied to 20.1 with ventilation. The air flow for each hood averaged 870 CFM, which corresponded to six air changes per hour in the room in which the cards were located. It may be seen that the type of hood and the air flow used reduce the dust concentration to approximately one-third the amount created without ventilation.

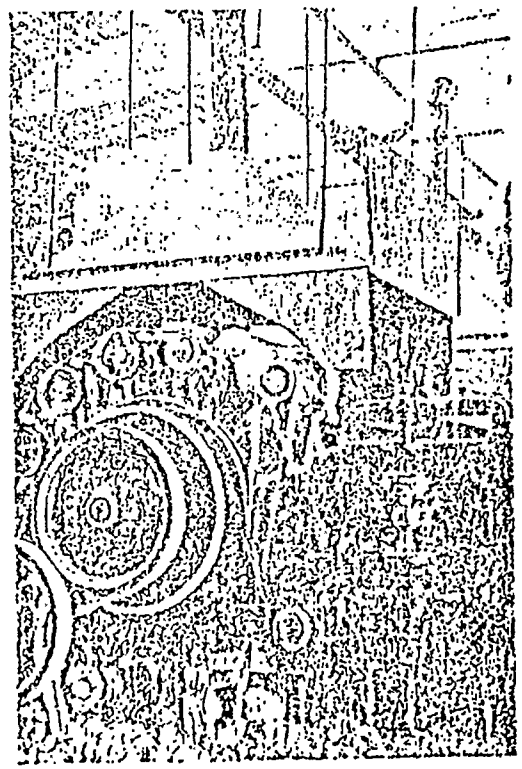


FIGURE 18.—VERTICAL EXHAUST VENTILATION APPLIED TO AN UNENCLOSED PRIMARY CARDING MACHINE.

The second type of exhaust system used proved more effective than the first in reducing dustiness. The dust concentration determined from numerous samples ranged from 1 to 3.9 million particles per cubic foot. This high degree of dust removal was accomplished with an arrangement similar to that indicated in figures 19 and 20. In the primary card[illegible] *a* and *b* discharge upward. The duct *c* exhausts from the bottom[illegible]e card. The secondary or roving card is hooded at *e* near the center of the machine. The openings *d*, *f* and *f'* are located at the bottom of the cards and exhaust the bottom fly which accumulates. The air volumes exhausted by each opening are indicated in the figure.

In addition to careful enclosure of the machine proper in the second type of system used, the feed box is carefully covered.

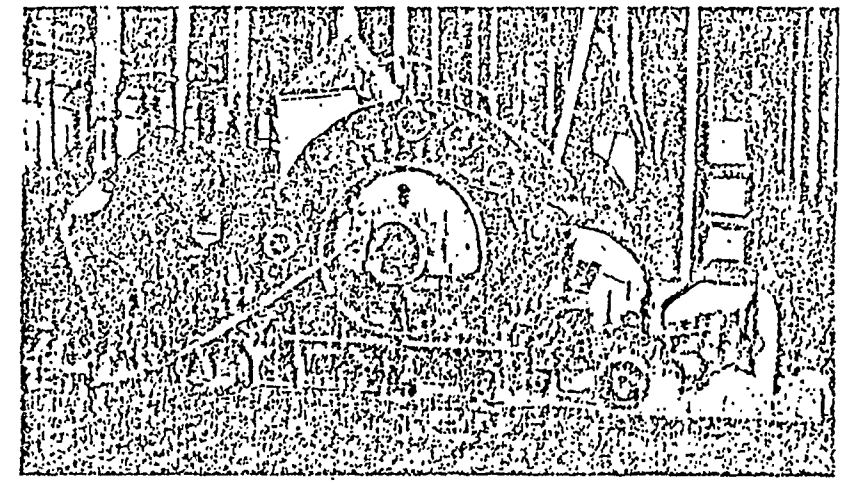


FIGURE 19.—TOTALLY ENCLOSED EXHAUSTED PRIMARY CARDING MACHINE.

The design of an exhaust system for carding machines is rendered exceedingly difficult owing to the ease with which carding material leaves the rolls. High air flows may seriously affect the quality of the roving produced. Likewise, a poorly constructed housing may create eddy currents which may prove bothersome. Any system developed requires care and experiment in design.

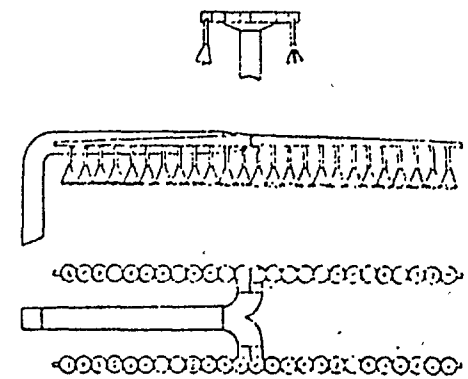


FIGURE 20.—ELEVATION AND PLAN FOR SPOOLING EXHAUST SYSTEM.

SPOOLING AND TWISTING

The dust generated by spooling operations may be ingeniously controlled by a system of cones in which the spools are inserted and exhausted. Such a system is shown in figure 21. Each case is constructed so as to offer a minimum amount of interference to the work being done. In the case of four Foster winders (200 spools) each cone is 9 inches in diameter across its widest part and 2 inches at the duct connection. The cones are each 10 inches deep. Two hundred cones were [illegible]o handle an

aggregate of 9,270 CFM or an average of 46.5 CFM per cone. The dust concentration averaged 2.9 MPPCF. Without the cone system discussed above, the dust concentration for the same operation was 13.1 MPPCF.

Twisting machines are more difficult to exhaust locally than spoolers. Figure 22 shows a system which has been partly completed. It consists of exhausting the lower section of the twisting machine by extending a duct with openings along the entire length of the equipment. Each system attached to the machine is intended to

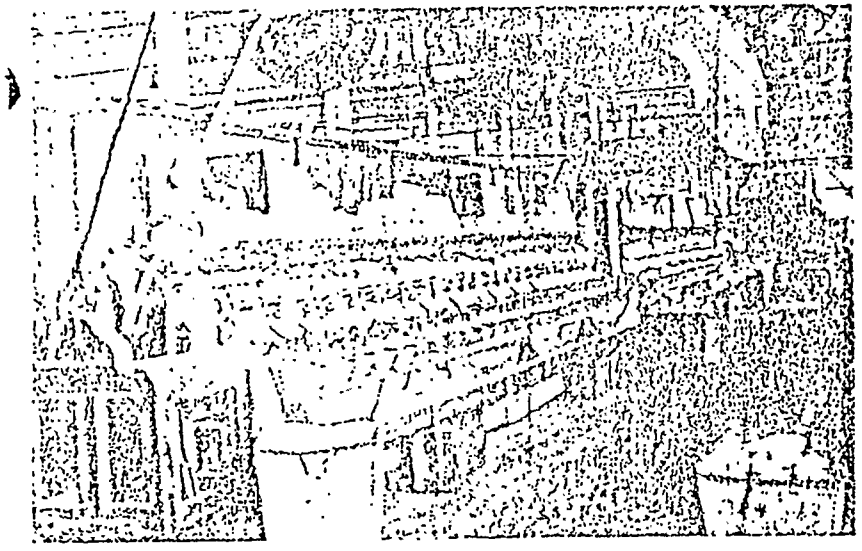


FIGURE 21.—EXHAUSTED SPOOLING FRAME.

exhaust 1,670 CFM. Dust determinations to establish the effectiveness of this system could not be made owing to the fact that only one of 10 twisting machines was equipped with exhaust and the counts obtained were affected by unexhausted machines operating close by.

WEAVING

Any system developed for removing dust generated by broad looms represents the accomplishment of one of the most difficult operations encountered in the design of exhaust equipment. Nevertheless, this has been done effectively both in this country and in England. The American system shown schematically in figures 23 and 24 consists of a downward-draft hood fitting close to the shuttle rack of the loom and a movable upper exhaust hood which operates on the surface of the woven material. The latter hood is actuated to and fro by the action of the loom lay to which it is attached. The hood is connected to the main exhaust duct by means of a flexible air tight joint. Both the downward and upward exhaust hoods are narrow and possess the advantage of high opening velocities. The upward-draft hood connected to the loom lay is segmented. Each hood used, as indicated in figure 24, handles 1,300 cubic feet of air per minute and reduces the dust concentration to an average of 0.7 MPPCF. The system is thus extremely effective, since on uncontrolled looms the concentration averages 49.7 and may exceed 140 MPPCF.

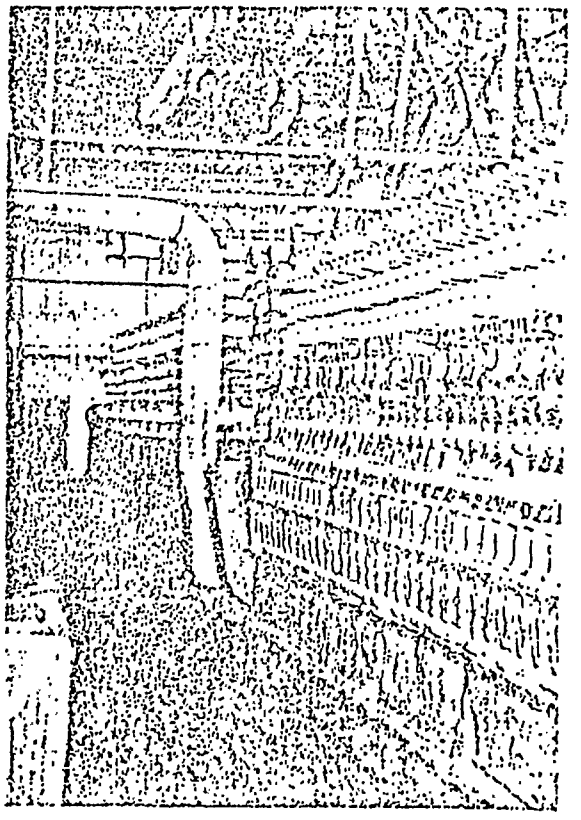


FIGURE 22.—EXHAUSTED TWISTING FRAME.

The English method for exhausting looms differs considerably from American practice. In the former system all parts are stationary (fig. 25). The looms are exhausted upward by hoods located in front of the operator and resting a short distance away from the lay. The hood consists of two parts which may be turned back whenever repairs to the loom are necessary. No data as regards air volumes used in this type of equipment or its effectiveness in reducing dust are available.

INSPECTION

In some plants, asbestos cloths are brushed during inspection. The usual procedure consists in brushing the cloth from above and below as it passes over the inspection table. An inspection table of this type with exhaust hoods is shown in figure 26. The hoods used are located near the cloth brushes, and extends the full width of the cloth examined. They are, however, only one-half inch in width.

A study made of an inspection table such as discussed above indicated that two hoods located on either side of the cloth handled approximately 730 CFM each. A third brush and hood located near

the jack spool at the end of the inspection table handled 192 CFM. These hoods reduced the dust generated from a value of 11.1 when no ventilation was applied to a value of 0.5 MPPCF with ventilation.

SUMMARY OF THE STUDY OF THE WORKING ENVIRONMENT

The engineering data contained in this report are based chiefly on a study of four asbestos plants. Data on dust control methods



FIGURE 23.—EXHAUSTED BROADCLOTH LOOM.

have been secured in the course of a separate study undertaken in a fifth plant. Two of the four plants investigated were engaged in the manufacture of asbestos textiles directly from raw crysotile (Canadian) asbestos received in 100-pound bags. The remaining two plants began operations with yarn received from other sources and were [illegible]gned only with the manufacture of asbestos, tape, braid, or brai[illegible]ding and with their treatment.

Dust concentrations were determined by means of the impinger technique using light field counting. In all, 242 impinger samples were taken so as to evaluate the environment of each occupation.

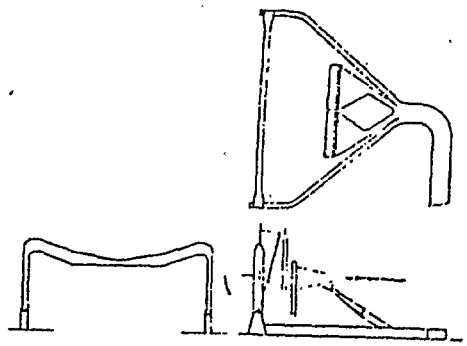


FIGURE 24.—DETAIL OF EXHAUSTED LOOM SHOWN IN FIGURE 23.

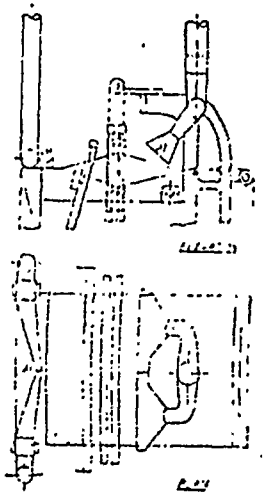


FIGURE 25.—DETAIL OF EXHAUST USED ON BRITISH TYPE LOOM.

The number of workers employed, the number of dust samples taken, and the dust concentrations determined for each occupation are presented in table 18. The concentrations range from values of 0.86

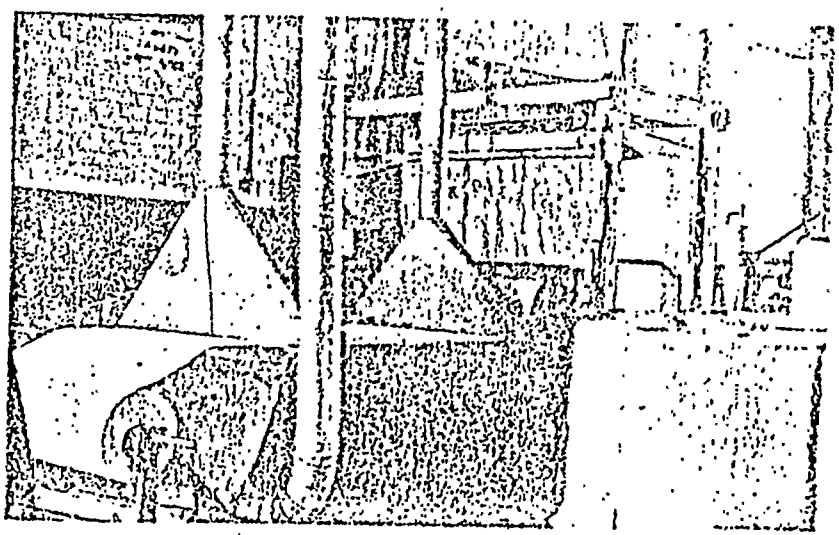


FIGURE 26.—EXHAUSTED BRUSHING AND INSPECTION TABLE.

for shippers to as high as 74.3 MPPCF for pickermen. Occasional samples exceeding 100 MPPCF were found for the occupations of carding and weaving.

TABLE 19.—*Occupational dust exposure of workers in four asbestos textile plants*

Activity and occupation	Number of workers	Number of samples taken	Dust concentration (MPPCF)
Preparation:			
Crushermen[1]	9	9	2.3–6.5
Willowers[2]	7	8	[3] 11.1–[3] 30.0
Pickermen[2]	9	21	34.3–71.3
Carding:			
Carders and tenders	42	38	29.1
Strippers	18	11	22.9
Spinning:			
Mule spinners and helpers	21	15	2.6–7.9
Ring spinners and helpers	26	13	3.2–8.3
Spooling: Spoolers	22	6	3.2–13.1
Twisting: Twisters and helpers	43	20	3.2–13.2
Winding:			
Cheese winders	26	5	1.2–6.0
Roving winders	2	1	6.5
Yarn packers	13	2	1.2
Yarn testers	12	3	1.7–3.6
Cloth weaving:			
Weavers and helpers:			
Dry	31	22	4.7–49.7
Wet		7	4.7–11.1
Inspectors and cylinderers	6	3	3.6–10.5
Creelers and helpers	10	4	3.6–9.8
Cop winders	2	6	3.6–8.0
Tape, wick, and brake-lined weaving:			
Weavers (tape and wick)	31	12	2.1–5.1
Weavers (brake-lined)	14	11	4.7–23.0
Creelers	8	3	2.4–20.3
Carnetters	3	1	3.6
Cord, rope, and braid workers	21	8	1.2–10.2
Brake-lined division:			
Truckers and driers	3	[4]	
Calenderers	1	1	1.5
Grinders	1	1	4.2
Cutters and drillers	6	1	1.8
Buffers	1	[4]	1.5
Cutters	1	[4]	1.5
General labor:			
Firemen	7	2	1.2
Laborers (shippers, etc.)	14	2	.85
Watchmen	6	1	1.2
Shipping clerks	7	1	.12
Yardmen	2	[4]	.10
Office:			
Superintendents (office)	6	2	.12
Foremen (plant)	19	----------	[5]
Clerks	29	[4]	.12
Total	481	243	----------

[1] Exposure weighted to take into account piling, breaking, and storage bin activities.
[2] Includes exposure due to operation of shaker screen.
[3] Average includes other samples.
[4] Dust counts obtained from samples of other occupations having similar dust exposure.
[5] Exposures according to associated activities.

The chemical and petrographic analyses of dust samples were determined from both settled dust samples and samples obtained directly from the air by means of a vacuum cleaner. No great differences in composition were noted between corresponding samples collected by these two methods. Total ash was approximately 77 percent for all the samples. No free silica (quartz) could be found on petrographic analysis.

Particle size determinations of Owens jet samples were made by three methods, namely, (1) with a euscope projection device (for determining the ratio of fiber to particulate matter), (2) by means of a filar micrometer attachment to a microscope (for measuring the size particulate matter), and (3) with photomicrographs (for measur the length of fibers). This study showed that the ratio of fibers particulate matter varied considerably from a maximum of 26 p cent for weavers to a minimum of 1 percent for crushermen. T median particle size (exclusive of fibers) was approximately microns. The median fiber length varied from 7 to 16.3 microns. distinction could be made between cotton and asbestos fibers.

In most of the plants studied the control of the dust hazard limited almost entirely to provisions of exhaust systems for the card machines. Data, however, are available to indicate the effectiven with which the dust problem can be handled in an asbestos tex plant. An effective method employs the use of hoods and exha equipment fitted at each dust-producing source. Data showing effectiveness of exhaust equipment is presented in table 20.

TABLE 20.—*Summary of results showing the exposure of asbestos textile work under controlled and uncontrolled working conditions*

Equipment	Number of duct connections	Volume of air handled (CFM)	Dust concentration with exhaust (MPPCF)	Dust c gene wi lea
Willowing (opening)	[1] 2	625–1,000	3.6	11.1
Piling[2]	1	1,025	2.0	
Picking	3	2,350	6.7	31.4
Carding (primary)	3	1,120	2.0	
Carding (roving)	4	1,410		
Spooling	[3] 1	46.5	2.9	
Weaving (broadloom)	2	1,360	.7	4.7
Brusher calenderer	3	1,650	1.0	

[1] Equipped with pneumatic conveyor.
[2] Exhausted bin or compartment.
[3] Individual cone for each spool and connected to exhaust manifold.

It is evident from the above table that the dust hazard in asbestos industry can be greatly reduced. It is further clear th the necessary control methods have been developed and are now use. With the exception of pickers who are exposed to concentratio of 6.7 million particles per cubic foot under controlled conditio exposure in all other occupations can be kept below 5 million partic per cubic foot. It is possible with improved methods of ventilati to reduce even the picker's exposure to less than the figure indicate The following recommendations are made:

1. Dust should be controlled at the point of origin by means local exhaust hoods so designed and operated that no dust is permitt to reach the breathing zone of workers or to contaminate the gene air. Dust-removal devices, which are capable of reducing the du concentration to a level of 5 million particles per cubic foot or low have been developed for all operations in the asbestos textile indust (7).

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 are 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..... Racial Stock.....M. S. W. D. Sep.

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

Age began work..... Years worked..... Time Idle.....

OCCUPATIONAL HISTORY

SPECIFIC JOB	INDUSTRY	YEARS		WHERE	WORKING CONDITIONS CONTROL MEASURES ETC.
		DUSTY	NON-DUSTY		
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	348	118	85	541
Under 20 years	14	12	3	29
20 to 24 years	23	26	17	66
25 to 29 years	55	28	25	108
30 to 34 years	51	16	12	79
35 to 39 years	34	20	13	67
40 to 44 years	23	7	9	39
45 to 49 years	14	4	3	21
50 to 54 years	7	4	3	14
55 to 59 years	10	4	1	15
60 to 64 years	2	0	0	2
65 to 69 years	1	0	0	1
70 years and over	0	0	0	0
Unknown age	1	1	0	2
Average	42.08	30.36	32.41	
Standard deviation	9.59	10.39	8.90	

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		Glass	35.3	162
Iron and steel	42.0	202	Gas	31.5	162
Granite cutting	39.0	187	Cigar	31.2	162
Anthracite mines	39.5	221	Cement	34.9	156
Fur cutting	39.2	231	Rubber	34.1	156
Pottery	39.1	176	Chemical	33.1	162
Post office	37.8	162	Garment	32.8	71
Foundry	37.8	162			

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 employed 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	541	73	260	112	47	16	2	1
White males	321	41	161	72	28	13	2	1
White females	111	19	59	19	13	1	0	0
Negro males	79	10	40	21	6	2	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.

FAMILY AND PAST MEDICAL HISTORY (INSERT DATE WHEN POSITIVE)

Family history of T. B._____

Exposure to T. B._____

Tuberculosis_____ Treatment_____ Pleurisy_____ Dry_____ Wet_____ Side_____

Hemoptysis_____ Dates_____ Amount_____ Asthma_____ Hay Fever_____

Pneumonia_____ Empyema_____ Bronchitis_____ Sinus Diseases_____

Influenza_____ In Bed_____ More colds in recent years_____

Average number of colds yearly_____ Head _____ Chest _____ Duration_____ Severity_____

Tonsillitis _____ Previous injuries _____

Operations _____ Heart Disease _____

Rheumatic Fever_____ Scarlet Fever_____ Hypertension_____

G. I. _____ Skin diseases _____

G. U. _____ Nervous system _____

Menstrual _____ Pregnancies _____

Other serious illnesses or complaints_____

_____

PRESENT HEALTH

Cough _____ How long_____ Type_____ When worse_____

Productive_____ Amount_____ Character_____ Odor_____

Dyspnea_____ Duration_____ Severity_____ Progressing_____

Appetite_____ Digestion_____ Bowels_____ Edema_____

Strength and energy_____ Hernia_____

Weight: Stationary_____ Lost_____ Gained _____ How long_____

Other complaints _____

_____

Progress of symptoms_____

_____

_____

HABITS

Alcohol _____ Sleeping _____

Tobacco _____ Eating _____

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 21.—*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 bold-face 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	26.34	94	**36.4**	30	*10.2*	25	*10.8*	11	*5.0*	34	*11.1*	4	*1.6*
Cough	118	25.81	.....	.....	32	*11.6*	22	*12.2*	13	*5.7*	28	*12.5*	3	*1.8*
Hemoptysis	39	8.42	.....	.....	.....	.....	9	*3.4*	3	*1.6*	11	*4.6*	1	*0.5*
Loss of weight	41	8.86	.....	.....	.....	.....	.....	.....	4	*1.7*	10	*4.7*	2	*0.5*
Chest pain	19	4.10	.....	.....	.....	.....	.....	.....	.....	.....	9	*1.7*	2	*0.2*
Weakness	42	9.07	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	4	*0.6*
Loss of appetite	6	1.30	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....

As table 21 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.[3] 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 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.[4]

[3] This value has been calculated on the assumption that 0.2634 represents the probability that any one of the 447 persons would have dyspnoea and 0.2981 represents the probability that a person would have a cough. These values are from the second column of figures of table 21. Then, 0.2634×0.2981×463=36.35.

[4] An example (table 18) has been given in the section entitled "Interpretation of Counts."

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

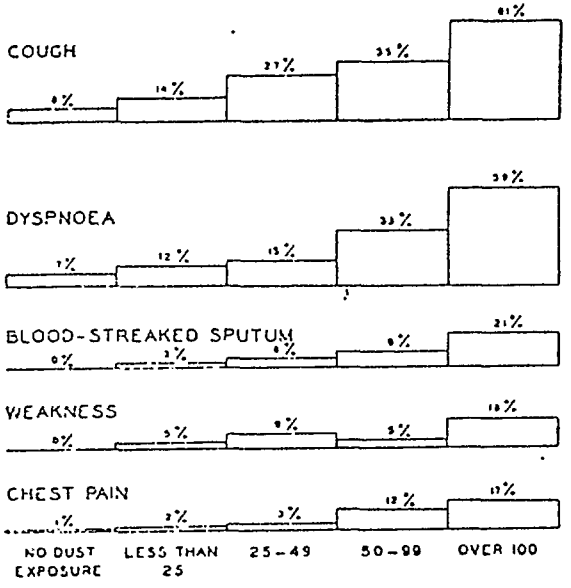


FIGURE 29.—PERCENTAGE OF PERSONS, CLASSIFIED BY DUST EXPOSURE, WHO HAD CERTAIN DISORDERS.

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								Total
			Less than 25		25 to 49		50 to 99		Over 100		
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	
Total	74	100	198	100	85	100	55	100	108	100	519
Cough	6	8	28	14	23	27	19	35	64	61	140
Dyspnoea	5	7	24	12	13	15	18	33	62	59	122
Weakness	0	0	10	5	8	9	3	5	19	18	40
Loss of weight	4	5	14	7	8	9	2	4	1 18	16	44
Blood-streaked sputum	0	0	6	3	5	6	5	9	22	21	38
Chest pain	1	1	11	2	12	3	16	12	16	17	19
Loss of appetite	0	0	0	0	------	2	2	1	2	3	3

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-

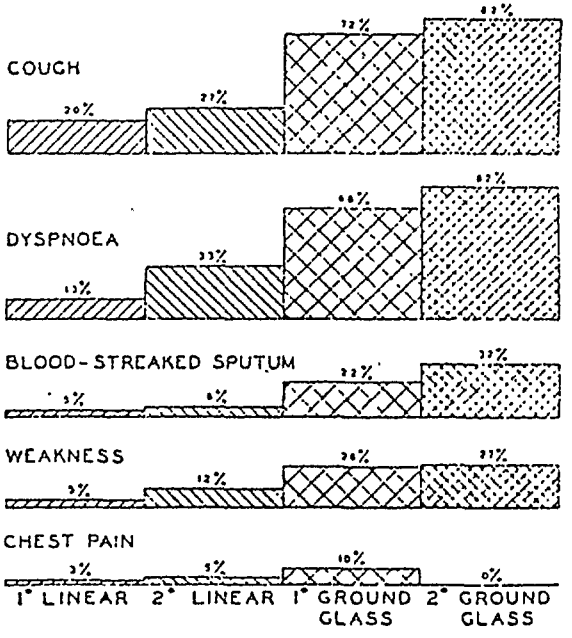


FIGURE 30.—PERCENTAGE OF PERSONS, CLASSIFIED BY LUNG-FIELD MARKINGS, WHO HAD CERTAIN DISORDERS.

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	50	100	22	100	462
Cough	62	20	21	27	36	72	14	64	133
Dyspnea	42	13	26	33	31	62	18	82	120
Weakness	15	5	9	12	13	27	6	27	43
Loss of weight	24	7	9	12	2	4	6	27	40
Coughs tracheal sputum	17	5	5	6	11	22	7	32	40
Chest pain	10	3	1	5	5	10	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

FLUOROSCOPIC EXAMINATIONS

Bony framework .....

Heart, Aorta and Trachea .....

Diaphragm .....

Lung fields .....

.....

Examined by: .....

X-RAY INTERPRETATION

Film No. ..... Technic ..... chest cm.

Quality of film ..... Bony framework .....

Heart, Aorta and Trachea .....

Diaphragm .....

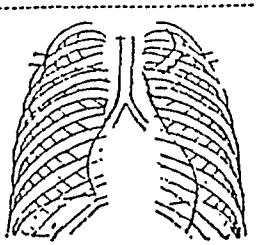
Lung fields .....

.....

.....

.....

.....



Examined by: .....

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 (25), 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

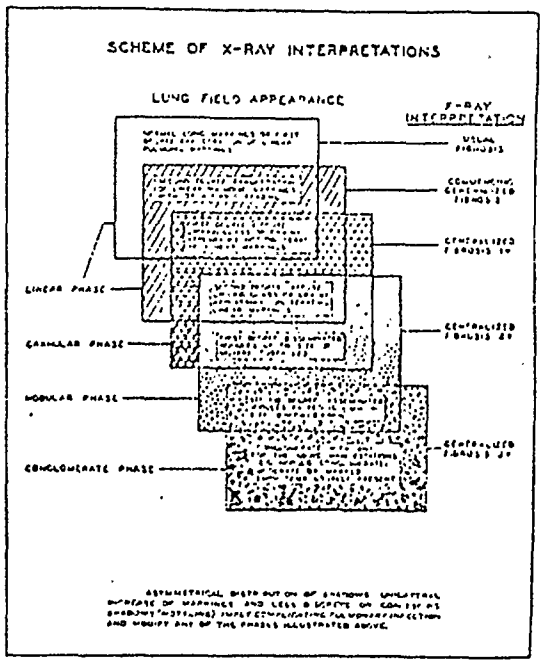


FIGURE 32.—SCHEME USED IN CLASSIFYING X-RAY MARKINGS

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 (25) 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,[5] do not regularly show the basal localization of fibrosis that is so characteristic of asbestosis.

[5] Unpublished data.

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

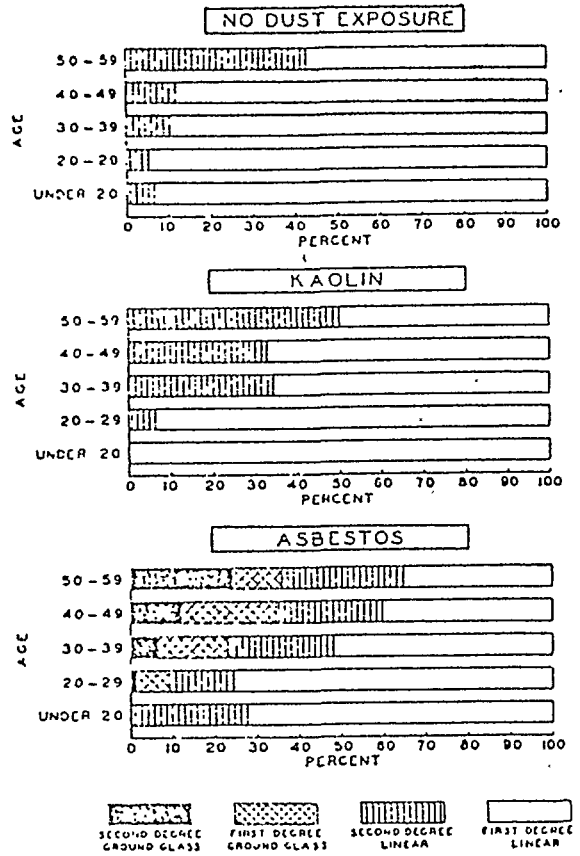


FIGURE 33.—PERCENTAGE OF MALES, CLASSIFIED BY AGE, WHO HAD CERTAIN LUNG-FIELD MARKINGS. 229 MEN HAD NO PREVIOUS INDUSTRIAL DUST EXPOSURE, 80 HAD BEEN EXPOSED TO KAOLIN DUST, AND 357 HAD BEEN EXPOSED TO ASBESTOS DUST.

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 in 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 (43*a*) 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 first-degree ground-glass markings	Percent with second-degree ground-glass markings
Total examined...	229	92	8	80	70	21	359	61	20	13	6
Under 20.....	58	93	7	7	100	0	11	73	27	0	0
20 to 29.....	107	94	6	29	93	7	109	76	14	8	1
30 to 39.....	38	90	10	24	65	35	112	52	21	17	6
40 to 49.....	17	88	12	12	66	33	48	40	25	23	12
50 to 59.....	7	57	43	6	50	50	17	35	29	12	24
60 to 69.....	2	100	0	3	100	0	2	50	0	0	50

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.

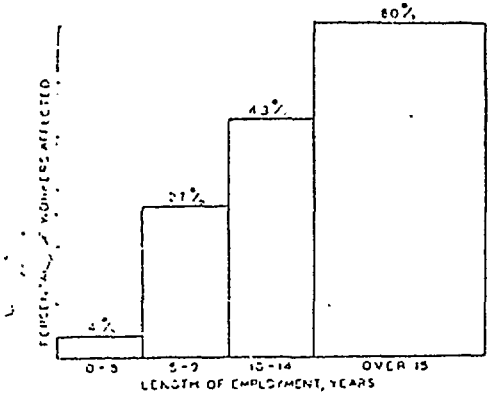


FIGURE 34.—RELATION OF GROUND-GLASS LUNG-FIELD MARKINGS TO LENGTH OF EMPLOYMENT.

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.

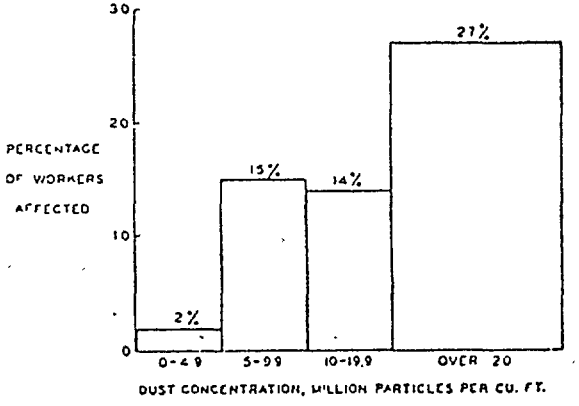


FIGURE 35.—RELATION OF GROUND-GLASS LUNG-FIELD MARKINGS TO DUST EXPOSURE.

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 p[illegible] 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.

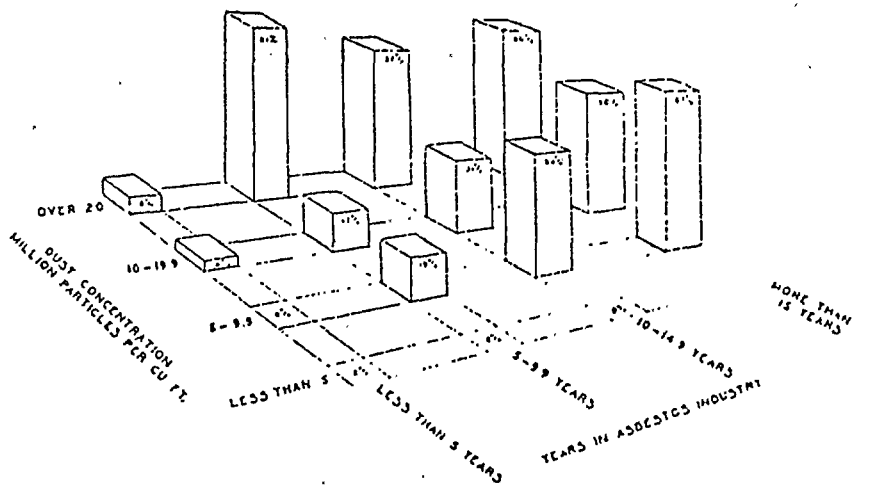


FIGURE 36.—INCIDENCE OF GROUND-GLASS LUNG-FIELD MARKINGS IN RELATION TO LENGTH OF EMPLOYMENT AND DUST CONCENTRATION.

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 1.9	Affected	0	0	0	0
	Exposed	82	19	4	0
	Percentage	0%	0%	0%	0%
5 to 9.9	Affected	0	7	7	4
	Exposed	68	36	13	8
	Percentage	0%	19%	54%	50%
10 to 19.9	Affected	3	3	4	2
	Exposed	50	18	13	4
	Percentage	6%	17%	31%	50%
Over 20	Affected	0	17	8	3
	Exposed	35	24	14	5
	Percentage	0%	71%	57%	60%

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 th[illegible]t 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

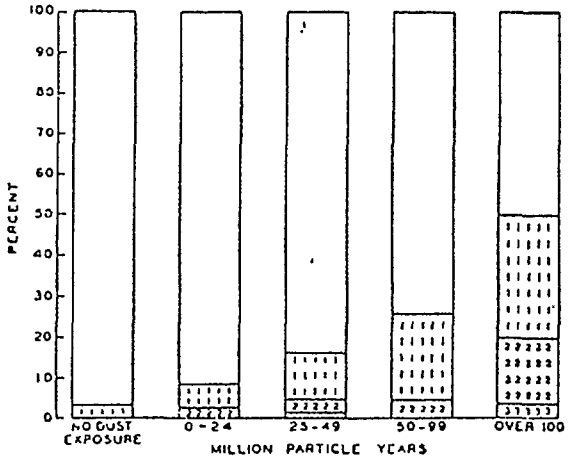


FIGURE 37.—RELATION OF DIAPHRAGMATIC FIXATION TO DUST EXPOSURE. THE PERCENTAGE OF PERSONS IN EACH EXPOSURE GROUP WHO HAD FIRST, SECOND, OR THIRD DEGREE DIAPHRAGMATIC FIXATION HAS BEEN REPRESENTED BY THE HEIGHT OF VERTICAL BARS.

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.

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 100
Normal	97	92	81	71	7
+	3	6	12	22	3
++	0	2	3	1	1
+++	0	0	1	0	

NUMBER OF CASES

Degree of fixation	Controls	Million particle years			
		Under 25	25 to 49	50 to 99	Over 100
Total tested	76	193	86	55	10
Normal	71	182	72	41	5
+	2	11	10	12	3
++	0	5	3	2	1
+++	0	0	1	0	1

[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 râles over the bases of the lungs and interscapular regions seem to accompany asbestosis. Various râles and adventitious sounds were heard in the chests of 5 percent of the persons in the control group. Râles 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 râles or other adventitious sounds. In about two-fifths of the cases, râles 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 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

PHYSICAL EXAMINATION

General Appearance..... Hair:.....Eyes..... Ht..... Wt..... Ur. Wt.....

Skin and mucous membrane..... Nose..... Mouthbreather.....

Eyes: Pupils regular.....Irregular.....React to light R.....L..... Glasses.....

Ears: Hearing..... Discharge.....Throat: Tonsils..... Pharyngitis.....

Teeth ..... Gums..... Pyorrhea.....

Glands: Thyroid, Normal..... Cervical..... Epitrochlear..... Axillary.....

Blood pressure ..... reflexes.....

Exercise		Rate at rest	Exercise	After 3 minutes rest
Pulse:	regular			
	irregular	.....	.....	.....
Respiration	.....	.....	.....	.....

Chest: Type.....

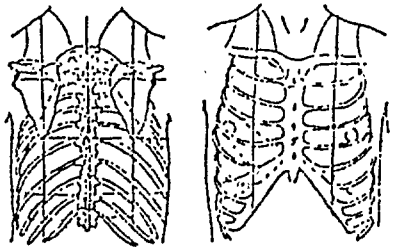
Expansion .....

Palpation .....

Circumf.: Inspiration .....

Expiration .....

Expansion .....



Heart: Size, Normal..... Enlarged..... Position, Normal.....

Murmurs .....Transmitted .....

Hernia .....Enlarged Rings .....

Extremities: Clubbing..... Cyanosis..... Bowed nails..... Edema.....

Special marks of identification.....

Remarks .....

Examined by: .....

FIGURE 38.—FORM USED FOR RECORDING PHYSICAL FINDINGS.

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 49	50 to 99	Over 100
Chest expansion, inches	3.12	2.75	2.69	2.38
Girth at expiration, inches	33.77	33.93	34.10	34.41
Ratio of expansion to girth	.092	.081	.079	.069

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

	Lung-field markings			
	First-degree linear	Second-degree linear	First-degree ground glass	Second-degree ground glass
Chest expansion, inches	2.97	2.62	2.39	2.10
Girth at expiration, inches	33.77	34.27	34.59	34.90
Ratio of expansion to girth	.088	.076	.069	.062

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 kaolin		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*
Under 20	5	2.60	4	3.25	9	3.28
20 to 29	30	3.38	27	3.21	165	2.98
30 to 39	22	3.18	18	3.11	109	2.67
40 to 49	11	2.59	5	2.50	47	2.35
50 to 59	6	3.17	3	2.83	10	2.35
60 to 69	4	2.88	2	2.00	2	2.00

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	202	62	31	8
Before exercise	18.45±0.17	19.32±0.32	20.22±0.[illegible]	21.50±2.21
After exercise	25.49± .31	26.32± .63	28.61±1.05	30.50±1.16
2 minutes after exercise	20.41± .07	21.30± .12	21.37± .58	21.21±1.08

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

Average	Exposed to dust, essentially normal	Anthraco-silicosis
Number of men examined	2,691	616
Rate before exercise	18.73±0.03	20.31±0.04
Rate after exercise	25.59± .07	31.19± .02
Rate after rest	22.73± .08	26.50± .04

[1] These data were not included in Public Health Bulletin No. 221.

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 (51) 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 35.—*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[1]	314	100	78	100	50	100	22	100	463
Normal	267	86	62	79	31	62	14	64	374
+	37	12	10	13	12	24	6	27	65
+ +	8	3	6	8	5	10	2	9	21
+ + +	1	1	0	0	2	4	0	0	3

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

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 discussed, 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.

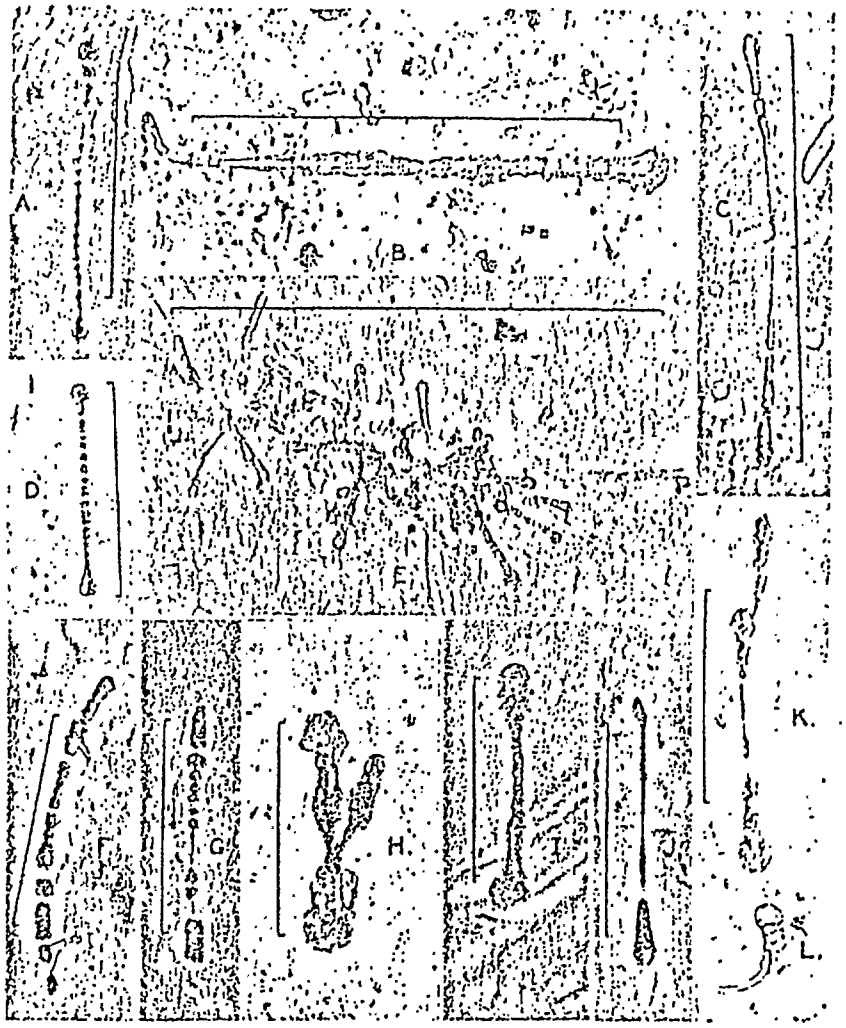


FIGURE 39.—PHOTOMICROGRAPHS OF ASBESTOSIS BODIES FOUND IN SPUTUM. ENLARGED 530 TIMES EXCEPT E, WHICH IS ENLARGED 310 TIMES. A SCALE, RULED IN UNITS OF 50 MICRONS, HAS BEEN DRAWN BESIDE EACH ASBESTOSIS BODY.

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. 39*a*). 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 the ends 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	60	25	42
Coilers	15	6	40
Tape and bobbining weavers	39	11	28
Inspectors, calenders, and finishers	15	4	27
Cloth weavers	36	8	22
Crushers	5	1	20
Yarn spoolers and winders	78	13	17
Mule and ring spinners	60	9	15
Carders	58	5	9
Preparation, stenographers, and clerks	30	2	7
Packers and pilers	16	1	6
Willowers and shakermen	4	0	0
Carders	4	0	0
Formers and grinders	2	0	0
Carpenters, yardmen, and night watchmen	16	0	0
Managers and superintendents	17	0	0
Pre employment	27	0	0
Total	485	85	18

[1] Does not include 17 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 asbestotic 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 [illegible] completely 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 deposits. Apparently asbestosis bodies are formed only in the lungs. They 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 clump or 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 broncho-pneumonia, 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 asbesto[illegible]



FIGURE 39A.—ASBESTOS CORN ON THUMB OF 36-YEAR-OLD MAN EMPLOYED 4 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.[6]

Asbestosis bodies can be demonstrated on sectioning lung tissue. Since they arise as a tissue reaction to an inhaled asbestos fiber, their size is important because it indicates the length of the asbestos fibers that may reach the lungs. The greatest length of the first 168 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 168 asbestosis bodies found in sputum smears*

Length in microns	Number of asbestosis bodies of the length specified	Length in microns	Number of asbestosis bodies of the length specified
Total measured	168	60 to 69	
		70 to 79	
0 to 9	0	80 to 89	
10 to 19	38	90 to 99	
20 to 29	57	100 to 109	1
30 to 39	39	110 to 119	6
40 to 49	25	120 to 129	1
50 to 59	29		

[6] Gloyne's (37) method was used in the present study of asbestosis bodies in sputum. A sample of sputum is digested with an equal volume of concentrated antiformin, centrifuged at moderate speed, the supernatant is pipetted off, and replaced with the following mixture: 2 percent potassium ferrocyanide, 1 part; 1 percent hydrochloric acid, 3 parts. The tube is shaken and allowed to stand for ½ to 1 hour. It is then recentrifuged and the material mounted as a wet preparation. With this technique the asbestosis bodies are colored 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 (31) 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	281	76	27.0
0 to 4.9	23	4	17.6
5 to 9.9	96	21	21.9
10 to 19.9	69	23	33.3
20 and over	74	26	35.1

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).[7] 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			
		0 to 4.9	5 to 9.9	10 to 19.9	Over 20
Milligrams silica per 100 cc	3.08±0.13	3.55±0.13	2.96±0.08	2.68±0.11	3.41±0.10
Milligrams silica per day	30.15±.14	36.52±1.26	30.30±1.28	28.47±1.30	37.55±1.31
Number tested	66	68	108	77	100

Silica was present in concentrations ranging from 0.7 to 11.1 mg per 100 cc. Average urinary silica concentrations for groups of persons whose dust exposure varied widely (table 39) do not indicate that there was any simple relation between the exposure to asbestos dust (which consists of hydrated magnesium silicate) and the concentration of urinary silica. Rather, it appears that the values for

[7] The colorimetric procedure for the determination of silica in the urine consists in the production of a silicomolybdic acid complex which is reduced to give a blue color, the depth of color being proportional to the 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 [6] showed that the daily silica output averaged 29.89 ± 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 (59) 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 (59) 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	
1.006 to 1.009	2.11	25
1.010 to 1.019	2.69	163
1.020 to 1.029	3.20	193
Over 1.030	5.10	23

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.

Occupational history.—In establishing a diagnosis of asbestosis, it is important to exclude the possibility that the observed effects may have resulted from inhalation of another kind of dust or some other disease process. An early case of silicosis, for instance, might present signs and symptoms which would differ from those of asbestosis mainly in degree. A diagnosis of asbestosis should be made only when the occupational history shows previous exposure to asbestos dust and no heavy exposure to other siliceous dust.

X-ray findings.—Although the ground-glass markings that are so characteristic of asbestosis are usually localized in the basal portions of the lung fields, nevertheless all regions of the chest must be examined roentgenographically in order to detect signs of other disease processes that might complicate diagnosis. It is important, of course, to look for the signs of pulmonary tuberculosis. The distribution and the character of lung-field markings are significant. Shagginess of the heart shadow (see p. 56) has been mentioned by several physicians who have reported cases of asbestosis. The physical findings should be referred to whenever cardiac enlargement is found during the X-ray examination. A consideration of the chest shape of the individual will sometimes modify the interpretation of certain of these findings. Fluoroscopic examination is, of course, the best means of detecting diaphragmatic impairments, but valuable information can often be gained from a study of a chest X-ray film. Heavy pectoral muscles may cast an X-ray shadow which resembles in a single plate the shadow cast by the increased fibrosis in the bases. This possibility must always be kept in mind during the examination of chest X-ray films. Notes on the thickness of the chest muscles should be entered on the physical examination form, or stereo films should be made in order to obviate this source of error. The presence of bronchiectasis caused difficulty in diagnosing one case.

Classification of lung-field markings.—In this study, after examining an X-ray film and before making a diagnosis, the film was classified according to the nature and degree of fibrosis into one of four classes: first- or second-degree linear exaggeration of the lung-field markings or first- or second-degree ground-glass lung-field markings. There is good reason to believe that these four classes represent successive stages through which a typical case of asbestosis would pass under continuous exposure to asbestos dust. Since, as has been said, the ground-glass markings appear first in the basal portions of the lung fields, it follows that any given chest film might have areas in which the markings were ground glass and other areas in which the markings were linear. Usually the basis of classification has been the type of marking that predominates in the lower third of the visible lung field.

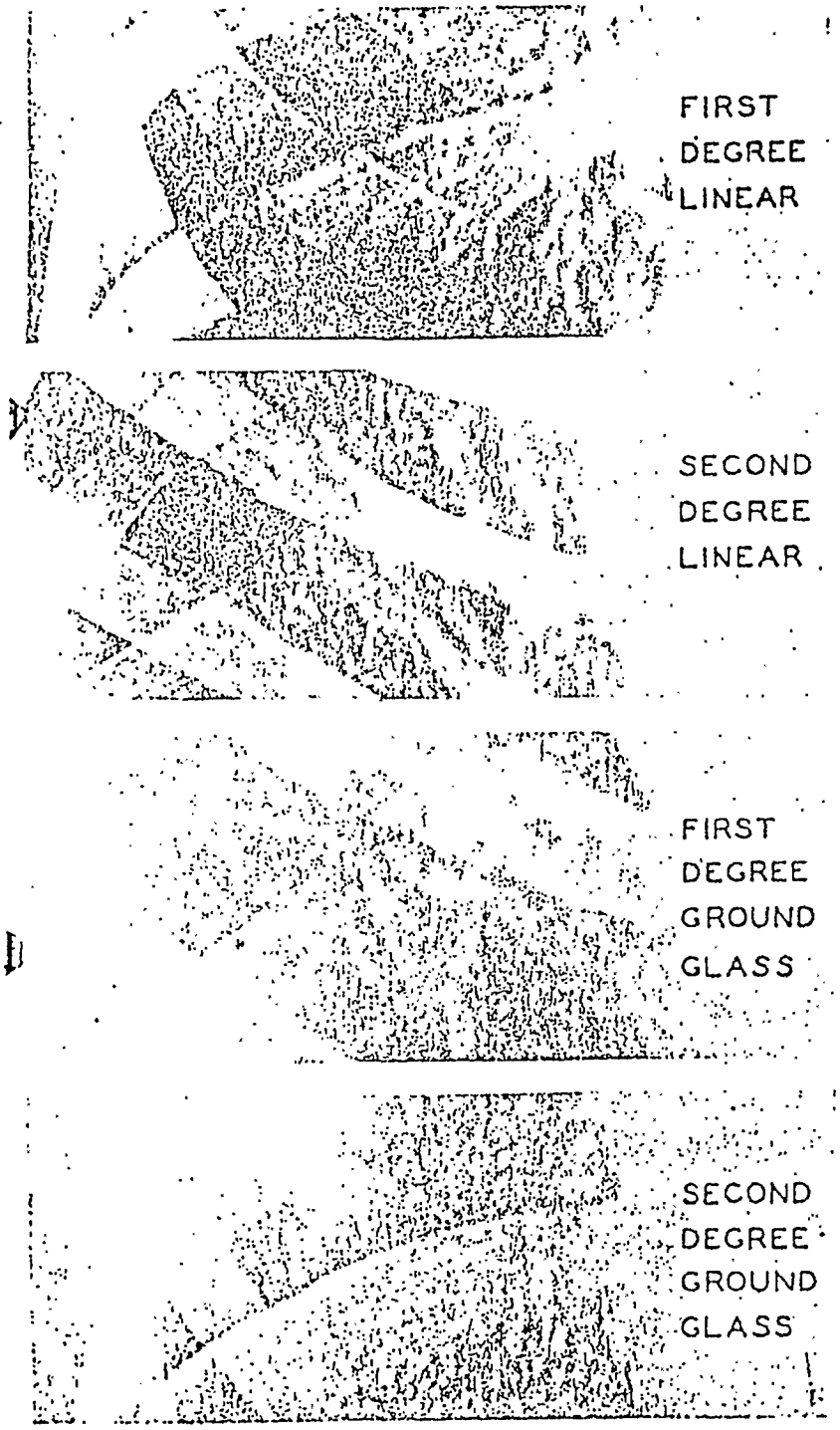


FIGURE 40.—PORTIONS OF CHEST X-RAY FILMS WHICH ILLUSTRATE THE SUCCESSIVE C[illegible]S BROUGHT ABOUT BY LONG-CONTINUED INHALATION OF ASBEST[illegible].

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[9] 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.

[9] Cooke has reported somewhat similar bodies in the sputum of anthracite miners, but they seem to differ morphologically from asbestosis bodies.

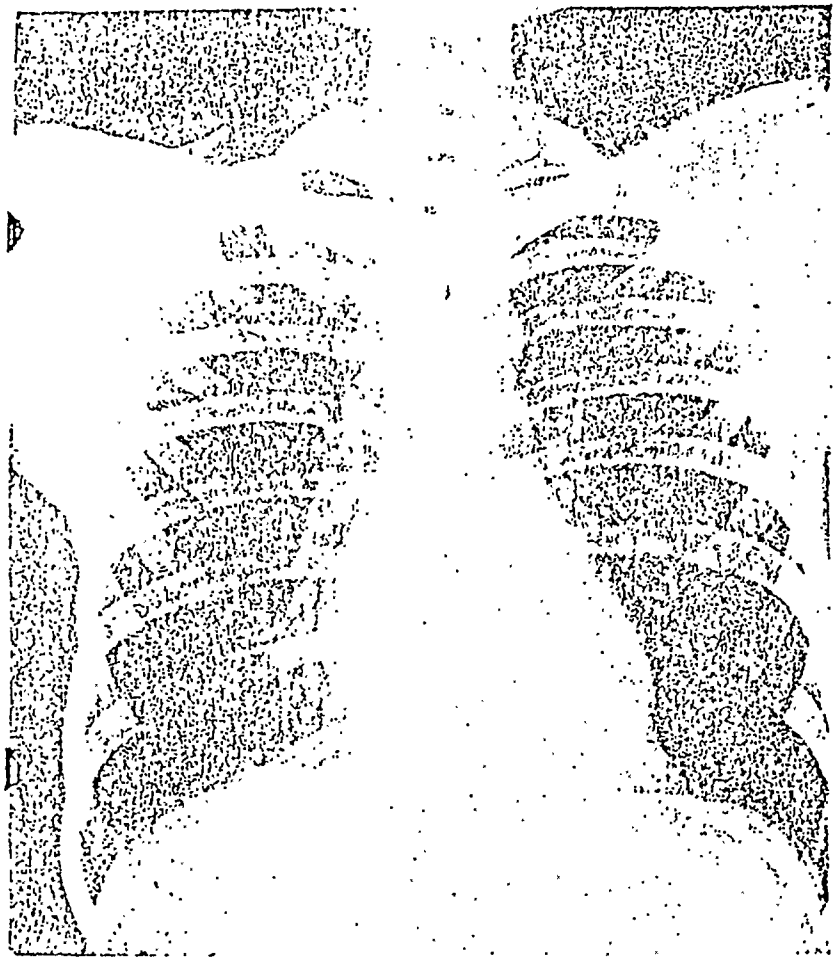


FIGURE 41.—NORMAL PULMONIC MARKINGS. WHITE MALE, AGE 23.

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.

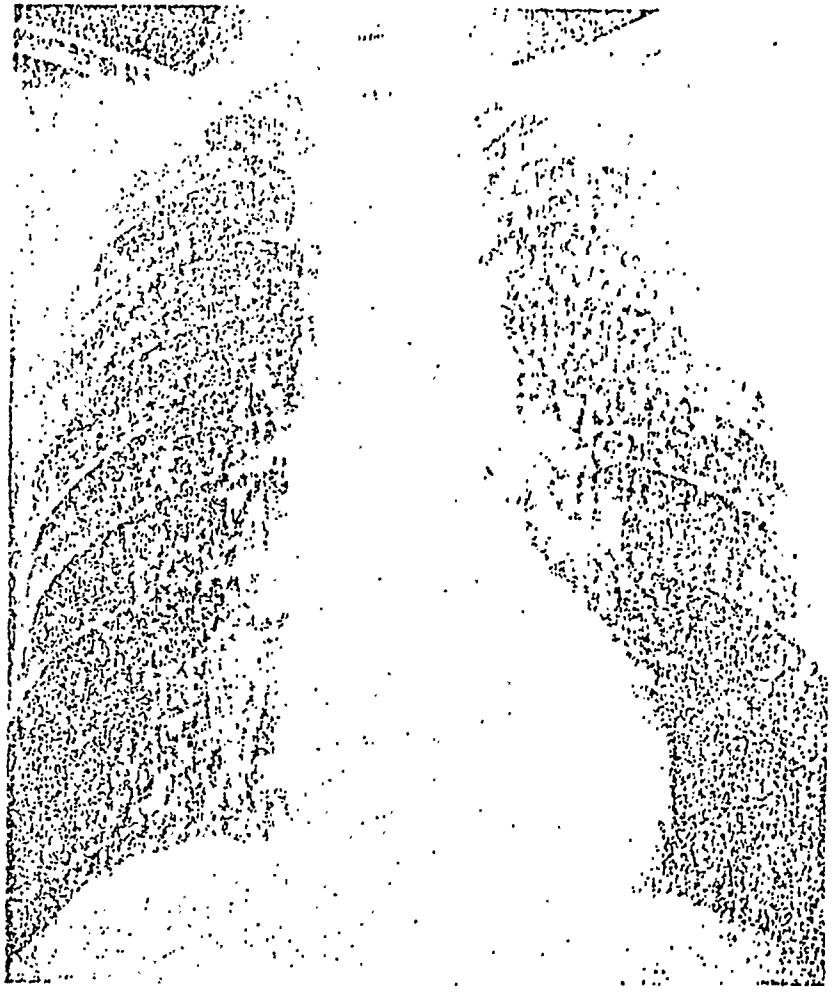


FIGURE 42.—SILICOTIC PULMONIC MARKINGS. WHITE MALE, AGE 61.

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.

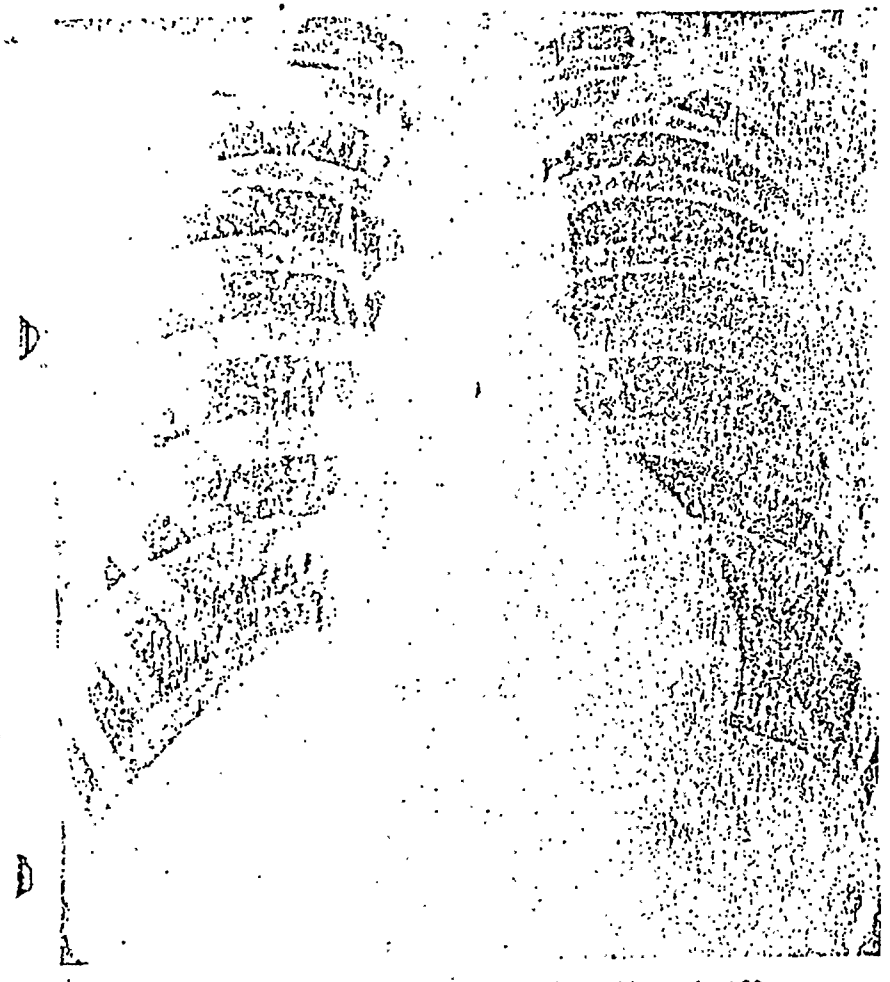


FIGURE 43.—EARLY ASBESTOSIS. WHITE MALE, AGE 29.

Occupational history.—Carder (asbestos), 4½ years; nondusty 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, 67¼ inches; weight, 142 pounds. Rather long chest; expansion, 2 inches. No change in fremitus, breath sounds, or percussion note. Blood pressure, 168/118. Pulse rate, 96, 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, close-set reticular markings just off each hilum and the apices remaining quite clear.

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

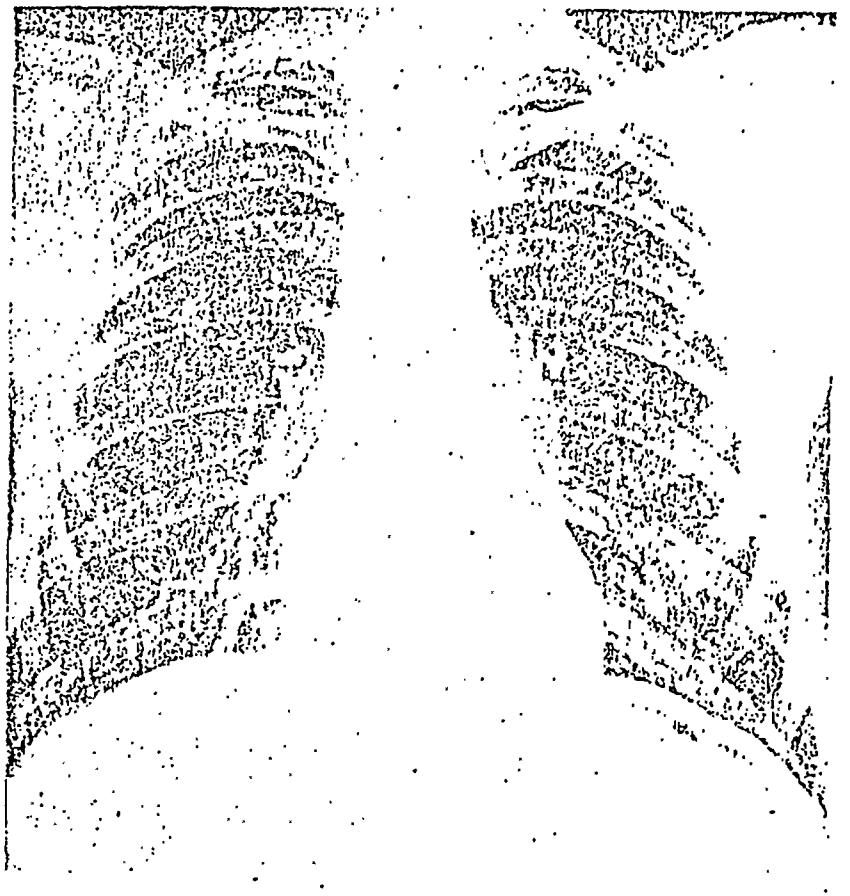


FIGURE 44.—EARLY ASBESTOSIS. WHITE MALE, AGE 39.

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, 3¾ inches. Blood pressure, 136 82. Pulse rate, 83, 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 râles at right base. Asbestos corn on palmar surface of left wrist. Slight cyanosis of lips and nails. A dusky red cast to ears. Sputum negative for *B. 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.

X-ray.—Note the first-degree grainy or ground-glass appearance, not obliterating the linear markings, giving general reticular appearance to lung fields. Generalized fibrosis 1 +.

Diagnosis.—Asbestosis 2.

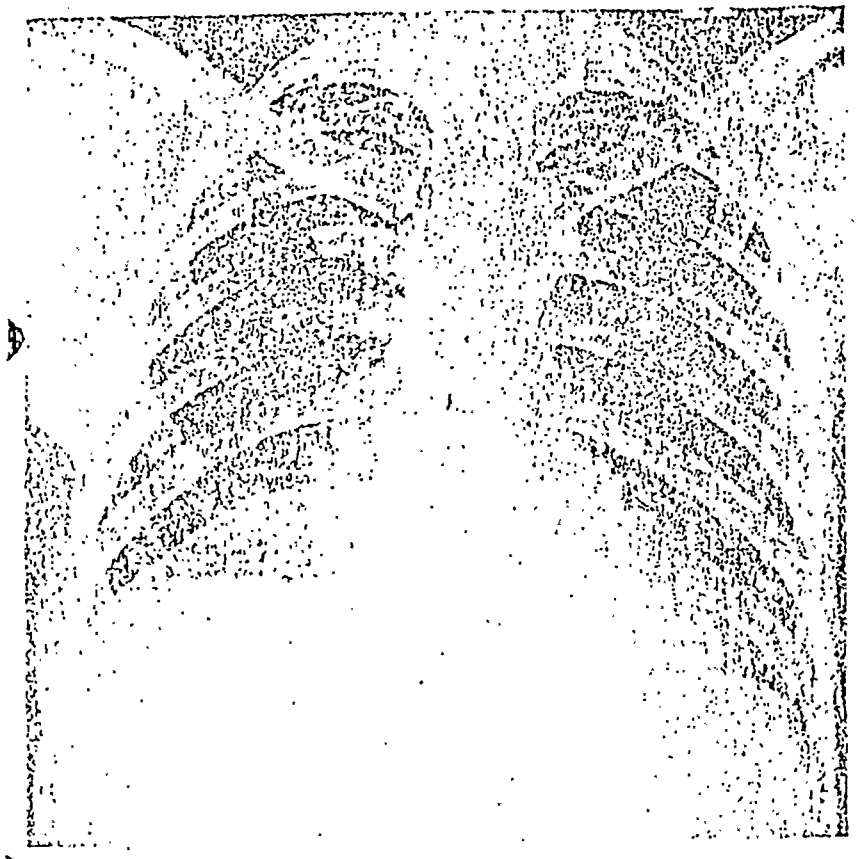


FIGURE 45.—MODERATELY ADVANCED ASBESTOSIS. WHITE MALE, AGE 46.

Occupational history.—Broadcloth weaver (asbestos), 7 years 5 months. Previously in nondusty trades, 15½ 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 82. 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 râles 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).

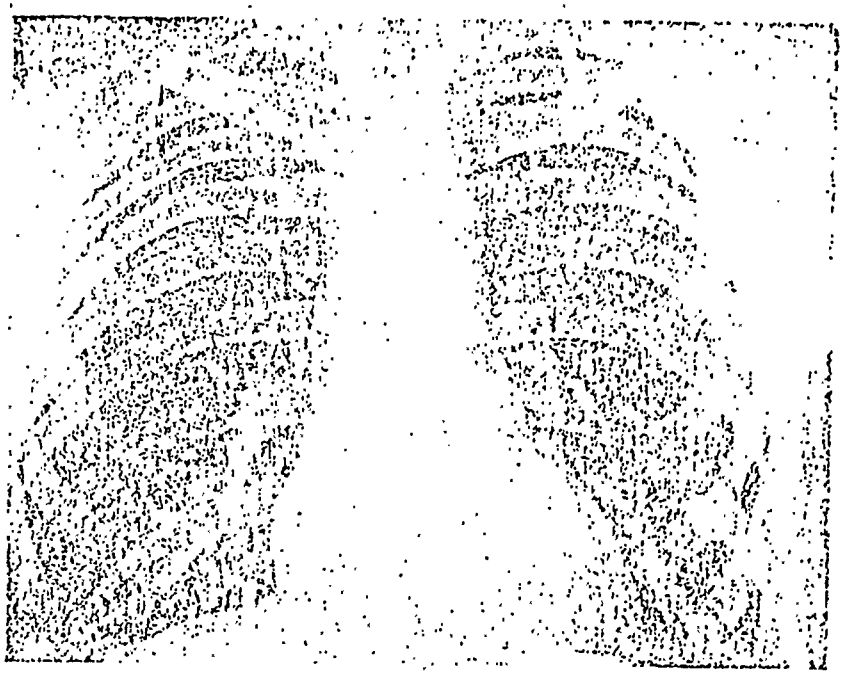


FIGURE 46.—MODERATELY ADVANCED ASBESTOSIS. WHITE MALE, AGE 40.

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

Estimated dust exposure.—(*a*) Weighted average, 13.5 million particles per cubic foot. (*b*) Million particle years, 175.7.

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 râles over bases of lungs. Urinary silica, 5.6 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.

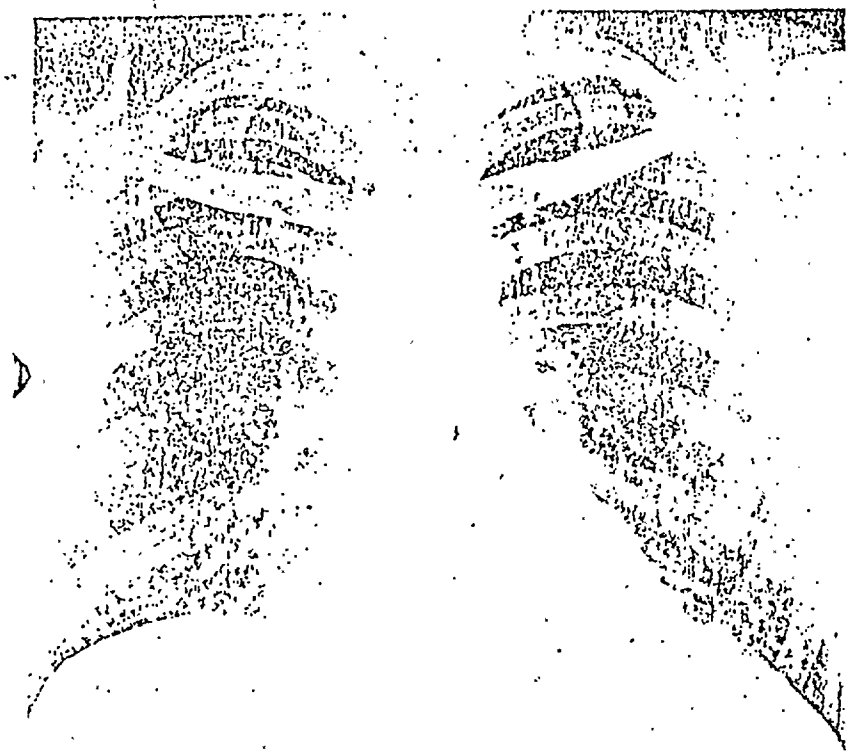


FIGURE 47.—MODERATELY ADVANCED ASBESTOSIS. WHITE MALE. AGE 49.

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 5 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, 100 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 râles at bases on deep breathing and after cough. Sputum (single specimen) was negative for acid-fast bacilli and asbestosis bodies. Urinary silica, 3.2 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.

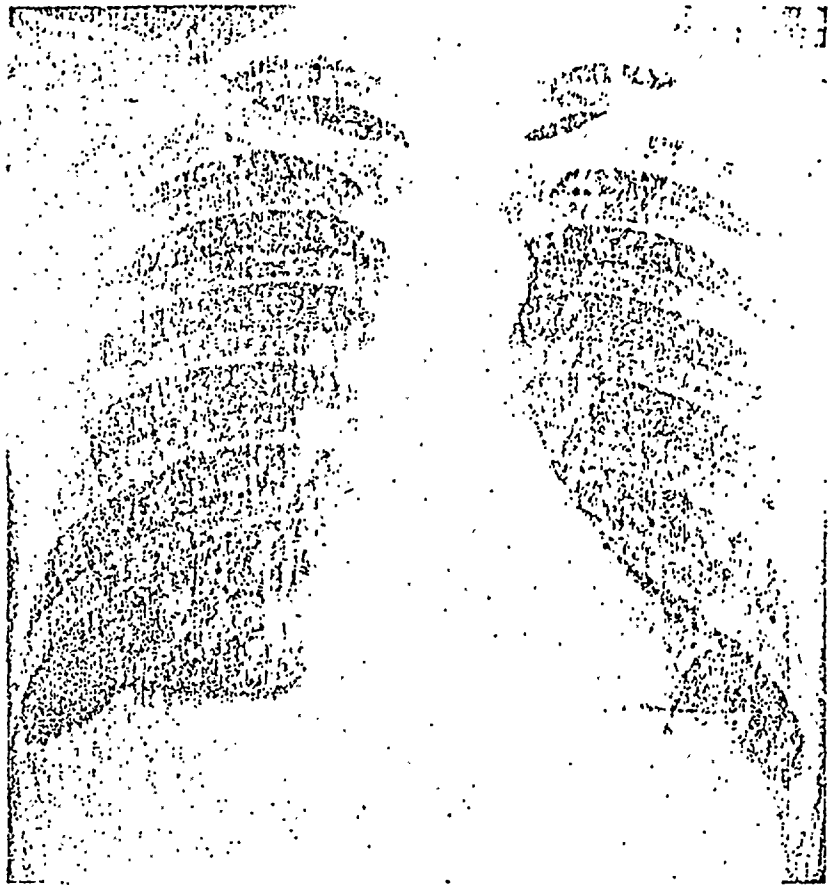


FIGURE 48.—MODERATELY ADVANCED ASBESTOSIS. WHITE MALE. AGE 39.

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

Estimated dust exposure.—(*a*) Weighted average, 49.7 million particles per cubic foot. (*b*) Million particle years, 459.7.

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, 101/70. 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 second-degree diffuse grainy or ground-glass appearance with emphysema.

Diagnosis.—Moderately advanced asbestosis (asbestosis 3).

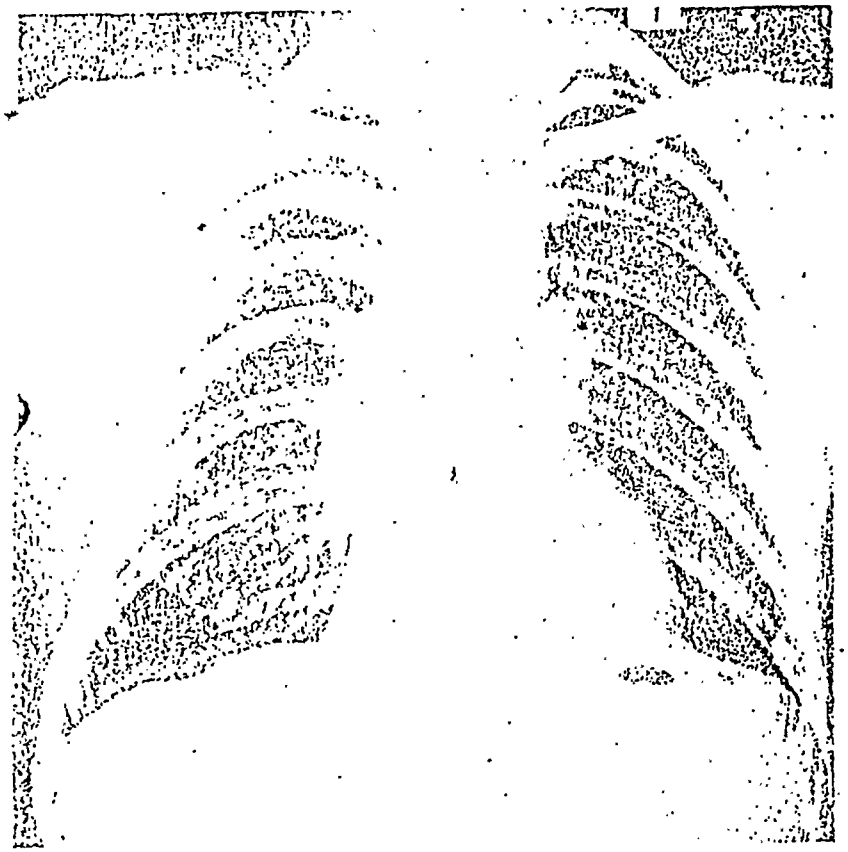


FIGURE 49.—ADVANCED ASBESTOSIS. WHITE MALE, AGE 48.

Occupational history.—Mule spinner (asbestos), 17¼ years (1½ years in same room with carding machines; 11 years he worked 11 hours a day; nondusty trades, 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 acidfast bacilli and asbestosis bodies.

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

X-ray.—Shows second-degree grainy appearance with almost complete obliteration of lin[illegible]markings. Generalized fibrosis 2+.

Diagnosis.—[illegible]-established asbestosis.

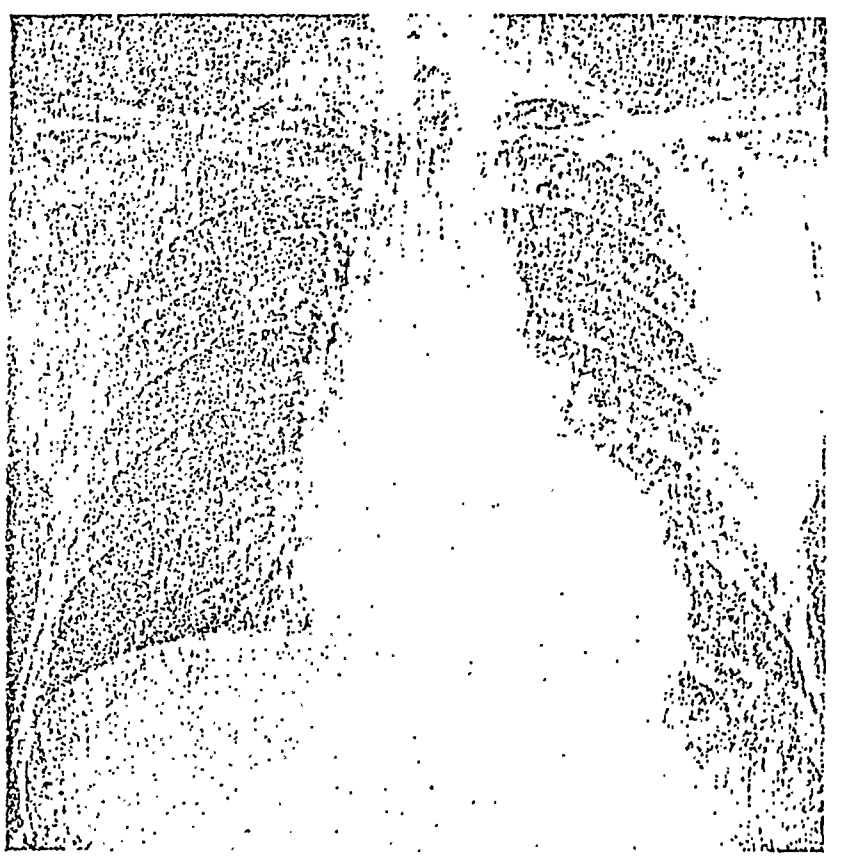


FIGURE 50.—ADVANCED ASBESTOSIS. WHITE MALE, AGE 55.

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

Estimated dust exposure.—(*a*) Weighted average, 38.9 million particles per cubic foot. (*b*) Million particle years, 545.

Past medical.—Maxillary sinuses drained in 1930. Typhoid fever as a child.

Complaints.—Dry cough, 10 years, productive of 2 ounces of white, thick sputum over a period of 24 hours. Rather severe dyspnoea, 3 years. He must walk very slowly to prevent dyspnoea. Occasional edema of ankles for the past 4 years.

Physical examination.—Height, 66 inches. Weight, 132 pounds. Blood pressure, 112/70. Moderate arteriosclerosis. Pulse rate, 72, 80, and 68. Respiratory rate, 48, 51, and 51 (before, immediately after, and 2 minutes after functional exercise test). Patient stopped on completion of about one-half of prescribed exercise, but expressed a willingness to continue. Chest expansion, 2 inches. Breathing is labored and shallow. Prolonged expiration. Fine crackles appear over the lower half of both lungs after coughing. Heart is enlarged to the left with the aortic second sound somewhat more prominent than the pulmonic second. First sound at the apex is split and is particularly snapping elsewhere; 2+ curving of the finger nails.

Fluoroscopy.—Aortic shadow is definitely widened. The cardiac shadow shows 1+ increase in size. Diaphragm on ordinary breathing moves a scant 1 cm; 3 cm on forced excursion.

X-ray.—Shows diffuse ground-glass appearance. Note that most of the distinctly linear markings have been obliterated.

Diagnosis.—Advanced asbestosis.

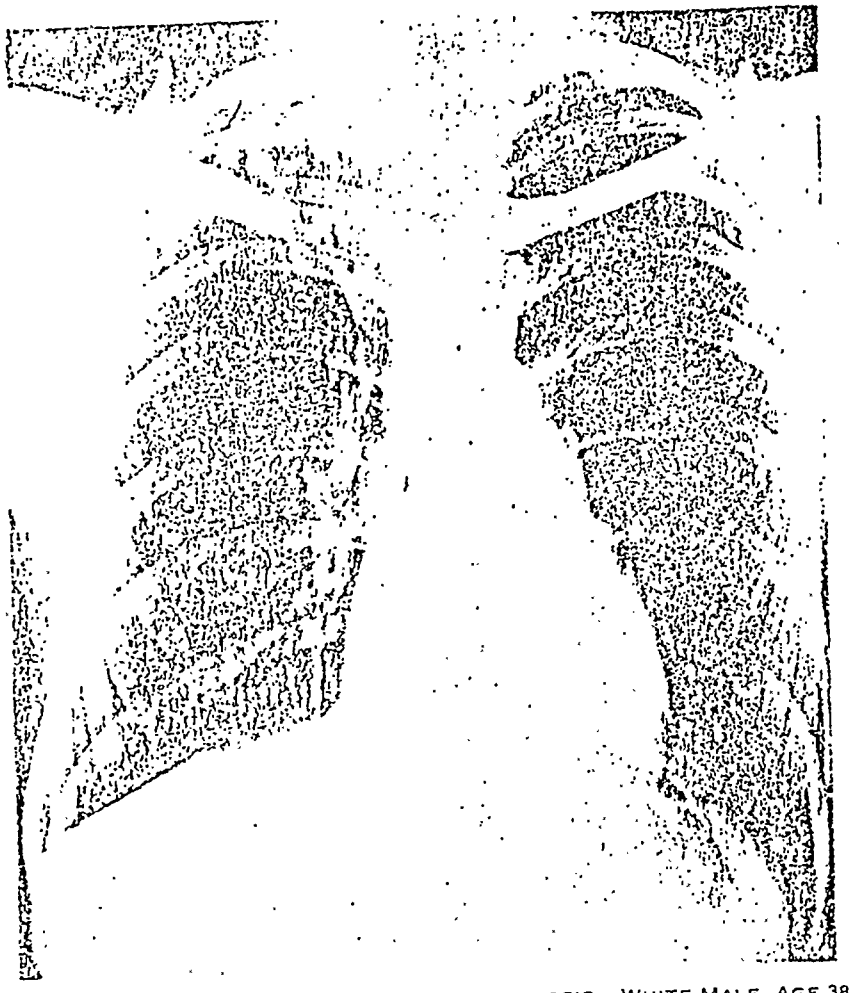


FIGURE 51.—ASBESTOSIS WITH FIBROID TUBERCULOSIS. WHITE MALE. AGE 38.

Occupational history.—Mule spinner (asbestos), 11 years 8 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 dyspnoea, 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, 1½ 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.

Fluoroscopy.—Diaphragm moves freely and equally on both sides. On forced respiration, excursion is 3 cm on right and 4 cm on left.

Diagnosis.—Moderately advanced asbestosis with arrested fibrotic 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 (15) 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	372	21	26	16	7	116
First-degree linear	297					297
Second-degree linear	65	11	3			79
First-degree ground glass	6	13	22	8		49
Second-degree ground glass	4		1	8	7	20

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 anthraco-silicosis 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* [1]

Diagnosis	Million particle years				Total
	Less than 25	25 to 49	50 to 99	Over 100	
Essentially normal	191	81	43	52	367
Suspect 1	5	2	0	12	25
Suspect 2	0	1	1	23	25
Suspect 3	0	0	0	11	17
Asbestosis	0	0	0	7	7
Total	196	84	56	105	441

[1] 1 person whose exposure could not be estimated and 2 persons with active pulmonary tuberculosis 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 1.9	Affected	2	1	0
	Exposed	81	19	5
	Percentage	2%	5%	0%
2.5 to 4.9	Affected	0	6	13
	Exposed	70	37	19
	Percentage	0%	16%	68%
Over 10	Affected	6	22	21
	Exposed	131	43	36
	Percentage	6%	51%	58%

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.

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 pilers	20		2	0	1
Carders	72		4	0	11
Spinning, twisting, and winding	216	14			
Mule and ring spinners	65		3	8	5
Twisters	61		5	2	2
Yarn spoolers and winders	83		3	2	0
Corders	4		1	0	0
Weaving	99	14			
Cloth weavers	43		3	4	4
Tape and brake lining weavers	41		1	1	0
Cleaders	15		1	0	0
Finishing	18	11			
Inspectors, calenderers, and finishers	15		0	1	0
Formers and grinders	3		1	0	0
Supervisory intendence 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	512	14	25	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 3[illegible] 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 1935:					
Silicosis	311	55.8	62	2.3	35.1
Silicosis with tuberculosis	392	52.4	67	2.0	31.7
Asbestosis	52	41.9	27	1.5	12.4
Asbestosis with tuberculosis	30	37.1	29	.8	9.5

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.

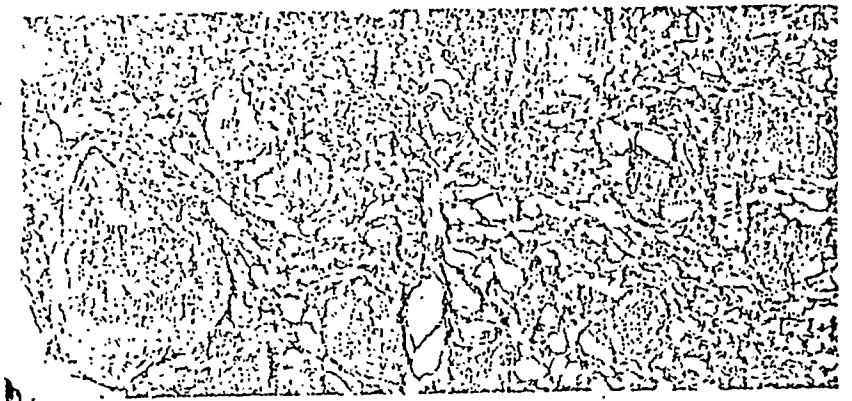
The hilar lymph nodes are not markedly increased in size. They are pinkish gray and contain about the usual amount of anthracotic pigment found in a city dweller.

MICROSCOPIC APPEARANCE

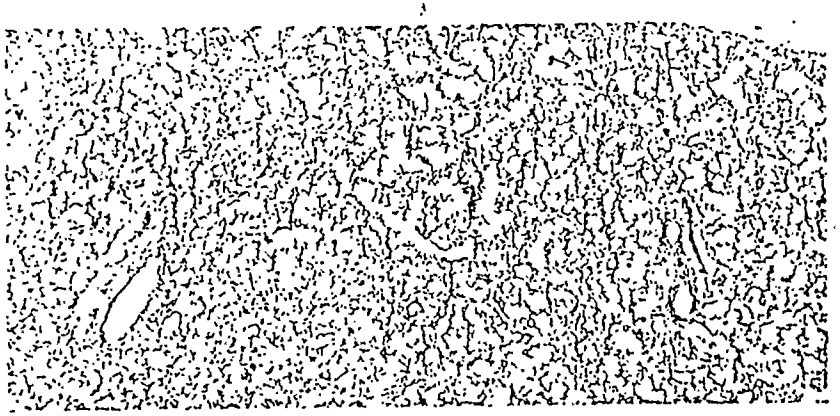
The pleura, when thickened, is made up of adult fibrous connective tissue cells. The collagen fibers are usually well developed and may show hyalinization. In some sections the thickened pleura is vascular, the blood vessels generally being small capillaries. Areas of variable size, containing anthracotic 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.

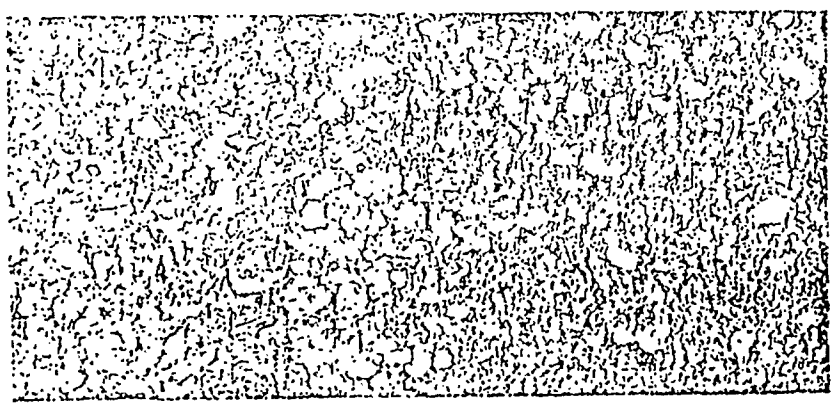
Asbestosis bodies are found in almost all parts of the pulmonary tissue. They occur most frequently in the areas of fibrosis and are accompanied by a greenish black pigment. They are also noted in the connective tissue about the small bronchi, in the alveolar walls, and in the alveoli. Macrophages and 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 ir[illegible] 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. 52255.)

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 nodules may

resemble the developing fibrous areas of asbestosis to a marked degree. It is not easy in many cases to differentiate caseation from old hyalinized connective tissue in the stained specimens. In one of the cases studied, the presence of acidfast bacilli in the alveoli and connective tissue of the alveolar walls without the presence of caseation was observed. This could possibly be an early tuberculous infection in the preinvasive stage, or it could well be a contaminating nonpathogenic acid-fast bacillus.



FIGURE 54.—ASBESTOSIS BODIES ACCOMPANIED BY GIANT CELLS IN AN AREA OF FIBROSED LUNG TISSUE. CASE H-21. MAGNIFICATION 705X. (ARMY MEDICAL MUSEUM NO. 52253.)

Samples of three varieties of asbestos (chrysotile, crocidolite, and amosite) were injected into the peritoneal cavities of guinea pigs, using a technique developed by the Public Health Service for studying the physiological behavior of dust. This method has been described elsewhere (60). All three samples of asbestos dust produced an inert type of reaction in the peritoneal tissue. On the basis of this response, asbestos dust has been considered to be potentially harmful, especially when inhaled over long periods of time. Asbestos dust, like talc (43, 44), which also produces an inert reaction, causes a diffuse, pulmonary fibrosis as has been observed on clinical and post-mortem examination of persons exposed to these dusts.

SELECTED AUTOPSY CASE REPORTS

Case H-21

(Figs. 55 to 58)

Male, white, aged 38 years.

Cause of death.—Myocarditis.

Occupational history.—Worked for 12 years in the weaving room of an asbestos plant. Prior to this he had worked in a cotton mill.

Medical history.—Confined to bed for the most part of his last 6 months of life because of weakness, dyspnoea, and unproductive cough.

Pathological examination showed well-defined advanced asbestosis, evidenced by much diffuse interstitial pulmonary fibrosis, accompanied by asbestosis bodies, emphysema, bronchiectasis, and bronchiolectasis.

Chemical examination of lung tissue.—Sample from right lung—ash 3.3 percent, silica 0.6 percent, silica in ash 18.32 percent. Sample from left lung—ash 3.8 percent, silica 0.76 percent, silica in ash 20.13 percent.

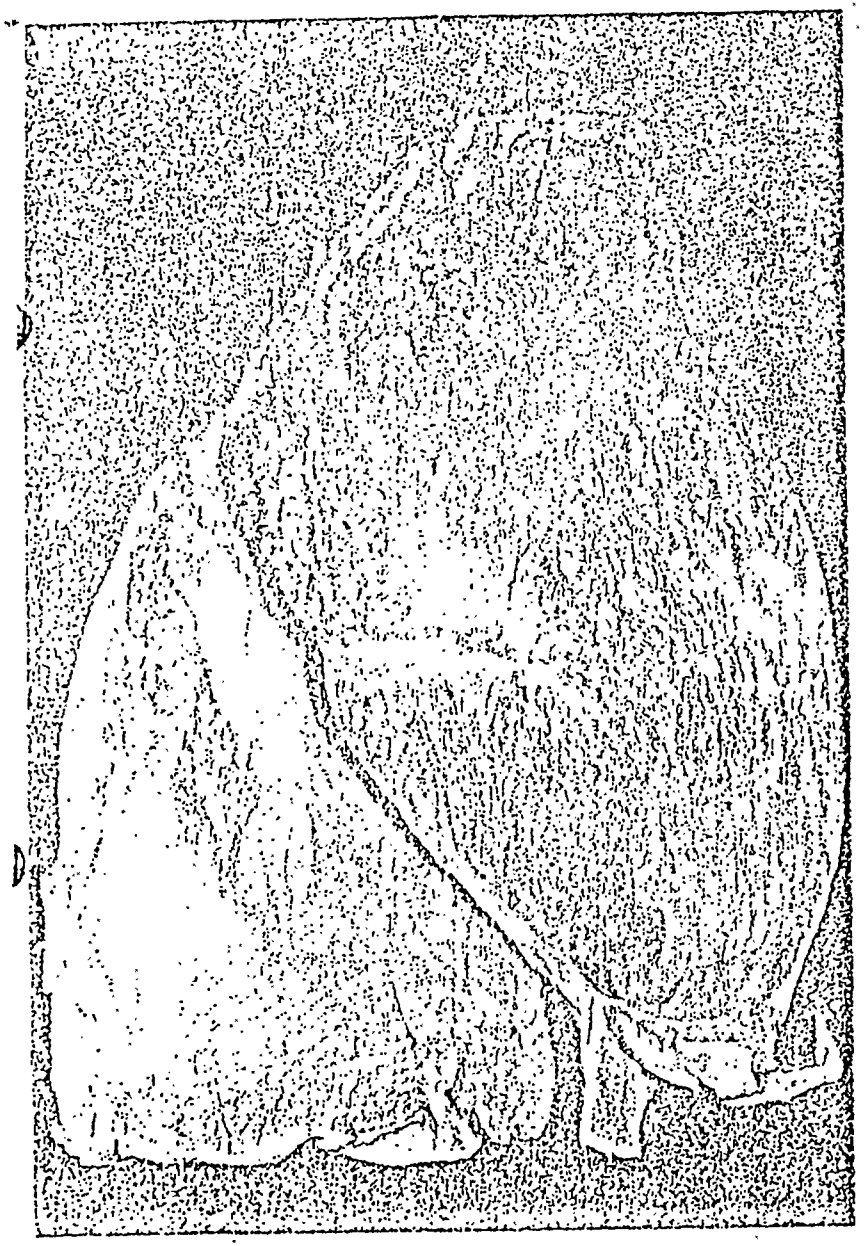


FIGURE 55.—CASE H-21. RIGHT LUNG. MARKEDLY THICKENED PLEURA WITH FUSION OF INTERLOBAR PLEURA. DENSE FIBROUS TAGS OVER ENTIRE SURFACE.

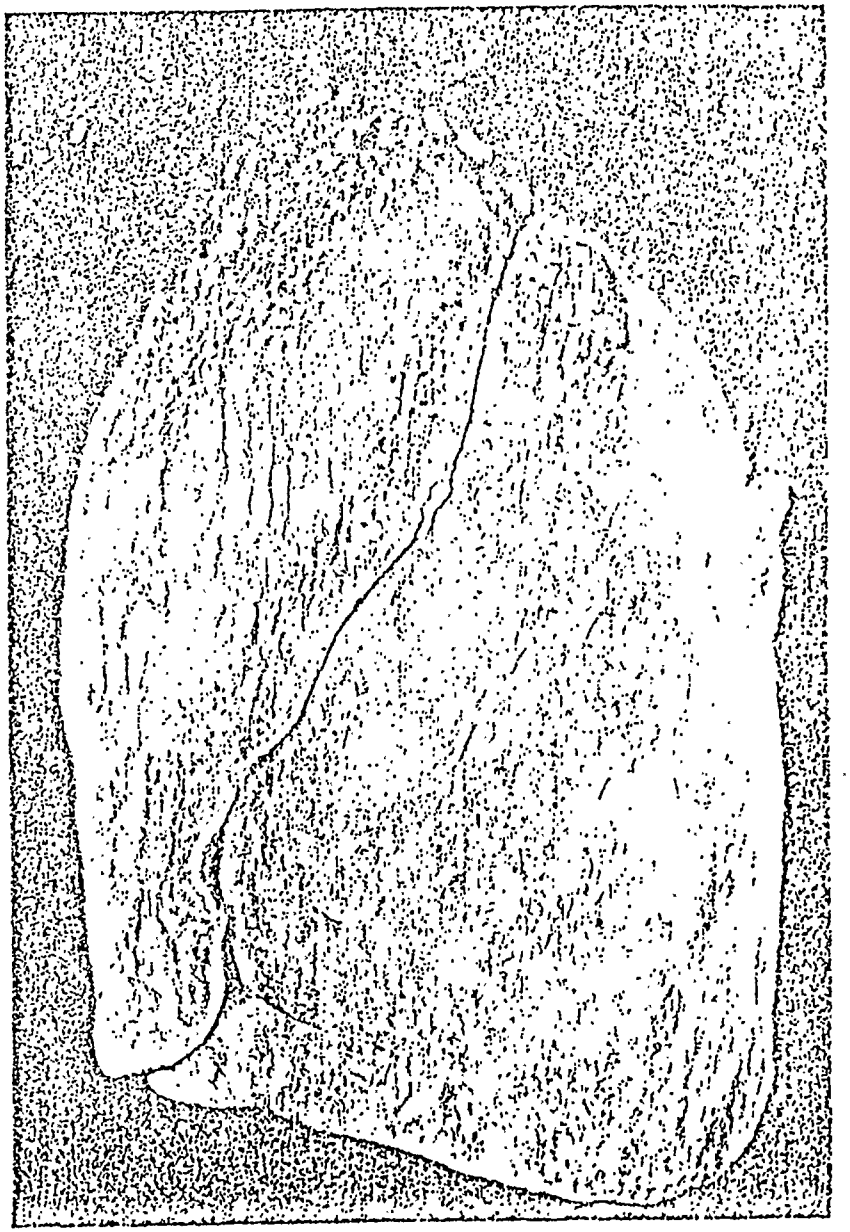


FIGURE 56.—CASE H-21. LEFT LUNG. PLEURA MUCH THICKENED. FEWER FIBROUS ADHESIONS THAN WERE NOTED ON THE RIGHT LUNG.

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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 H-21. SECTION OF LEFT LUNG. DISTENDED BRONCHI AND AIR SPACES AT APEX. RATHER UNIFORMLY DISTRIBUTED FIBROUS AND EMPHYSEMATOUS AREAS THROUGHOUT REST OF LUNG.

Case H–23

(Figs. 59 to 63)

Male, Negro, aged 35 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, 158.9.

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, 70¼ 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 0.41 percent, silica in ash 16.3 percent.

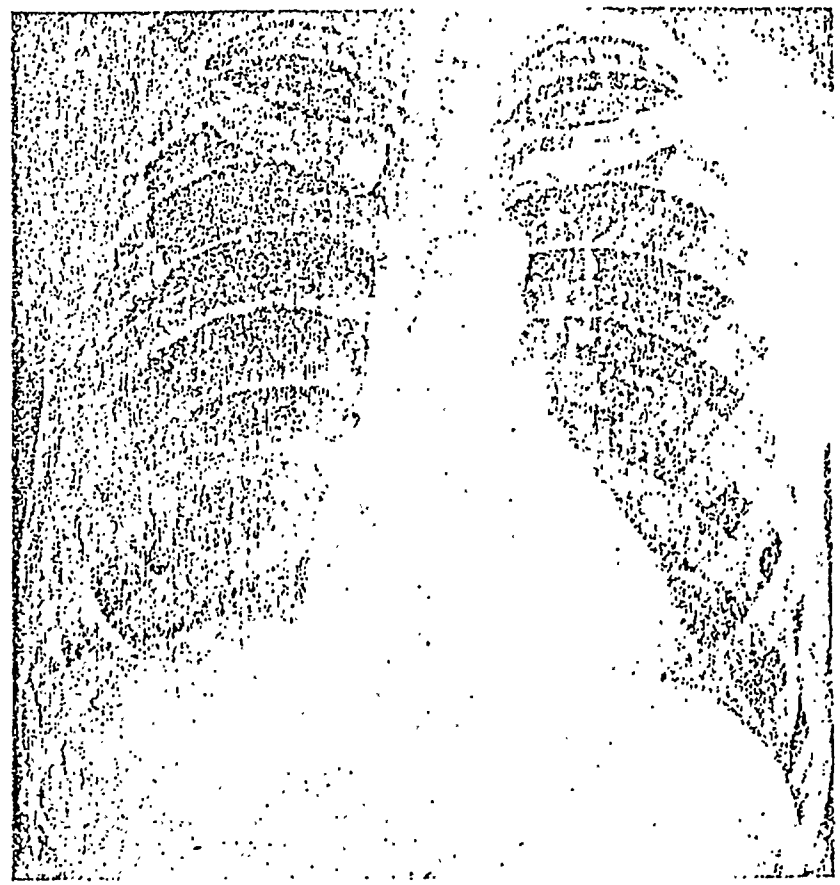

FIGURE 59.—CHEST X-RAY OF CASE H-23 15 MONTHS BEFORE DEATH.



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



FIGURE 61.—CASE H-23. LEFT LUNG. VERY FEW FINE FIBROUS TAGS ARE NOTED ON THE PLEURA.



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

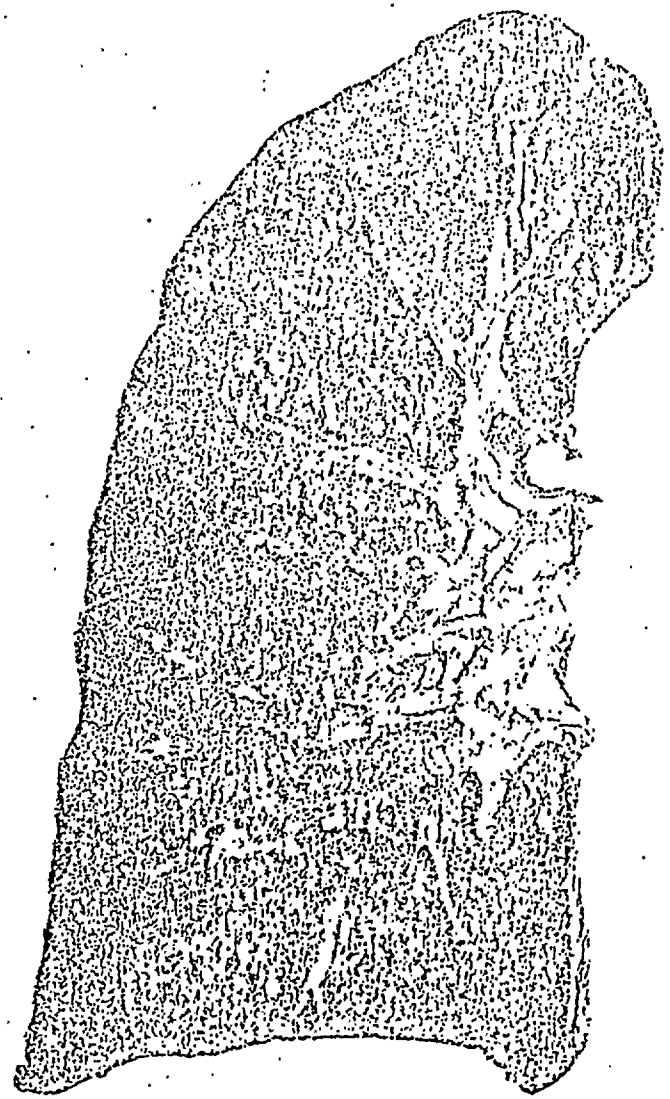

FIGURE 63.—CASE H-23. SECTION OF LEFT LUNG. SHOWS LESS INVOLVEMENT THAN RIGHT LUNG (FIG. 62).

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.—Films 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.

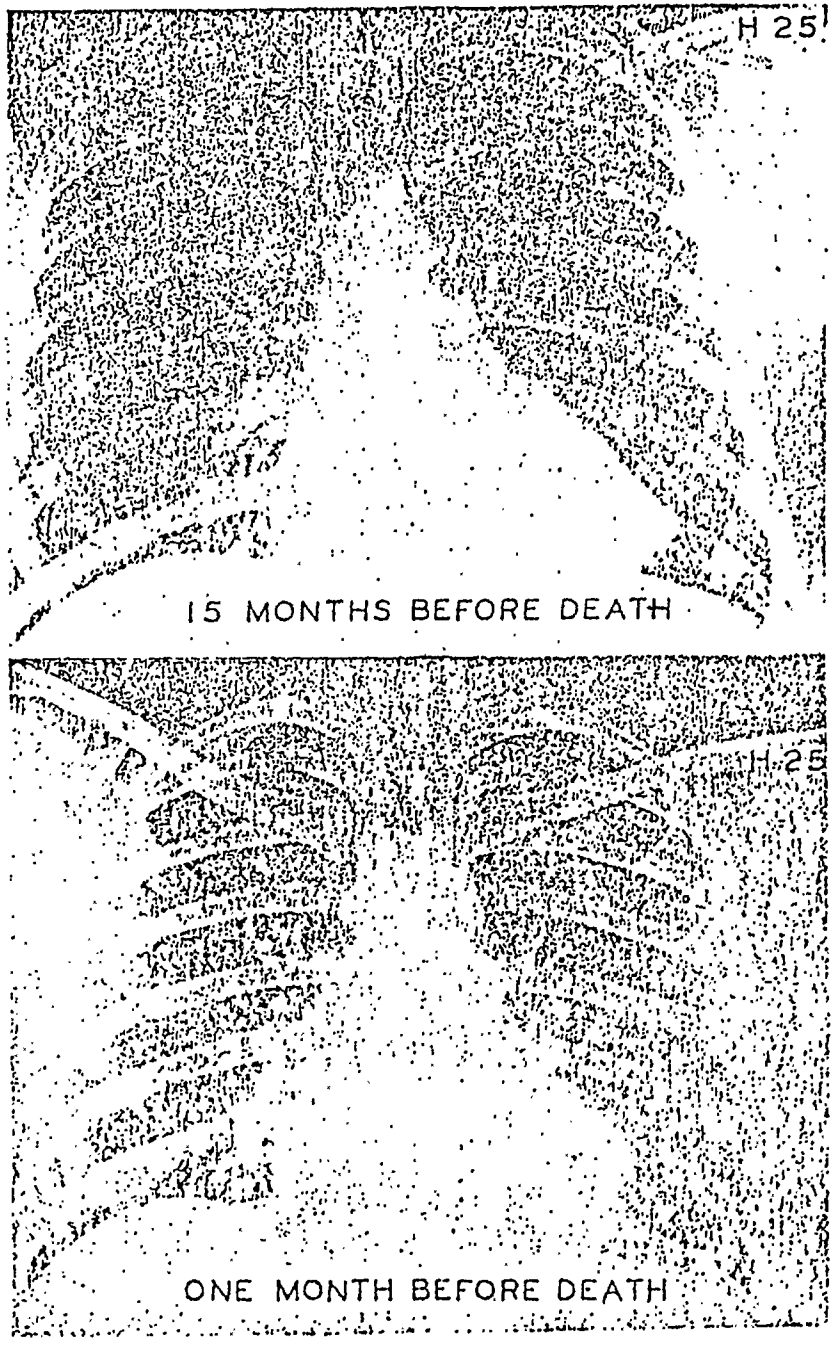

FIGURE 64.—CASE H-25. CHEST X-RAYS TAKEN 15 MONTHS AND 1 MONTH BEFORE DEATH. PART OF THE DIFFERENCE BETWEEN THE TWO ROENTGENOGRAMS IS DUE TO A DIFFERENCE IN X-RAY TECHNIQUE.



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 air-borne 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: 289-293. 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. Münchener 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 für 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. Sundius and Bygden (41a) have reported on the asbestosis bodies found in this case.

8b. Buresch, Annemarie. Pneumonokoniose, Berufskrankheit und Entschädigungspflicht. 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 casation and cavitation were found.

10. Dewirtz, 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: 1275-1281. 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: 571 584. 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. Elkman, P. Pulmonary asbestosis: Its clinical, radiological, and pathological features, and associated risk of tubercuIous 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 tubercuIous 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. Lavisetto, Domenico. 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. 506–509.

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 pneumoconiosis; direct ratio between quantity of dust inhaled and pneumoconiosis. 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, 46 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 5-year period showed mottling 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: 69 81; 109–118; 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. Mussa, 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. 509.

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 bases."

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. Pancoast, 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 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 percentages 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 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	48	7
5 to 9.9	28	17
2.5 to 4.9	15	32
Under 2.5	9	44

[1] Data from table 43.
[2] Data from (7).

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

Dust count in million particles per cubic foot	1,135 anthracite miners exposed to dust containing less than 5 percent free silica			602 men employed in haulageways who were exposed to dust containing about 13 percent free silica			151 rock workers exposed to dust containing about 35 percent free silica		
	Years in industry			Years in industry			Years in industry		
	Less than 15	15 to 21	Over 25	Less than 15	15 to 21	Over 25	Less than 15	15 to 21	Over 25
5 to 50	1.1	1.5	7.1	0.4	3.1	23.3	(2)	(2)	(2)
100 to 761	1.8	11.1	51.0	0	0	26.3	12.5	(2)	(2)
30 to 250	0	28.9	71.1	(2)	(2)	(2)	23.5	71.4	(2)
Over 761	1.7	58.1	88.8	(2)	(2)	(2)	8.8	50.2	92.0

[1] From table 30, p. 76, 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. If 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, 63 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.

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.

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, duty-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. Shall, 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. Steck, 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. Stroebe, H. Bericht über den Fall von Lungen-asbestosis, welcher der Arbeit des Herrn Prof. Beger zugrunde liegt. Virchow's Archiv. f. pathologische Anatomie u. Physiologie 290: 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, flocculent 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.

34. 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, nonproductive cough, and slight [illegible] 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, widespread 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, 53 had uncomplicated asbestosis, 21 had asbestosis with active tuberculosis, 9 had asbestosis with obsolescent 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. Steward, 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: 737–741. 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. Bygdén. Der Staubinhalt einer Asbestosislunge und die Beschaffenheit der sogenannten Asbestosiskörperchen. Archiv für 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 workers. Indust. Med. 6: 195–200. 1937.

Results of a medical survey of 201 men exposed to dust in a cement mill. Morbidity data for 31 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 pneumoconiosis in 66 workers.

45. Dreessen, W. C., and R. R. 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. Lab. Stat. No. 231, pp. 176–180. 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. 1935.

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. Egbert, 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 fingerlike 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 59-year-old man employed 22½ years in the storage department of an asbestos factory. A neoplasm about the size of a golf ball was found in the pleural portion of the lower lobe of the left lung, continuous with the pleural growth