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SUBJECT INDEX TO TEXT AND BOOK REVIEWS. VOLUME 18

This is a subject index to all the reading matter in the JOURNAL OF INDUSTRIAL HYGIENE, and one should, therefore, look for the subject word. The name of the author follows the subject entry in parentheses. Items starred are book reviews.

For author index, see page 801.

PULMONARY ASBESTOSIS: INCIDENCE AND PROGNOSIS*

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WITHIN a comparatively short time the occupational disease, pulmonary asbestosis, has become a matter of considerable importance, not only to manufacturers of asbestos products, but also to the workmen engaged in the industry. That the serious industrial hazard of this type of work has not previously received sufficient attention is becoming more apparent. The manufacture of asbestos products has increased more than four-fold in the last 20 years, and hence, because of the greater number of workers exposed, and the more frequent recognition as a pathological entity of the resulting pulmonary condition, the subject of asbestosis has rapidly assumed greater importance.

It is indicated that the owners of asbestos plants, and the workers themselves, are beginning to realize that exposure to asbestos dust is a serious occupational hazard, and it also is apparent that these workers *must* be protected against the hazard as effectively as is possible. That this protection can be made complete seems very doubtful. It seems to be the opinion of engineers that the most effective types of suction apparatus now in use will remove a maximum of not over 90 per cent of the dust, but the remaining 10 per cent may be definitely injurious. In 1916 protective devices of the suction type were placed in a certain asbestos mill, and yet 15 years later cases of asbestosis with fatal termination were reported among workers from this plant. Since the most efficient type of protective machinery known apparently does not completely protect, although the number of workers affected is probably somewhat reduced, and the time required for the production of an advanced type of disease is probably lengthened, the problem becomes a matter for serious consideration.

In the case of any industrial employee presumed to be physically handicapped by any condition or disease, supposedly caused or aggravated by conditions under which he has worked, there enters the question of compensability. Pulmonary asbestosis has not as yet been widely recognized as a compensable disease in states having laws governing the compensation of workers. If such cases are viewed as compensable under the Industrial Compensation Laws, it will be necessary to decide the degree of disability in each individual in order that proper rating may be made.

*Received for publication January 2, 1936.

Read before the Industrial Hygiene Section of the American Public Health Association at the Sixty-fourth Annual Meeting in Milwaukee, Wis., Oct. 8, 1935.

Practically all writers on the subject are agreed that the principal symptom of the condition is a varying degree of dyspnea, and that the physical signs are those found in fibroid tuberculosis. Hence a definite diagnosis cannot be made by a physical examination alone. The x-ray film, however, is distinctive, in that it shows a picture of pathological changes in several particulars different from those found in the x-ray films of any other respiratory abnormality. The presence of the condition can be demonstrated by the x-ray film alone, but the degree of disability cannot be estimated unless the symptoms and physical signs are also considered. The dyspnea is usually out of proportion to other symptomatic manifestations, and is the one symptom which, in the more advanced cases, precludes any form of muscular exertion. For that reason, an asbestotic victim is unable to sell his labor on the open market, and must be rated as totally disabled.

I have seen no statistics in the literature in regard to the incidence of asbestosis among the workers in the industry. It would seem to be a matter of some importance to determine the average number of employees who may be now, or who are likely to become, disabled by the condition. I have lately had occasion to review the x-ray films of 151 workers in asbestos mills. Of these, fifty-two showed definite evidence of asbestosis in varying degrees. Of the 151, eighty-six had worked in asbestos for periods varying from 4 to 20 years, and in this group were found fifty-one of the positive cases, a percentage of 59.3. There was only one case with definite x-ray evidence of asbestosis who had worked less than 4 years in the industry. Of the 52 workers with evidence of asbestosis only four had worked less than 5 years, forty-eight having been employed for 5 years or more. There were eleven with an advanced type, no one of whom had worked less than 8 years. One of these advanced cases had been exposed to asbestos dust for 8 years, two for 9, and one each for 10, 11, 12, 14, 15, 16, 17 and 20 years.

Unfortunately I have no record of the length of service of these employees in the various departments of the mills, all of which are unquestionably dusty, but it is agreed that the carding room is the dustiest, and the weave room probably next. It is presumed that the dustier the work, the more likelihood there is that pathological changes in the lungs may result. It is noteworthy, however, that some employees work in asbestos plants for years without suffering any pathological changes in the lungs, as far as can be detected in the x-ray films.

In this series of 151 employees, ninety-nine showed no positive evidence of asbestosis in the films. Five had worked in asbestos for 6 years, three for 7, three for 8, four for 9, six for 10, one for 11, and one for 15 years. All the others had worked for periods varying from a few months to 5 years. Since the percentage of those in the series who were affected was so high, it seems remarkable that so many could be entirely free from the condition even after years of exposure. Since this process is non-infectious, the term "physical resistance to disease" is not applicable. It is true that muscular development avails

nothing as a protection against the disease, since well developed men are as frequently affected as those not so well developed. It is also possible that some individuals may be susceptible even though exposed to low concentrations of dust.

It is the opinion of some writers that the inhalation of asbestos dust and the consequent production of asbestosis has a tendency to exacerbate old tuberculous lesions. This opinion is apparently not universal. Merewether (1) says "from a review of 42 fatal cases of confirmed asbestosis it appears that asbestosis . . . is less frequently accompanied by tuberculosis than is silicosis." Gardner and Cummings (2) state that "primary tuberculous infection is influenced only to a limited degree by inhaled asbestos." Egbert (3), in an analysis of 28 cases of pulmonary asbestosis with fatal termination as reported in the literature, found that tuberculosis was present in only 6 cases of the 28, and in only 3 instances was death due primarily to tuberculosis.

It is universally agreed that silicosis does tend to exacerbate old tuberculous lesions. In fact more or less rapidly progressive tuberculosis is considered the most frequent and most serious complication of silicosis. Boisliniere (4) says that silica is the only phthisis-producing dust. In observations for several years I have been unable to find evidence that the inhalation of asbestos dust, or the condition asbestosis itself, has any tendency to render active old, apparently healed tuberculous lesions. In this series of 151 workers there were three who had a definite asbestosis in addition to an apparently healed tuberculosis. One had been working in asbestos for 10 years, another for 4 years, and the third for 2 years. There was no evidence in the films that the presence of asbestosis had yet had any tendency to activate the tuberculous lesion. The films of 23 workers showed evidence of tuberculous infection without any pathological changes indicating asbestosis. Of these, eleven showed healed childhood type tuberculosis, but none of these had worked longer than 15 months in asbestos. Two workers showed healed childhood type tuberculosis with no asbestosis, one of whom had spent 8 years in the work and the other 6 years. There were 5 films showing apparently arrested adult type disease with no asbestosis. These films were of employees who had spent the following periods in this work: One each for 4, 6, 7, and 9 years; and one, no term of service given. There was 1 case with an apparently healed and calcified miliary tuberculosis with a history of only 8 months' service in the industry.

In the 151 films there were only four which, from an x-ray standpoint, were diagnosed as probably active tuberculosis of the adult type. One of these had been in service for 5 months, and one each for 15 months, for 6 years, and for 10 years. Since 1 case had worked only 5 months and another 15 months, it would seem improbable that this type of work could be the cause of their active disease, particularly as the films of both cases indicated a chronic type of disease and apparently not an acute exacerbation. There remain for consideration 2 active cases with 6 and 10 years' service respectively. Agreeing that

the work in asbestos may have aggravated their pulmonary condition, 2 cases of active tuberculosis in 151 workers seems a decidedly low percentage, if this type of industrial occupation has any tendency *per se* to exacerbate old tuberculous lesions. Tuberculosis of an active type may be found quite as frequently in the routine examination of workers in any industry, regardless of whether or not the occupation be dusty.

Case I. The x-ray films of one man who has worked approximately 10 years in asbestos illustrates the fact that employment in this industry even for a long time does not tend always to re-activate old tuberculous lesions. The first film, taken April 2, 1930, shows some fine fibrosis on both sides from the 6th to the 9th posterior ribs, thickened interlobular pleura with some involvement of the diaphragmatic pleura, and a slightly "shaggy" heart outline, these pathological changes indicating a probable asbestosis which is not very far advanced. In addition to this condition the film shows a tuberculous infiltration in the right apex, which appears to be in a quiescent state. The second film, taken May 3, 1935, indicates that the tuberculous lesion still remains inactive, although a period of 5 years has elapsed since the previous film was taken. This man left the asbestos industry for a while during this period, but returned to it about 2 years ago.

One of the most important problems in the consideration of pulmonary asbestosis is whether or not the disease is progressive, even after an individual handicapped by it ceases to work in the industry. Information on this question is of considerable moment in rating the eventual degree of disability of a worker, either in the assessment of damages in civil suits or in claims under State Compensation Laws.

Sparks (5) states that the disease is apparently progressive, not even the cessation of exposure to the dust checking its spread. Wood and Gloyne (6) state that once asbestosis bodies are found in the sputum the course of the disease appears progressively downward, and cessation of exposure then does not check the spread.

Many writers consider that silicosis is a progressive disease, regardless of whether or not there has been cessation of exposure. However, Sayers (7) says that "a man suffering from simple silicosis generally improves when removed from the dusty atmosphere and placed in suitable surroundings." Boisliniere (4) says: "Silicosis by no means always progresses after the hazard has ceased, nor does it always produce tuberculosis."

It is reasonable to assume that the eventual effect of the inhalation of a dust which sometimes contains as much as 99 per cent silica might be somewhat more severe than that caused by a dust which contains a maximum of only 2.6 per cent. It seems to be true, however, that the fibrosis in asbestosis is produced by a much shorter exposure than is the rule in silicosis. Gardner and Cummings (2) say: "The particles and elongated fibers of asbestos dust do not, during this period (2 years and 5 months), penetrate so deeply into the air passages of the lung as do the other dusts previously studied. The major portion is held up and phagocytosed in the lumina of respiratory

bronchioles and the alveoli given off from their walls." Since the condition is not an infectious process, it should not continue to progress on removal of the causative factor. Gardner and Cummings (2) state that the asbestosis body and the production of fibrous tissue in the lung is caused by hydrolysis of asbestos fibers in the tissues. Theoretically, then, extension of the pathological process should continue until all asbestos fibers remaining in the lung are hydrolyzed, which may require a considerable time.

Several of my cases seem to indicate that the rapidity of progress of the fibrotic change in the lungs after cessation of exposure is dependent to some extent on the amount of involvement which occurred before the individual ceased work. To illustrate this point, consideration of additional x-ray films of 3 cases reported in 1933 may be of interest.

Case II. D. S. W., white male. Ceased work in card room of asbestos plant in January, 1929, after working only 18 months. The first film was taken June 13, 1931, and shows a fairly extensive fine fibrosis, a "shaggy" heart outline and some involvement of the pleura. The second film was taken on March 8, 1935, approximately 4 years later. Comparison of the two films indicates that in the 4 year interval the densities at the hila have increased noticeably, the pulmonary fibrosis has extended toward the apices, and the hypertrophy of both the right and left heart has increased. Symptomatically, the dyspnea has noticeably increased within the last year. The man has not been exposed to asbestos dust since January, 1929.

Case III. C. A. B., white male. Quit his work in the card room of an asbestos plant in June, 1931, after having worked continuously for 8 years. The first film was taken June 13, 1931; the second film, January 30, 1935. The first film shows a bilateral fibrosis over the lower two-thirds of the lungs, the usual "shaggy" heart outline and adhesive pleurisy. The case was diagnosed well-advanced asbestosis. The last film shows an extensive progression of the condition, the fibrosis having extended into the apices, and also become more dense. In spite of the fact that this man has had no exposure for a period of 3½ years, the condition has steadily become more extensive. At the time this is written he has begun to show symptoms of progressive heart failure, and a fatal termination seems inevitable.

Case IV. R. G. J., white male. Quit his work in the card room of an asbestos plant in April, 1930. The first film was taken April 23, 1930, and shows fine fibrosis and mottling over the lower lung fields with some "shagginess" of the heart outline and pleural adhesions. The next film, taken September 2, 1931, 16 months after the first film, shows some increase in the pulmonary fibrosis. This man, under rest treatment, improved his general physical condition materially. He was advised not to return to work in an asbestos plant, but he did not heed this advice because he felt that he must make an attempt to care for his family. He started to work again in May, 1933, and continued until October, 1934, when he was forced again to leave his job. The final film, taken August 30, 1934,

indicates extensive progression of the fibrosis well up into the apices, and a definitely hopeless outlook. In fact this man is beginning to manifest the physical signs and symptoms of progressive cardiac failure, which, in my opinion, is the terminal result most frequently met with in pulmonary asbestosis.

The rate of progress of the condition in Case II, and the extensive progress in Case III, even though there was cessation of exposure in both cases, qualifies the statement of Wood and Gloyne (6) that "once asbestosis bodies are found in the sputum the course of the disease appears progressively downward." Asbestosis bodies were not found in the sputum of Case II, but were found in that of Case III. The sputum of Case IV was at first negative for the bodies, but they are present since the condition has become far advanced. Incidentally careful examination of the sputum of cases with definite asbestotic involvement in the lungs frequently fails to exhibit asbestosis bodies.

The high percentage of pulmonary asbestosis (34.4) found in this series of 151 workers, indicates unquestionably the need for more complete protection of employees in asbestos mills. Sufficient evidence has been produced to prove that the inhalation of asbestos dust is productive of serious impairment of health. In fact, the victim of asbestosis, as a rule, eventually becomes totally disabled from engaging in any form of labor. The most prominent and lasting symptom of this disease is dyspnea, and this is out of all proportion not only to the physical signs found in the lungs, but also to the pathological changes depicted in the x-ray films. It is this symptom which very frequently precludes any form of physical exertion. Furthermore, even in those cases in which the x-ray films show a negligible increase in the pathological process over a period of years, there is no indicated improvement in the dyspnea. Serial x-ray films indicate that the condition is slowly progressive even when exposure has been discontinued for several years. The prognosis for extension of life after cessation of exposure in the cases in which involvement is not far advanced is encouraging, but the hope for amelioration of the dyspnea is not encouraging.

An industrial worker is entitled to every protection that may safeguard his health, so that he may earn a livelihood for himself and family for at least a reasonable period of years in the work in which he is most skilled. If he is prevented from continuing in such work because of impairment of health through no fault of his own, he is entitled to some remuneration for his loss of earning power. That protection of asbestos workers has been woefully lacking in the past has been definitely shown. It is imperative that such protection, as nearly complete as possible, be provided by mill owners. Efficient protective devices will be far less expensive in the final check up than the aggregate of numerous claims for compensation and frequent damage suits. That complete protection can be afforded by the devices in use at the present time seems to be somewhat doubtful, but workers are entitled to the highest type of protection which the engineers familiar with the hazard can provide.

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A SURVEY OF A GROUP OF EMPLOYEES EXPOSED TO ASBESTOS DUST*

S. B. McPHEETERS

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THE dangerous dust diseases, silicosis and asbestosis, are being widely studied at present. It has been estimated that there are more than 750,000 silicotics in the United States while there are only 35,000 to 40,000 persons engaged in the asbestos industry in the world. In the United States in 1929 a total of 9,237 persons were said to be employed in the manufacture of asbestos products other than steam packing and pipe and boiler covering. While it employs a relatively small number of persons, the asbestos industry has had a remarkably rapid expansion and by reason of the widespread and varied uses of its products which include "matches, filter pads, paints, roofing, high pressure jointing, electrodes, brake linings, clutch rings and insulating material in a great variety" the industry occupies an important, increasing, and permanent place in our economic establishment.

Asbestos is described chemically as a hydrated silicate of magnesia containing little iron and almost no calcium. It is a fibrous mineral which can be split up into flexible fibers for spinning and weaving. When it is processed some of these fibers are fragmented. The fine fragments are inhaled by the workers. It is the particles smaller than 10 microns which are the cause of the distinctive pulmonary fibrosis called asbestosis. Gardner writes that "these asbestos particles are phagocytosed as they are caught on the irregular walls of the terminal bronchioles. The phagocytes for the most part migrate directly into the substance of the adjacent walls and there stimulate growth of connective tissue cells. There results a collar of fibrous tissue about the terminal bronchioles which contracts and constricts the tubes. The constriction produces collapse of the distal alveoli (atelectasis). The collapsed area becomes fibrous from mechanical causes (collapse induration). In asbestosis unlike silicosis there is little or no transport of dust into the lymphatic system and no nodule formation." "Intervening units (between the affected lobules) not being involved still contain air and permit the passage of x-rays. Consequently the roentgenogram fails to show massive homogeneous shadows." The appearance on the film has been frequently likened to ground glass.

This paper reports the results of a survey of 210 persons exposed to asbestos dust. The conditions imposed upon the survey did not permit the thoroughness of study to be desired, but all of the employees were given a brief, general clinical examination which included taking their past

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health and occupational history and a physical examination. No laboratory examinations were made. All had a single film made of the chest.

The dust concentrations are not equal in the various departments. In the preparation department the processes of grinding, crushing, and mixing are carried out. Here the quantity of dust is greatest and consists almost entirely of asbestos particles. In the other departments the material processed is a mixture of asbestos and cotton. Here the proportion of asbestos particles is less and the total dust is also less.

TABLE 1

	NUMBER	PER CENT
Total examined	210	
Normal lungs	123	58.8
Pulmonary fibrosis, linear type	9	4.3
Heavy linear shadows in the cardiophrenic angle	11	5.2
Cardiovascular pathology	24	11.4
Pleural adhesions	7	3.3
Azygos lobes	2	0.9
Bony pathology	4	1.9
Pleuro-pericardial adhesions	5	2.3
Bronchiectasis	3	1.4
Asbestosis questionable	20	9.5
Asbestosis moderate	25	11.8
Asbestosis advanced	8	3.8
Primary pulmonary tuberculosis:		
Primary foci	6	2.8
Calcified lymph nodes	5	2.3
Secondary pulmonary tuberculosis:		
Minimal	2	0.9
Moderately advanced	4	1.9
Far advanced	0	0.0

Table 1 summarizes the various types of pathology noted in the films. Of the 210 persons examined, forty-five were negroes. The films of one hundred and twenty-three, 58.5 per cent, were regarded as showing lung-fields that were normal, except for a small primary tuberculous focus in one. Of the 210 films, fifty-three, 25.2 per cent, were regarded as showing definite or questionable evidence of asbestosis; of the fifty-three, 20 films were classed as questionable. Had stereoscopic retakes been allowed, it is believed that this number could have been greatly reduced. The indispensability of a competent roentgenologic technic needs to be emphasized. Thirty-three films, 15.7 per cent, showed definite asbestosis; in twenty-five, 11.8 per cent, the process was moderate in extent; and in eight, 3.8 per cent, more advanced. In the remaining 31 films various types of thoracic pathology were noted, none of which were ascribable to asbestos dust.

Table 2 presents the normal lung group and shows the distribution of the group in the various age-periods; the incidence of cardiovascular abnormalities seen in the films and dyspnea according to age-periods; the number with chest expansions averaging approximately ½ inch, 1½ inches, 2½ inches and 3½ inches; the total number of employees in each department whose lungs were normal, and the numbers and lengths of exposure to asbestos dust of those in the several departments.

Table 3 shows the incidence and extent of asbestosis from increasing periods of exposure in the several departments. In this table employees are placed in the department where they work at present. It is obviously a practical impossibility to trace the transfer of each individual from department to department and as the

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ANALYSIS OF GROUP OF

	15-24 years
Number in each age period	36
Dyspnea	1
Cardiovascular abnormalities	
Chest expansion	0-1
Number in each grade	2
Years of exposure	1-2
Departments:	
Preparation	4
Carding	6
Mule spinning	5
Spooling	3
Twisting	11
Winding	19
Ring spinning	1
Weaving	8
Maintenance	4
Total	61

escaped asbestosis; of 32 persons
posed for more than 10 years
similar concentrations, fourteen,
per cent, escaped; of 70 persons
exposed for more than 5 years fo
five, 64 per cent, escaped, and of
persons exposed for more than 2 y
and less than 5, twenty-four, 85
cent, escaped. Prolonged exposur
disease-producing concentrations
dust alone does not seem to have

management states that something like 95 per cent of employees continue in the department in which they began, it has been assumed in the discussion that the employee has been at his present work throughout his exposure.

From this analysis it appears that of 9 persons exposed for more than 15 years to dust concentrations effective in producing disease, four, 44 per cent, escaped asbestosis; of 32 persons exposed for more than 10 years to similar concentrations, fourteen, 43 per cent, escaped; of 70 persons so exposed for more than 5 years forty-five, 64 per cent, escaped, and of 28 persons exposed for more than 2 years and less than 5, twenty-four, 85 per cent, escaped. Prolonged exposure to disease-producing concentrations of dust alone does not seem to have been sufficient in a great percentage of these cases to produce asbestosis. Attention is called to the fact that in the same department, that is, where the concentration of dust is the same, some exhibit the disease after a short exposure while others, after much longer exposures, escape. Nor is the incidence of disease always as great among those who have been exposed for equal periods in departments where concentration of dust is greater as among those who work in departments where it is less.

The incidence of disease increases with the duration of exposure and the concentration of effective dust, but not directly or constantly. No length of exposure or concentration of dust produced the disease in more than 60 per cent of persons exposed in any

TABLE 2

ANALYSIS OF GROUP OF EMPLOYEES WITH NORMAL LUNGS

	AGE					TOTAL
	15-24 years	25-34 years	35-44 years	45-54 years	55-64 years	
Number in each age period	36	57	22	6	2	123
Dyspnea	1	6	1		1	9
Cardiovascular abnormalities		2	3	2	2	9
Chest expansion	0-1	1-2 in.	2-3 in.	3-4 in.	Not recorded	
Number in each grade	2	67	48	3	3	123
Years of exposure	1-2	2-5	5-10	10-15	15-20	
Departments:						
Preparation	4		1			5
Carding	6	4	4	2		16
Mule spinning	5	1	4	1	2	13
Spooling	3	2	4	1		10
Twisting	11	1	5	1		18
Winding	19	1	7	3		30
Ring spinning	1			1		2
Weaving	8	2	8	1	1	20
Maintenance	4	3	2			9
Total	61	14	35	10	3	123

department. It seems to be true everywhere, that some workers are not affected however long the exposure or high the concentration. These facts suggest differences in susceptibility. As to what deficiencies in defense or possible reinforcements of the attack exist in those in whom asbestosis occurs, other than the recognized difference in the competency of the protective mechanisms in different individuals, no conjecture is ventured.

TABLE 3

ANALYSIS OF EFFECT AND DURATION OF EXPOSURE IN VARIOUS DEPARTMENTS

DEPARTMENTS	DURATION OF EXPOSURE																								TOTAL
	0-2 years					3-5 years					5-10 years					10-15 years					15-20 years					
	Number of employees	No asbestosis	Questionable	Moderate	Advanced	Number of employees	No asbestosis	Questionable	Moderate	Advanced	Number of employees	No asbestosis	Questionable	Moderate	Advanced	Number of employees	No asbestosis	Questionable	Moderate	Advanced	Number of employees	No asbestosis	Questionable	Moderate	Advanced	
Preparation	3	3									7	2	3	2		1				1						3
Carding	8	7	1			8	6		2		13	5	1	3	4	7	1	3	2	1	1		1			12
Mule spinning	5	5				3	3				8	6		2		3	2		1		4	2		2		5
Spooling	3	3				2	2				6	5		1		3	2	1								1
Twisting	15	15				1	1				9	7	1	1		3	2		1		1		1			2
Winding	20	20				3	3				9	8	1			6	4	1	1							1
Ring spinning	1	1				1	1				1			1		1	1									1
Weaving	10	10				5	4	1			12	9	1	2		5	1	1	3		1	1				5
Maintenance	6	6				5	4	1			5	3	1		1	3	1	1	1		2	1			1	3
Total	71	70	1			28	24	2	2		70	45	8	12	5	32	14	7	9	2	9	4	2	2	1	33
Number with dyspnea		1					3					7	2	3	2		2	3							1	24
Cardiovascular disease		3	1				2					2	4	2	1		2	5	1	1						24

As of silicosis, dyspnea may be said to be the characteristic symptom of asbestosis. The dyspnea here reported does not refer to more frequent, more forceful or embarrassed operations of the respiratory mechanisms objectively manifest. No tests were made to observe the behavior of respiration in response to the changing ventilatory demands of graded physical exercise. Dyspnea here means that the persons replied "yes" when asked, "Do you have any shortness of breath under ordinary circumstances?" This "yes" is understood as meaning that these persons when engaged in usual activities were with greater or less frequency conscious of an unaccustomed sense of inadequacy attending the ventilatory process. This experience was not infrequent among persons with lungs roentgenologically and by physical examination normal, occurring in 8.1 per cent. The experience was more than twice as frequent among those with definite asbestosis. Six of the 33 asbestotics

(18.1 per cent), stated that they h
some shortness of breath; three of
25 moderate cases and three of th
more advanced. But the most
vanced cases showed no special resp
tory effort during examination

FIG. 1.—Moderate asbestosis. He

no cyanosis and none declared
of or impaired ability to carry o
their jobs. Among the thirty-t
asbestotics, dyspnea was twic
frequent in those under 45 yea
age, 42.1 per cent, as in those
45 years of age (21.4 per cent).

(18.1 per cent); stated that they had some shortness of breath; three of the 25 moderate cases and three of the 8 more advanced. But the most advanced cases showed no special respiratory effort during examination and

FIG. 1.—Moderate asbestosis. Heavy pectoral muscles exaggerate the appearance of fibrosis.

no cyanosis and none declared lack of or impaired ability to carry on at their jobs. Among the thirty-three asbestotics, dyspnea was twice as frequent in those under 45 years of age, 42.1 per cent, as in those over 45 years of age (21.4 per cent). Five of the twenty-four with dyspnea showed cardiovascular pathology in the films and also had high blood pressure, but none of the five had definite asbestosis. Dr. William S. McCann reminds us: "Respiration is not a function of the lungs alone but of the heart, blood and tissue ability to absorb oxygen, and also to undergo oxygen want such as the trained muscles of athletes withstand oxygen want."

Decreased chest expansion may be

said to be the cardinal sign of asbestosis. Among the normal lung group, 56 per cent had an expansion under 2 inches, 44 per cent above 2 inches. In the group with definite asbestosis, 69.6 per cent, had a chest expansion of less than 2 inches, 30.4 per cent, had an expansion above 2 inches. The proportion of those with the lower degree of expansion was increased about 24 per cent among the asbestotics. Chest expansion depends not only on lung distensibility but on the mobility of the bony and soft tissues of the thoracic cage and of the diaphragm. It is often astonishing how training and practice can rapidly increase chest expansion. The individual learns to exercise fully anatomic capacities which were present prior to the training.

From the foregoing findings it appears that the assumption that a sense of air want and restricted chest expansion are due to an existing asbestotic involvement of the lungs roentgenologically manifest, should not be made too hastily.

Studying the compatibility of asbestosis with length of life and work it is to be noted that: One employee with advanced asbestosis above the age of 55 who had worked in dust more than 15 years was still able to work. One employee, 54 years old, with advanced asbestosis who had worked in dust more than 10 years was still able to work. Three employees with moderate asbestosis and above the age of 44 who had worked in dust more than 10 years were still able to work. Of this group of 5 asbestotics above the age of 44 (three with moderate asbestosis and two with advanced asbestosis), one complained of shortness of breath. Of the eleven with moderate asbestosis and the three with advanced asbestosis who had worked more than 10 years, one, (7.1 per cent), complained of shortness of breath; these fourteen men had chest expansions which compared favorably with the normal lung group. Sixty-six and two-thirds per cent of these 14 asbestotics had a chest expansion under 2 inches, whereas 56 per cent of the normal lung group had the same limits. But 78.9 per cent of those with definite asbestosis who had worked in dust under 10 years had chest expansion under 2 inches and 26.3 per cent complained of shortness of breath. The older persons, whether with moderate or more advanced asbestosis, appear better adjusted to the disease.

The average exposure for the entire group with asbestosis was 10.2 years, for those with moderate asbestosis 10.3 years, and for those with advanced asbestosis, paradoxically, 10.0 years. If the entire group with asbestosis is divided into those below and those above 40 years of age, the exposure of those with moderate asbestosis below 40 years of age was 9.5 years and above 40 years of age, 12.4 years. For those with advanced asbestosis the corresponding averages were 9 and 11 years respectively.

Advanced asbestosis may exist in late age-periods with working ability not impaired below the level of continued employability in the department where the disease was incurred. No very rapid development of the disease was noted. Only 2 cases occurred where the exposure had been less than 5 years. As a rule 5 or more years of exposure was required

to cause the disease. No
erate or massive lesions such
in silicosis where infection is
posed were found in these a
cases. The progression of th
in this group was very slow.

FIG. 2.—Advanced asbestos

The evidence presented as
effect of asbestosis upon tuber
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Apical tuberculous infiltratio
noted in the films of 17 emp
there was no other evidence of tu

to cause the disease. No conglomerate or massive lesions such as occur in silicosis where infection is superimposed were found in these asbestosis cases. The progression of the disease in this group was very slow.

losis seen in these films. If it can be assumed that most of these infiltrations formed in the late 'teen age or before 25 years of age, then exposures to asbestos dust varying from 5 to 15 years had produced no evidences of

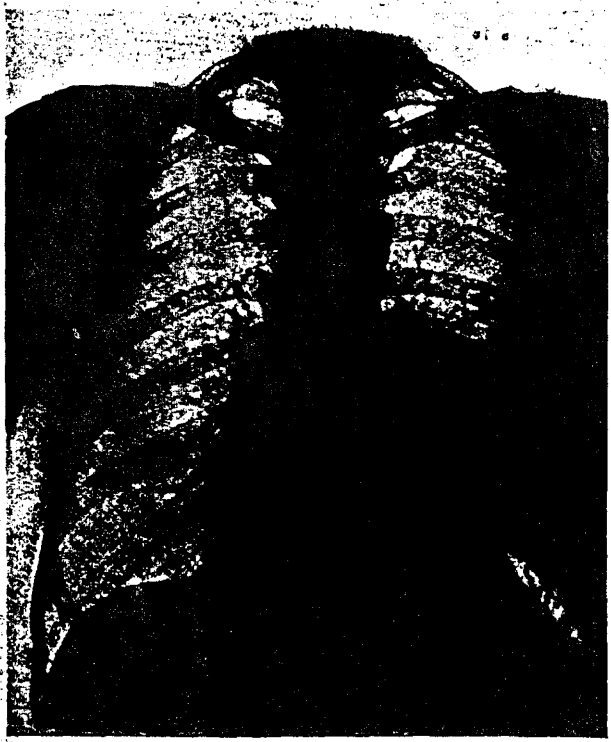

FIG. 2.—Advanced asbestosis, emphasized at left base. Upper lung fields emphysematous.

The evidence presented as to the effect of asbestosis upon tuberculosis in this survey is somewhat limited. Apical tuberculous infiltration was noted in the films of 17 employees; there was no other evidence of tuberculous activation to be seen in the films. Four of these apical tuberculous infiltrations were found in asbestotics, two with moderate and two with more advanced processes. Six lesions with clinical significance were observed: 2

minimal and 4 moderately advanced (American Sanatorium Association Classification). One moderately advanced lesion was found in a man 24 years old who had been exposed to asbestos dust in the winding department for 6 years. The lesion appeared "soft" and unstable; there was, however, no evidence of accompanying asbestosis. The disease in this moderately advanced case was clinically manifest, though the employee regarded himself as in good health. One of the moderately advanced lesions was found in an employee 37 years of age who had worked in the winding department for 9 years and showed no evidence of asbestosis. A third employee with a moderately advanced tuberculous lesion had worked in the twisting department for 10 years but had no asbestosis. The fourth, an employee 37 years of age who had worked in the weaving department had moderate asbesto

FIG. 3.—Lateral position. (Same case as Fig. 2.)

The tuberculous lesion in this c could satisfy rigid criteria of a hea lesion. Three of these lesions w active and were accompanied by cl cal manifestations. The one les associated with asbestosis was hea and the employee apparently wa good health. One of the two mini lesions was in a man 24 years of who had worked 15 months in twisting department and whose f did not show asbestosis; one in a 37 years of age who had worke years in the winding department

GROUP OF 24 EMPLOYEES WITH AB

	15
Number of employees in each age-period	
Dyspnea	
Hypertension	
Years of exposure	
Number of employees	
Asbestosis	

had no asbestosis. One of minimal lesions was apparently h

It is felt that no evidence aggravating effect on tuberculo asbestos dusting is to be fou these cases. Only one of th clinically significant lesions wa companied by asbestosis and lesion had every appearance of healed. The incidence of pulm tuberculosis (2.8 per cent), is reg as comparable to the incide many other industrial groups in the association is close and pro and whose general standards of and hygiene are not as good as

partment had moderate asbestosis. The tuberculous lesion in this case could satisfy rigid criteria of a healed lesion. Three of these lesions were active and were accompanied by clinical manifestations. The one lesion associated with asbestosis was healed and the employee apparently was in good health. One of the two minimal lesions was in a man 24 years of age who had worked 15 months in the twisting department and whose films did not show asbestosis; one in a man 37 years of age who had worked 12 years in the winding department and had no asbestosis. One of these minimal lesions was apparently healed.

It is felt that no evidence of an aggravating effect on tuberculosis by asbestos dusting is to be found in these cases. Only one of the six clinically significant lesions was accompanied by asbestosis and that lesion had every appearance of being healed. The incidence of pulmonary tuberculosis (2.8 per cent), is regarded as comparable to the incidence in many other industrial groups in which the association is close and prolonged and whose general standards of living and hygiene are not as good as might

The films were studied to discover any accentuating effect of a focus of tuberculosis on the surrounding asbestosis. No convincing evidence of such an effect was seen. Tuberculosis and asbestosis were coincident in only 5 employees. The average duration of exposure for the five was 10.4 years and for the entire group with asbestosis 10.2 years. No accelerating effect of tuberculosis on asbestosis could be inferred.

Twenty-four films showed abnormalities of the heart and aortic shadows (Table 4) and eleven of the twenty-four abnormalities were found in the 45 negro employees. A Wasserman test was not allowed. Ten of these negroes were under 45 years of age and one over 55 years of age. In this group of 24 employees with cardiovascular changes, three had a normal blood pressure and the rest had hypertension. Five of the group with cardiovascular abnormalities had definite asbestosis, three of these were negroes under 35 years of age (Table 5).

Waring and Black in an admirable paper discuss, "The Syndrome of Obstruction in the Lesser Circulation" (Am. J. Med. Sci. May 1934) and

TABLE 4

GROUP OF 24 EMPLOYEES WITH ABNORMALITIES OF THE HEART OR AORTIC SHADOW

	AGE				
	15-24 years	25-34 years	35-44 years	45-54 years	55-60 years
Number of employees in each age-period	0	7	9	5	3
Dyspnea	0	1	2	1	1
Hypertension	0	6	8	4	3
Years of exposure	0-2	2-5	5-10	10-15	15-20
Number of employees	4	2	9	9	
Asbestosis			3	2	

give as its clinical features, cyanosis, dyspnea, polycythemia, and right ventricular preponderance.

The blood was not examined in the cases here described and no electrocardiograms were made. Cyanosis and dyspnea were not observed in any of the 5 asbestotics with cardiovascular disease. In view of the pathology characteristic of asbestosis, namely, constriction of the terminal bronchioles with no perilymphatic or perivascular fibrosis as in silicosis, less obstruction of the lesser circulation would seem to be expected in asbestosis than in silicosis and consequently less strain on the right heart.

The frequency of respiratory infections was investigated. Each employee was asked, "Have you had severe colds, 'flu,' or pneumonia and how often?" None said they had had pneumonia, six said they had had "flu," eight mentioned having had colds since being exposed to asbestos dust, and one spoke of having frequent colds. The reported respiratory infections in the group does not appear to be excessive.

To make a scientific appraisal of the effect on the employees in this plant of exposure to asbestos dust, it is necessary to know how many employees during the 15 to 20 years covered in the study have died or quit and how many of these had asbestosis. This knowledge is not available. Certainly it might add greatly to the gravity of the picture. The most that could be done was to note the effect on those now present in the plant, after varying periods of exposure.

TABLE 5

ASBESTOSIS WITH CARDIOVASCULAR LESIONS

	EMPLOYEE NO. 61	EMPLOYEE NO. 92	EMPLOYEE NO. 131	EMPLOYEE NO. 138	EMPLOYEE NO. 167
Age	44	53	27	30	30
Race	White	White	Black	Black	Black
Blood pressure	174/96	170/110	158/104	130/92	134/90
Years of exposure	13	8	7	10	7
Asbestosis	Moderate	Advanced	Moderate	Advanced	Moderate
Dyspnea	No	No	No	No	No
Chest expansion	2.5 in.	1.5 in.	1.5 in.	1.5 in.	1.5 in.
X-ray pathology	Enlarged aortic shadow	Enlarged aortic shadow. Enlarged left ventricle	Enlarged aortic shadow. Enlarged right and left ventricles	Enlarged heart shadow. Both ventricles enlarged	Enlarged aortic shadow

SUMMARY

1. In addition to duration of exposure and concentration of effective dust, it appears that individual response to the inhalation of asbestos dust is an important factor in the production of asbestosis.

2. Asbestosis is infrequently found before 5 years of exposure to industrial concentrations of asbestos dust. In

this group even under du
continued 15 years or more,
did not progress to an
involvement attended by m
cal manifestations or disabi

3. Roentgenologically a
bestosis was not incomp
ability to work at the proce
it was incurred. Contin
entailing exposure to diseas
concentrations of asbestos
increases the extent of fibr
is to be advised against.

4. The evidence present
study warrants the opinion
the reduction of dust con
now feasible by available
inating devices, the incide

this group even under dusting which continued 15 years or more, the disease did not progress to an extent of involvement attended by marked clinical manifestations or disability.

3. Roentgenologically advanced asbestosis was not incompatible with ability to work at the process in which it was incurred. Continued work entailing exposure to disease producing concentrations of asbestos dust slowly increases the extent of fibrosis and so is to be advised against.

4. The evidence presented in this study warrants the opinion that with the reduction of dust concentration now feasible by available dust eliminating devices, the incidence of significant asbestosis would in time approach the vanishing point.

5. No activating or aggravating effect of asbestos dust or asbestosis on tuberculosis was observed.

6. The evidence of this study, as to the effect of asbestosis on the lesser circulation, is inadequate for forming more than impressions. It does not suggest any marked tendency on the part of asbestosis, with the degree of involvement observed to obstruct the lesser circulation.

7. No increase of respiratory infections was reported in this group.

8. There is some evidence which suggests that asbestosis develops more rapidly in younger persons than in older.

motely resembling those of silicosis by injecting sericite." The authors realize that their results "may not exactly represent the pathological changes produced by the inhalation of sericite by miners" but they feel that the results may be useful for the comparisons which are made possible between quartz, sericite, and other dusts.—*Philip Drinker.*

FAMILIAL DISPOSITION TO SILICOSIS. *I. Lochtemper. Aerztl. Sachverständ.-Zeitung, vol. 41, pp. 174–175 (1935).*

The author writes of a family several members of which contracted silicosis shortly after working in the same industry, where they were exposed to quartz dust. Members of another family, on the other hand, showed only light cases of silicosis, although they had worked some years under the same conditions. The author draws attention to the fact that there may be a family disposition to silicosis, although he cannot assert that this is true.—*L. Teleky.*

EPIDEMIOLOGICAL ASPECTS OF SILICOSIS AND TUBERCULOSIS. *A. S. Pope and D. Zacks. Am. Rev. Tuber., vol. 32, pp. 229–242 (1935).*

1. In representative groups of both granite- and foundry-workers in Massachusetts the frequency of silicosis, and of silicosis with tuberculosis, was found to be positively correlated with the duration of exposure to dusts containing free silica and to the concentration of such dusts in the occupational environment.

2. Among the granite workers examined, silicosis alone was found in 15.2%, and silicosis complicated with tuberculosis in 7.6%.

3. Tuberculosis is the cause of death in over one-third of all granite-workers, a proportionate mortality three times that in foundry-workers, and four times that in all males of 20 years and over.

4. Among the employees of 10 Massachusetts foundries, silicosis was found in 8.8% and silicosis with tuberculosis in 2.6%, a total frequency of one-half that found in granite-workers.

5. The silicosis found in foundrymen was not only less frequent but also less advanced in degree than that in granite-workers.

6. Though there is an excess of tuberculosis deaths among foundry-workers, pneumonia, which is the cause of about one-fourth of all deaths of foundrymen, appears to be the greatest occupational hazard in the industry.—*Authors' summary.*

TWO CASES OF SQUAMOUS CARCINOMA OF THE LUNG OCCURRING IN ASBESTOSIS. *S. R. Gloyne. Tubercle, pp. 5–10 (Oct., 1935).*

The two cases described were women workers in an asbestos factory. One, aged 35, had been a spinner for 8 years and survived for further 9 years. The other, aged 71, worked only for two periods of 6 and 13 months as a mattress maker and lived for 15 years afterwards. No etiological association between the carcinoma and the asbestosis is claimed. The malignant lesions were small and were not recognized during life. They were circumscribed with no extension to important structures; no secondary deposits were present. The asbestosis was fairly advanced and of long standing; pigment, asbestos fibres, and asbestosis bodies were pushed aside by the advancing growth. Neither disease seemed to be sufficiently advanced to have caused death by itself; the combination was fatal.—*E. L. Collis.*

LUNG FUNCTION AND X-RAY PICTURES IN THE LEGAL ESTIMATION OF LUNG DAMAGE FROM DUSTS IN COMPENSATION CASES. *W. Schmidt, E. Gaubatz, and Holtzmann. Aerztl. Sachverständ.-Zeitung, vol. 41, pp. 178–182 (1935).*

The authors have investigated lung damage from dust inhalation by procedures other than x-ray. Lung function was determined spirometrically, the apneic pause and residual air was determined and work tests were performed using an ergometer. Vital capacity alone was found insufficient for judging functional disability but the apneic pause and residual air gave a convenient objective scale for judging emphysema while the work studies gave useful data on the heart action. It was established that working capacity and disability could not be judged by x-ray alone—facts which are in general acceptance.—*L. Teleky.*

REVIEW OF LITERATURE ON BREATHING DUSTS WITH SPE ENCE TO SILICOSIS, PART III *rington and S. J. Davenport. Mines, I. C. 6857, (Oct., 1935)*

In this Information Circular deal with the economic and leg dust disease in industry, and chosen list of references.

A summary and general con complete the series and it is that this valuable information in book form.—*H. N. Lawson.*

DUST STORMS AND THEIR POSS ON HEALTH. *E. G. Brown, and R. L. Labourn. U. S. Repts., vol. 50, pp. 1369–138 1935).*

"1. The dust storms which l in the central west are the clim period of decreased rainfall, a l of vegetation, and high winds.

"2. Crop and livestock loss large. It is believed by aut although there has been mu

DUST, SMOKE, FUME

PRESIDENTIAL ADDRESS. *R. . de Smidt. J. Chem. Met. Africa, vol. 36, pp. 18–28 (A*

A review of the methods of tion and dust control in the S gold mines. The author em little progress was made unti was approached with the vie mine air must be treated in p same way as gassy mine air. should be controlled to prod dilution. Mine air filtration given highly satisfactory resu mines.

The author questions the w cation of cooling as a means the high temperature and hig deep mines. Dry methods o but face one serious obstacle wet methods of control are no the miners and the general pu mental to safe work in dusty will have to be educated to methods.—*T. Hatch.*

DUST HAZARDS AND THEIR EFFECTS

STUDIES ON THE ABSORPTION OF DUST IN THE RESPIRATORY PASSAGES. II. ABSORPTION OF MANGANESE DIOXIDE DUST. *O. Ehrismann. Ztschr. f. Hyg. u. Infectionskrank., vol. 117, p. 662 (1935).*

The author subjected rabbits to daily exposures of 4 hours, over a period of 3 months, to manganese dioxide concentrations of 10–20 mgs. per cubic meter or he gave them a maximum of 3 gms. of manganese dioxide by means of throat probe over a period of 33 weeks. He found increased amounts of manganese in the organs, though no more in the lungs than in the other organs; there was a decrease in the erythrocyte count and in the hemoglobin content. The white blood picture showed no changes. Studies with cats gave similar results. Thus, it was shown that inhaled and swallowed manganese behave similarly; pneumonia did not result from manganese dust inhalation.—*L. Teleky.*

ASBESTOSIS. *A. J. Lanza. J. A. M. A., vol. 106, pp. 368–69 (Feb. 1, 1936).*

As this paper is shorter it is necessarily less complete than that by Lanza, McConnell, and Fehnel (*These Absts., vol. 17, p. 55*). It is general in character, gives the history of asbestosis, the clinical picture, a brief discussion of the roentgenogram and of the pathology. Lanza estimates that there are about 10,000 men exposed to asbestos dusts in plants employing about 12,000. Merewether and Price estimated that there were about 2200 similarly exposed in England in 1930.—*Philip Drinker.*

THE ACTION OF SILICA. *C. P. McCord, H. Ainslee, J. Johnston, and R. L. Fleming. Indust. Med., vol. 5, pp. 17–20 (Jan., 1936).*

The often encountered assertion that co-existence of silica and alkali dust in industries constitutes extra hazardous work conditions is unproved, but not necessarily unfounded. Extensive investigations of the workers in six plants simultaneously exposed to silica and alkali dusts provided no evidence of an accelerator action by alkalies. The entire absence of silicosis suggests the possibility of an inhibitor action.

Animal experiments with guinea pigs in which the peritoneal cavities were injected one or more times with 0.1 or 0.2 gm. each of silica and alkali (sodium carbonate, sodium bicarbonate, magnesium carbonate, or limestone), yielded no results proving either an accelerator or inhibitor action of alkalies in the production of silica nodules. The presence of alkalies markedly increased the distribution of silicotic nodules throughout the abdominal cavity, which action is apparently solely due to the mechanical distribution of the silica by increased motions of abdominal organs, induced by alkali irritation.

The primary toxicity of silica and silica gel mixed with alkalies is much greater than is true for silica, silica gel or alkalies alone. —*Authors' summary.*

SILICOSIS AND SILICO-TUBERCULOSIS IN METALLURGICAL PLANTS. *M. Francesco. Med. del Lav., vol. 13, pp. 241–253 (Jul., 1935).*

The author describes and discusses 13 cases of silicosis or silico-tuberculosis in metal workers and points out necessary precautions in plants where the silicosis hazard exists.—*L. T. Fairhall.*

DUST, SMOKE, FUMES, AND VAPORS: THEIR DETERMINATION AND ELIMINATION

DETERMINATION OF PHOSGENE. *W. P. Yant, J. C. Olsen, H. H. Storch, J. B. Littlefield and L. Scheflan. Ind. and Eng. Chem., vol. 8, pp. 20–25 (Jan. 15, 1936).*

An article of comparatively recent publication (by Olsen and others) criticized the results previously reported by the Bureau of Mines in measuring phosgene in the gases from excelsior fires which were extinguished by carbon tetrachloride on the basis that the analytical method gave erroneously high values. This bureau, with the cooperation of the manufacturers of the carbon tetrachloride-type fire extinguisher, has repeated the investigation under a reproduction of the former conditions, except that a differ-

ent analytical method w
phosgene found in these sixt
in which excelsior fires were
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briefly discussed.

It is not the intention o
discourage the use of carb
type fire extinguishers, wh
for stopping incipient fire
determine the decompo
which may result from the
should be recognized and
ated.—*Authors' summary.*

THE DETERMINATION OF
QUANTITIES OF HYDROCY
PLICATIONS IN PLANTS
TOXICOLOGY. *M. T. I
Laffitte. Bull. soc. chi
pp. 1088–1096 (1935).*

One cc. of the slightly
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which is suspended a da
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2, p. 866). After standin
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starch or ammonia and i
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do not interfere; heart t
the hydrocyanic and ca
error.—*Chem. Abstr.*

lution of fibrosis in chronic silicosis, beginning with lymphatic obstruction followed by accumulation in the parenchyma of the lung and nodular fibrosis. In "acute silicosis" the usual order is reversed. In the case of asbestosis he inclines to the belief that the magnesium is leached out of the molecule of hydrous magnesium silicate and that the residual silica then produces silicosis indirectly. His experience with sericite indicates that this substance remains unaltered in the tissues for a period of 1 year without producing anything but a foreign body type of reaction. He is not yet willing to conclude that sericite is entirely innocuous. Tuberculosis, the usual cause of death in silicosis, is discussed only parenthetically; the author mentions the various theories suggested to explain its frequency.—*L. U. Gardner.*

SILICOSIS. SOME DIFFERENTIAL DIAGNOSTIC PROBLEMS. *E. Mayer and W. Grethman. Am. Rev. Tuberc., vol. 33, pp. 313–321 (Mar., 1936).*

It has been estimated that in the United States there are 500,000 to 1,000,000 people employed in occupations in which a silicosis hazard exists and that there are about 65,000 such persons in New York City. The disease is often unrecognized in the general hospital because clinicians are unfamiliar with it and its differential diagnosis is difficult.

The diagnosis of silicosis is based on occupational history, symptoms, physical examination and x-ray. Occupational history and x-ray are very important in the diagnosis.

The occupational history should be specific as to kind of exposure and length of time in each dusty occupation. It is necessary to establish a definite history of exposure.

The silicotic patient has few complaints; the most constant are dyspnea and cough. Fever is absent unless infection is present. There is great disproportion between the patient's complaints and the x-ray findings.

Extensive disease (silicosis) may be present and but few abnormal physical signs elicited. Diagnosis is really made by exclusion. The physical findings are common to general pulmonary fibrosis with emphysema. There is restricted diaphragmatic movement. There may be diminished or intensified breath sounds and hyperresonant areas. Râles are absent in pure silicosis which is a more consistent finding than in any other pulmonary disease.

The essential types of abnormal shadows seen in silicosis are (1) small discrete shadows, (2) linear strands and (3) homogeneous shadows of varying size, indicating the presence of silicotic nodules, thick fibrous trunks and conglomerate massive fibrosis, respectively. Infection (tuberculosis) is considered to be present when the shadows begin to lose their sharp outline and to coalesce. Compensatory emphysema adds to the difficulty in making a diagnosis.

Four case histories and x-rays are presented.

The diagnosis of silicosis is often difficult because it is frequently impossible to obtain an accurate history from a patient in a general hospital. The physical signs and symptoms are inconclusive and frequently the x-ray is unreliable because of confusion with other pulmonary diseases. Silicosis not infrequently presents itself in atypical form.—*A. E. Russell.*

Asbestosis. *W. Aewens. Ztschr. f. Gewerbehyg. u. Unfall., vol. 43, pp. 1–6 (1936).*

The author reports on the lung examination of 35 employees of an asbestos plant. Thirteen workers gave negative x-ray results, 12 were doubtful first degree asbestosis, 7 showed first and second degree asbestosis, and 3 third degree; these last had worked in asbestos for 21–24 years; one of them, who had worked also with kieselguhr showed silicosis as well.—*L. Teleky.*

DUST, SMOKE, FUMES, AND VAPORS: THEIR DETERMINATION AND ELIMINATION

ATMOSPHERIC POLLUTION OF AMERICAN CITIES FOR THE YEARS 1931 TO 1933. *J. E. Ives, R. H. Britten, D. W. Armstrong, W. A. Gill, and F. H. Goldman. U. S. Pub. Health Bull. No. 224 (Mar., 1936).*

This study extended from June, 1931 to March, 1933 and included determinations in 14 cities of dust concentrations by count, shade intensity on white filter paper (Owen's automatic filter) and by weight.

The number of condensation
obtained. Continuous rec
tained with the automatic f
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except in Washington where
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It was found possible t
cities into three groups ac
level of atmospheric pollu
figures are given in the fol

GROUP	
I.	Baltimore, Boston, Chicago, Pittsburgh, St. Louis
II.	Buffalo, Cleveland, New Orleans, New York, Philadelphia
III.	Detroit, Los Angeles, San Francisco, Washington
	All cities

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rain was found to hav

CLOSIS: ROENTGENOLOGIC
THE DIFFERENTIAL DIAGNO-
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l chests and the close coöpera-
should exist between the
ist and clinican.—*N. De-*

IS PROBLEM: SOME MEDICAL
L PHASES. *O. Sanders. Wis-*
l. J., vol. 35, no. 5 (1936).
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He stresses the need for pre-
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yee; another copy of the exam-
s delivered by the employee to
yer, thereby fulfilling an ob-
o all parties concerned.—*N.*
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OF ARTIFICIAL ABRASIVES IN THE
INDUSTRY. *Fritz and Kremer.*
chutz (Reichsarbeitsblatt, Part
. 125–130 (1936).
tool and cutlery industry, 1929–
ut 1000 cases of pneumoconiosis
308 cases were compensated as
licosis of which 172 died. The
anization handling the insurance
ore recommended the substitution
al abrasive stones as they do not
osis whereas the natural ones do.

Against this the manufacturers of the natural stones appealed.

The authors, among the foremost industrial inspectors, explain that after overcoming the initial difficultiee, the introduction of artificial abrasives proved to be technically and economically satisfactory. They then appealed after an opinion in favor of the manufacturers of natural stones had been handed down, and showed that the artificial abrasives contained only 7.57% free silica, and that the dust generated by them consists mostly of magnesium oxides which, according to a decision of the Reich health authorities, is not to be considered as causing silicosis.—*L. Teleky.*

SILICOSIS IN SANDBLASTERS: THE EXAMINATION OF SANDS ASSOCIATED WITH SANDBLASTING. *F. S. Fowweather. J. Hyg., vol. 36, pp. 67–78 (1936).*

In sandblasting the sand is rapidly ground into a fine siliceous dust, whether used as the abrasive agent or introduced on the articles treated. The use of steel shot does not decrease the hazard if any sand is present. At least some of the dust is of a micaceous nature, in the form of sericite or a similar mineral. In spite of the opposite view of some, free silica dust is the major factor of sandblaster's silicosis.—*Chem. Abstr.*

ASBESTOSIS. *A. Bohne. Deutsch. med. Wchnschr., vol. 62, pp. 928–930 (1936).*

This is a short discussion of asbestosis, a description of a fatal case and data on the spread of asbestosis among the workers in an asbestos factory. An investigation showed that 4 of 14 workers showed manifestation of the disease after 1 year (the duration of work of the others is not given). —*L. Teleky.*

DUST, SMOKE, FUMES, AND VAPORS: THEIR DETERMINATION AND ELIMINATION

MEASUREMENT OF THE RADIATION TO WHICH ROENTGEN AND RADIUM WORKERS ARE EXPOSED. *I. C. Jacobsen and J. Ambrosen. Acta Radiologica, vol. 17, p. 252–255 (June, 1936).*

In modern roentgen installations with completely enclosed tubes the screening against stray radiation is very effective, but by carelessness the staff may be exposed to considerable irradiation. In the packing of radium applicators the danger is greater, since effective screening cannot be carried out.

This paper describes the results of observations made on roentgen and radium workers. The workers carried small condenser chambers while going about their work, and the charges in these condensers were afterwards measured. The measurements were carried out regularly over a period of 3 months. In the case of roentgen workers most of the readings fell in the interval of 0.02 to 0.03 *r* per day; for the packing of radium the results were roughly 5 to 10 times higher.—*T. Bedford.*

DETERMINATION OF HYDROCYANIC ACID IN GAS. *H. A. J. Pieters and K. Penners. Het. Gas, vol. 55, pp. 402–404 (1935).*

A study of different hydrocyanic methods. The polysulfide method determines the cyanide as well as hydrocyanic acid and likewise some of the carbon disulfide (by decomposition of the ammonium carbon polysulfide while boiling); hence high results are given; however, it is satisfactory for practical purposes. The complex ferric cyanide method (Feld) gives a possibility of thiocyanate formation if the gas contains oxygen and hydrogen sulfide. Only one-half of the cyanide is determined as hydrocyanic acid; the results are somewhat lower than those of the first method. The easiest method for all-around use is the method of absorption of hydrocyanic acid in strong sulfuric acid and titration of it as ammonia. It is sufficiently accurate.—*Chem. Abstr.*

MICROCOLORIMETRIC DETERMINATION OF BENZENE IN BLOOD AND URINE. *S. J. Pearce, H. H. Schrenk, W. P. Yant. U. S. Bur. Mines Rept. Invest. no. 3302 (1936).*

A microcolorimetric method for the determination of small amounts of benzene in blood or urine is described. The benzene is aerated from blood or urine and swept into a small amount of nitration acid, after which the method described in Bureau of

once, 7% twice, and 1.4% several times. In other words, 5015 stone cutters had at least one examination, and of these, 686 (13.7%) showed some silicotic changes.

This study indicated that 8656 or 88.3% of the persons examined had no silicosis, 10.6% were found to have a slight silicosis, while the remaining 1% were classed as moderate or severe silicosis. It was found that it took about 20 or more working years to develop silicosis and that 50% of the workers show a slight silicosis in about 13 years. The beginning of silicosis was found to be independent of age. The number of cases with a change of occupation was found to be too small to draw any conclusions on this point.

It is shown that in approximately 500 cases where marked silicosis appeared, the trade life averaged 18.3 years. The length of life among those suffering from severe silicosis was found to be approximately 6–7 years. "In a mortality study of 493 severe silicosis cases, 37% were found to be due to uncomplicated silicosis following heart failure, while 63% of the deaths were due to silicosis accompanied by tuberculosis." It is unfortunate that no data are presented on the severity of exposure, or on the composition of the dust, so that more information would be available on the etiology of this disease.—*J. J. Bloomfield.*

PNEUMOCONIOSIS WITH SPECIAL REFERENCE TO THE SILICO-ANTHRACOSIS OF COAL MINERS. *S. L. Cummins. Brit. Med. J., pp. 287–290 (Aug. 17, 1935).*

The author believes that coal dust may exert a beneficial effect by adsorbing toxic substances and thus reduce the likelihood of tuberculosis in silico-anthracotic lungs. "The classical state of miners' dyspnea (in coal miners) is very marked and is expressed in the death returns as a high bronchitis mortality, the terminal effect of a high tuberculosis mortality is much diminished through the operation of coal dust as an adsorbent for the toxic products of tuberculous foci in the lungs." The importance of preventing the dissemination of dust, as from drilling, is emphasized. The work of W. R. Jones indicates that it might be well to study carefully the stone dusts used to prevent coal dust explosions.—*Philip Drinker.*

PREVENTION OF SILICOSIS IN THE PORCELAIN INDUSTRY. *Hartmann. Zentralbl. f. Gewerbehyg. u. Unfall., vol. 12, pp. 141–150 (Aug.–Sept., 1935).*

Hard porcelain is made from 40–60% clay, 25–40% quartz, and 20–30% feldspar. Soft porcelain is made from 25–40% clay with various proportions of quartz and feldspar.

The general processes of porcelain manufacture are described in considerable detail and illustrations are given. Having particular reference to silicosis prevention, there have been in force in Germany since April 1, 1934, in the ceramic trade, rules requiring (1) cleanliness and order, with removal of spilled material; (2) workplaces where silica dustiness cannot be properly controlled otherwise, should be enclosed or the dust be exhausted. When these methods fail, suitable respiratory protective equipment is required; (3) workplaces must be cleaned up not less than once a week, using wet sawdust or vacuum sweepers.

The author stresses the need of coöperation between physicians and engineers, and points out that in 1933 the ceramics association paid out 585,025 RM for accidents and 600,101 RM for silicosis. In that year there were 35,936 workers employed in this trade. —*Philip Drinker.*

ETIOLOGY OF SILICOSIS. *W. R. Jones. Zentralbl. f. Gewerbehyg. u. Unfall., vol. 12, pp. 151–155 (Aug.–Sept., 1935).*

This paper is a good summary of the author's well known sericite theory and particularly of his unrefuted proofs of the difference between the composition of fine air-floated dust and that of the parent rock. The eighteen photomicrographs and camera lucida drawings are essentially the same as those published previously.—*Philip Drinker.*

ASBESTOSIS. PART 2. THE NATURE AND AMOUNT OF DUST ENCOUNTERED IN ASBESTOS FABRICATING PLANTS. PART 3. THE EFFECTS OF EXPOSURE TO DUST ENCOUNTERED IN ASBESTOS FABRICATING PLANTS ON THE HEALTH OF A GROUP OF WORKERS. *W. B. Fulton et al. Penna. Dept. Labor and Industry. Spec. Bull. No. 42, Sept. 20, 1935.*

Employing the sampling methods outlined in Part I of this series (*Spec. Bull. No. 37, Oct. 1, 1934*), impinger samples col in alcohol, a study was made of the as fabricating industry in Pennsylvani Part 2 of the series, the results of this are given. It was found that the conc tion of dust in the plants decreased quality or length of the fibers increased the various milling operations, whic described in detail, dust concentratio creased in the following order: prep carding, weaving, spinning, twisting, ing, and warping. Due to the fi nature of the dust, the average partic is stated in two diameters, longitudin transverse. Asbestos dust in the wo breathing zone was shown by petrogr analysis to contain no free silica. The part of this series consists of an accou complete physical examinations of 64 sons. Fifty-six of the subjects had previous exposure to asbestos dust, had had very little exposure and were as controls, while the other subject found to have had previous exposu silica dust. Fourteen of the 56 worke ployed in the textile departments of fabricating plants showed both clinical roentgenological evidence of asbest The study was too limited to permit formulation of permissible standard dustiness, but a reduction in the asb dust concentration in the operations stu is shown to be necessary.

The bulletin closes with a bibliograph 125 references on asbestosis and gen dust determination.—*R. Page.*

HYGIENIC AND CLINICAL-ROENTGENOL CAL INVESTIGATION OF PULMONARY S COSIS IN A JAPANESE FOUNDRY. *S. I oda, Chief Doctor, Steel-mill Hospita Yawata, Japan, 1935.*

This is the report of an extensive inv gation of pneumoconiosis and atmosph pollution in Japanese foundries. A gr of 715 workers were examined clinically 314 of these were radiographed. Th workers included electric truck operat manufacturers of firebrick, cement operators, molders and general plant la ers as well as stamp mill operators and sa blasters. From the cases studied, 25 w diagnosed as silicosis, 81 as possible pn moconiosis, and 2 as tuberculosis. Dur the two years (1933 to 1935) covered by

57, *Oct. 1, 1934*), impinger samples collected in alcohol, a study was made of the asbestos fabricating industry in Pennsylvania. In Part 2 of the series, the results of this study are given. It was found that the concentration of dust in the plants decreased as the quality or length of the fibers increased. In the various milling operations, which are described in detail, dust concentrations decreased in the following order: preparing, carding, weaving, spinning, twisting, winding, and warping. Due to the fibrous nature of the dust, the average particle size is stated in two diameters, longitudinal and transverse. Asbestos dust in the workers' breathing zone was shown by petrographic analysis to contain no free silica. The third part of this series consists of an account of complete physical examinations of 64 persons. Fifty-six of the subjects had had previous exposure to asbestos dust, seven had had very little exposure and were used as controls, while the other subject was found to have had previous exposure to silica dust. Fourteen of the 56 workers employed in the textile departments of the fabricating plants showed both clinical and roentgenological evidence of asbestosis. The study was too limited to permit the formulation of permissible standards of dustiness, but a reduction in the asbestos dust concentration in the operations studied is shown to be necessary.

The bulletin closes with a bibliography of 125 references on asbestosis and general dust determination.—*R. Page.*

HYGIENIC AND CLINICAL-ROENTGENOLOGICAL INVESTIGATION OF PULMONARY SILICOSIS IN A JAPANESE FOUNDRY. *S. Kuroda, Chief Doctor, Steel-mill Hospital of Yawata, Japan, 1935.*

This is the report of an extensive investigation of pneumoconiosis and atmospheric pollution in Japanese foundries. A group of 715 workers were examined clinically and 314 of these were radiographed. These workers included electric truck operators, manufacturers of firebrick, cement kiln operators, molders and general plant laborers as well as stamp mill operators and sandblasters. From the cases studied, 25 were diagnosed as silicosis, 81 as possible pneumoconiosis, and 2 as tuberculosis. During the two years (1933 to 1935) covered by the study five cases became totally incapacitated and six more died (4 from silico-tuberculosis). It was estimated that among those working with quartz, 8.65% had silicosis, 16.21% possible pneumoconiosis, and 1.08% pulmonary tuberculosis, while among the workers on steel castings, 6.61% had silicosis and 12.2% possible pneumoconiosis. Workers on magnesite and chromite ores, slag-stamping operations, cement kilns, limestone, dolomite and iron ore plants showed few symptoms of lung fibrosis. Detailed accounts of the examinations, classification of findings, and results of autopsies are given.

The author estimates the average age and average exposure as follows: quartz workers —42.9 and exposed 17.6 years, chamotte workers—42.6 and exposed 17.1 years, other pulverizing workers—40.1 and exposed 17.11 years. The average incubation period for incapacitating pneumoconiosis is estimated at 18.1 years while the duration of the sickness (which is usually complicated with pulmonary tuberculosis) averages 2.2 years making the total course 21 years.

The morbidity of the workers exposed to dust was as follows: (a) with quartz—80.9% among edge-mill workers, 45% among kiln operators, 33.6% among electric truck operators, 31.4% among molders; (b) with chamotte—30.5% among molders, 27.3% among electric truck operators, 22.7% among edge-mill workers, 22.2% by workers on raw materials, 12.3% by oven operators; (c) with magnesite ore—37.3% among clinker workers, 12.9% by formers; (d) in cement work—22.7% by workers on raw material. During 1926–1931 the general annual accident and sickness rate of workers in industry was 1.98% of which 0.76% were from respiratory diseases, while the loss by fire brick workers amounted to 3.48% of which 1.88% were from respiratory diseases.

Gravimetric dust determinations by thimble and gasometer showed 920 mg. dust per cbm. of air in the quartz screening room, and 65.2 mg. per cbm. near the edge-mill, 85.3 mg. per cbm. near the chamotte edge-mill, 486.5 mg. per cbm. near the cement kiln, and 298.4 mg. in the cement bagging room. [Dust counts taken with Shimazu's apparatus appear too low to compare with gravimetric determinations.—*Abstractor*] Silica contents of the composite dusts are

50 mgs. per cu. meter and in the presence of approximately 1500 dust particles per cc. up to 10 to 15 microns in size. Those under 1 micron (43.4%) and 1 micron (30.2%) were the most common. The exposures lasted from 10 days to 8 months. Macro- and microscopic observation of the organs of the rabbits showed that äthrol dust produced about the same changes as the dust of other plastics (ebonite and bakelite), that is, inflammatory affections of the nasal and adjoining cavities. In the long exposures, dust thrombi and especially terminal alveolitis ensued. In the experiments lasting up to 2 months it was ascertained that there was an increase in blood neutrophiles and a decrease in lymphocytes, whereas in the longer experiments a reversal of the behavior of this elementary form of the blood was noticed. Thus äthrol, in its effect on the organism, seems to occupy a middle place between the organic and inorganic dusts.—*From the author's German summary.*

RESULTS OF X-RAY CHEST EXAMINATIONS AMONG 2500 WORKERS IN A "HEAVY INDUSTRY" PLANT. *L. M. Warfield. Indust. Med., June, 1935, vol. 4, pp. 302–306.*

A study of x-ray films (flat) of 2500 workers in a heavy industry plant is reported. Based on a classification of silicosis according to nodule size, of mild, moderately severe, and severe, 145 (5.8%) of those examined had evidence of silicosis. Of these, 90% were foundry workers. Of the remaining 10%, most gave histories of previous work in mines.

Among 692 foundry workers, 81.3% were negative, 1.3% doubtful, 8.8% mild, 8.2% moderately severe, and only 0.4% severe. Of these last cases, three in number, one had been a miner for 10 yrs.; the other two were 60 and 71 years of age. None of these men complained of symptoms nor were they disabled.

Silicosis among foundry workers appears to be milder and slower in its development than among granite cutters or sand blasters.

There were 11 cases of healed tuberculosis with silicosis and 13 cases in non-silicotic lungs. "In this particular foundry the menace of tuberculosis appears to be no greater than it is in the city at large."

The impression is gained from this study that foundries may be operated practically without undue hazard to the health of the workers or limitation of their working capacity.—*Theodore Hatch.*

SILICOSIS AND ASBESTOSIS: A MEMORANDUM. *Home Office, H. M. Stationery Office, London, 1935.*

The present position regarding compensation for pulmonary fibrosis is usefully summarized in this brochure. During the three years 1932–4 there were certified 435 deaths from silicosis and 1,444 instances of disablement, and 5 deaths with 60 cases of disablement due to asbestosis. A short description is given of the origin and development of silicosis; and then are stated the industries and processes in which silicosis occurs. Means of prevention are grouped under (i) suppression of dust, generally by the use of water, (ii) ventilation so applied at the point of origin of the dust as to prevent its access to the air of workplaces, (iii) other preventive measures such as furnishing each worker with a separate supply of dust-free air to breathe or wearing efficient respirators, and (iv) initial and periodic medical examinations, under which workers with defective respiratory physique are excluded from the industry and anyone found to have pulmonary tuberculosis is suspended from further employment. The medical arrangements for examinations and for certifying cases are described. All these medical matters are carried out by a Board of experts from whom there is no appeal. The compensation schemes differ in minor details for various industries; they cover the getting of ganister and other highly siliceous materials, the getting and manipulation of sandstone, the grinding of metals on sandstone wheels, a long list of processes entailing risk of inhaling silica dust in such industries as the manufacture of pottery, fettling of steel castings and sand-blasting, and the asbestos industry.—*E. L. Collis.*

SILICOSIS—AN INTERPRETIVE REVIEW OF ACCUMULATED EXPERIENCE. *C. O. Sappington. Indust. Med., Apr., 1935, vol. 6, pp. 173–177.*

A brief discussion has been given covering