INDUSTRIAL DUST

Hygienic Significance, Measurement, and Control

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Silicosis is diagnosable by x-ray only if a good history, with proof of adequate dust exposure, is available. Anyone with normal vision can follow the changes that appear in a series of x-ray plates of the chest taken of a man who has had a severe dust exposure over a number of years and ultimately died of silicosis. Such a series is, today, something of a curiosity. The modern medical student is about as apt to see a case of typhoid fever as he is one of silicosis; so familiarity with the disease is not to be expected.

Silicosis and Duration of Exposure. Silicosis may not become disabling until some years after dust exposure has ceased. Watkins-Pitchford (437) gave examples of Welsh miners who passed the physical examination for enlistment in the British army, fought in the trenches through World War I, then came back to England and died of silicosis. Britton and Head (54) gave more detailed examples of similar latent effects in the United States.[1]

This problem of latency may be embarrassing—it is not reassuring to a client to state that some dust-control measure you recommend cannot really be appraised until the men exposed have worked through this vague latent period.

Harrington (207) of the U.S. Bureau of Mines assembled data on the length of exposure required to give definite silicosis; these showed that first-stage silicosis could develop in as short a time as 8 months. Among foundry workers where the risk is low, McConnell and Fehnel (305) in 1934 and Pope and Zacks (338) in 1935 found first-stage silicosis only after long employment. But in severe quartz-dust exposure the condition is very likely to progress and to become complicated by tuberculosis whether the man leaves his dusty occupation or not. Thus the length of exposure that will produce the disease varies with the working conditions and individual susceptibility.

In Great Britain the reports of the Chief Inspector of Factories show the number of deaths from silicosis as well as the ages of the victims. We show Tables 6 and 7 taken from the reports of 1934 and 1947. One could compile the yearly figures and the rates for silicosis, but we believe that the progressive increase in age at death is the important item. We doubt the value of yearly figures or of rates because of this vague latent period of the

[1] We emphasize it further in Fig. 34, p. 107, from South African data.

disease which makes it impossible to fix causative exposures with any semblance of accuracy.

From all parts of the industrial world the indications clearly point to a lengthening of the time required to produce silicosis.

TABLE 6. FATAL CASES OF SILICOSIS AND ASBESTOSIS INVESTIGATED UP TO END OF 1934 (GREAT BRITAIN)

	Number of deaths	Average age at death	Duration of employment, years		
			Maximum	Minimum	Average
Silicosis	261	55.4	60	2.3	34.8
Silicosis with tuberculosis	315	52.5	67.0	2.0	32.0
Asbestosis	41	41.0	27.0	1.5	12.9
Asbestosis with tuberculosis	26	38.0	29.0	0.8	9.9

Source: After Bridge (52).

TABLE 7. FATAL CASES OF SILICOSIS AND ASBESTOSIS INVESTIGATED UP TO END OF 1947 (GREAT BRITAIN)

	Number of deaths	Average age at death	Duration of employment, years		
			Maximum	Minimum	Average
Silicosis	1037	58.2	62.0	1.5	34.3
Silicosis with tuberculosis	1046	53.6	67.0	0.7	31.3
Asbestosis	160	47.5	48.0	0.5	14.9
Asbestosis with tuberculosis	72	39.0	29.0	0.8	10.4

Source: After Barnett (25).

The improvement is partly due to the decreased incidence of tuberculosis and not solely to better working conditions. In 1913 the Metropolitan Life Insurance Company established a tuberculosis sanitarium for its own employees, and in 1945 they celebrated its abandonment because they no longer had enough patients to justify maintaining it. This event comes close to being a milestone in public health and shows how spectacularly the tuberculosis rate is being reduced.

Joseph (249), summarizing 25 years' experience (1913 to 1938) in the Rand mines, shows (Fig. 17) the remarkable lengthening in the exposure time needed for development of silicosis in that famous mining community.

We have no data to prove it, but our opinion is strongly that this graph indicates the epidemiological trend of silicosis in the

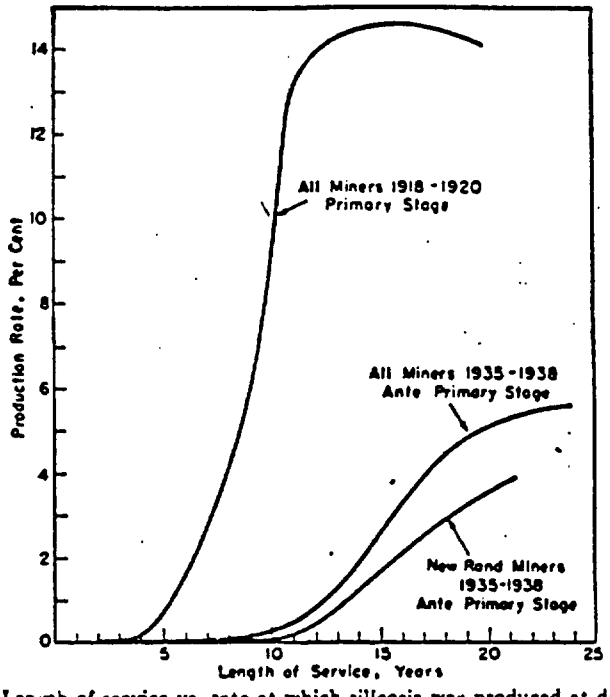

FIG. 17. Length of service vs. rate at which silicosis was produced at dates indicated; Witwatersrand mines. (*After Joseph, courtesy J. Chem., Met. Mining Soc. S. Africa.*)

modern industrial world—a steady and consistent lengthening of the time required to develop the disease. This is tantamount to saying that both the frequency and the severity of silicosis are steadily decreasing.

We are all familiar with Agricola's epidemiological observation (2) that many women in the Carpathian mining district married seven husbands who were carried off to an early death—presumably by silicosis. We cannot question his mortality figures, but we do question his etiology. This remarkable book

on metallurgy was published in 1556. Blasting and pneumatic rock drilling, the main dust producers in mining, are a product of the end of the last century. Dust exposures, as we know them, did not exist in Agricola's time. If tuberculosis had been controlled among the Carpathian miners, we doubt that their mortality would have been unusual.

A significant part of the increase in pneumoconiosis in Britain occurs in their coal miners. It is not a new disease in coal mining, it is found only after years of work in coal dust, it is responsible for peculiar and distinctive chest x-ray markings, it is often disabling and it is compensable.

There is no thought among the British authorities that coal miners' pneumoconiosis is a new disease. Fletcher (159, 238) sums up the present British situation by stating that from 1931 to 1948 there were 22,000 men (90 per cent from South Wales) certified as disabled from coal dust. These men came from about 100,000 underground workers, while only 600 were certified as disabled from 600,000 miners exposed elsewhere in Great Britain.

The importance of the dust hazard, in its effect upon both the death rate for tuberculosis and the death rate for all causes, is shown in Table 8, giving the number of actual and of expected cases (based upon general experience) among workers in the chief dusty trades. These data, which represent the combined experience of 12 life-insurance companies from 1915 to 1926, show excess mortality varying from 114 to 450 per cent of the expected rate for all causes and from 103 to 1833 per cent for tuberculosis. In general, the mining, quarrying, and stone-dressing operators show a higher hazard than the general manufacturing workers.

The Employability of Workmen with Silicosis. We like what Cummins (89) wrote about the disability of the person with silicosis: "It may be said of pneumoconiotic cases in general that what most interests the clinician is the mottling seen in x-ray films; what chiefly attracts the pathologist is the mystery of the silicotic nodule; and what distresses the patient is shortness of breath."

There has been much work directed toward appraising this disability, for there is nothing more distressing than difficulty in breathing. The subject is complicated, and it is overoptimistic, perhaps, to expect a formula whereby percentage disability can be fixed by physiologic tests, with or without x-rays. No one

TABLE 8. NUMBER OF DEATHS EXPECTED FROM SPECIFIED CAUSES AND NUMBER WHICH ACTUALLY OCCURRED AMONG PERSONS ENGAGED IN CERTAIN OCCUPATIONS EXPOSED TO SILICA DUST (UNITED STATES)

Occupation	Deaths from all causes			Deaths from respiratory tuberculosis		
	Actual	Expected	Percentage actual of expected	Actual	Expected	Percentage actual of expected
Metal-mine operatives (underground):						
Gold and silver mines	44	9.77	450	9	1.12	804
Lead and zinc mines	22	5.03	437	11	0.60	1833
Copper mines	83	22.63	367	24	2.63	913
Iron mines	33	12.12	272	4	1.54	260
Quarry operatives	37	22.45	165	7	2.66	263
Stone cutters:						
Granite and sandstone	29	17.47	166	16	1.64	976
Marble and limestone	12	10.55	114	3	1.02	294
Metal industries:						
Grinders	46	28.65	161	7	3.33	210
Buffers and polishers	89	64.10	139	10	6.80	147
Chippers (not ship)	38	14.14	269	8	1.30	615
Core makers and sand-molders	32	22.65	141	3	2.92	103
Iron and steel foundries:						
Molders, founders, and casters	202	130.14	155	24	13.38	179
Drillers, flask makers, machine hands, mixers, etc.	45	31.03	145	7	3.03	231

Source: Ordinary department mortality experience of 12 life insurance companies, 1915–1926 (274).

appreciated these difficulties better than the late L. U. Gardner, whose wealth of experience gives his opinions unusual value. At the request of the Department of Labor and Industry of Minnesota he discussed the subject in their Thirtieth Biennial Report (1945–1946) with special reference to the silicosis problem in their iron mines. What he says applies so generally that we quote parts of his report (171), one of the finest pieces of work in this field:

In the absence of associated tuberculosis of the lungs, silicosis does not ordinarily cause symptoms or disability. Most of the disability in silicotics is a result of associated tuberculosis.

Silicosis is a menace largely because its presence predisposes to fresh tuberculous infection from without, and because, in combination with the tubercle bacillus, inhaled quartz produces massive fibrosis of the lungs.

Contraction of this fibrous scar causes the air spaces in the rest of the lung to over-distend in order to fill the chest cavity. This process of distention is known as compensatory emphysema, and it is emphysema, rather than fibrosis, that gives rise to shortness of breath. Since so many of the older silicotics had associated chronic tuberculosis, shortness of breath came to be recognized as the cardinal symptom of the disease. However, it should be borne in mind that, without tuberculosis, silicosis alone causes little emphysema. Some uncomplicated silicotics may also have emphysema because it is prone to develop in older men, but in them it is not caused by the dust disease.

The problem that faces employers, industrial physicians and compensation officials today is the disposition of cases of silicosis, largely produced by exposure to heavy dust concentrations fifteen or more years ago. At that time there was no general appreciation of a hazard. Today's adjustments to this situation must involve compromises, which it is hoped will become less frequent after the present generation is gone. New employees who have worked only under controlled atmospheric environments should create few problems of this nature. To discharge all men now discovered to have silicosis will not correct the damage that has already been done. Change to surface employment may be temporarily acceptable to a miner but, being a specialist, he is rarely able to command as high wages in another job. Compensation may ameliorate the economic strain, but such payments can only be temporary. The mere knowledge that he has a pulmonary disease of such nature that it necessitates loss of earning capacity may transform a strong and healthy workman into a neurotic invalid. For these reasons, it would seem ill advised to recommend that every case of simple silicosis be discharged as soon as the diagnosis is made.

If the subject is comparatively young, in his twenties or early thirties, there is more reason to advise change of employment than in men over 45. If an iron miner, he may be kept out of development work where there is apt to be some exposure to quartz dust.

In men over 45 who develop silicosis not complicated by tuberculosis, there is even less reason for change of employment. In all probability the disease has taken a lifetime to develop under the high dust concentrations which prevailed before the hazard was locally recognized. Transfer to positions where there is less free silica and much less dust because of modern methods of control may ultimately do less harm than discharge. As long as such men remain with their original employers, they are subject to periodic examination, which will detect superimposed tuberculosis in early stages when it is amenable to treatment. Discharged from their jobs, they lose the benefits of such control, for the silicotic nodulation in their chests is a cause for rejection in any plant using x-rays as part of its physical examination program. A few such rejections may turn an able workman into a discontented neurotic.

The young silicotic workman who develops tuberculosis should be sent to a sanatorium as promptly as possible. Only there has he any chance to cure his infection, and there he is not a menace to the public at large.

Old silicotics with massive conglomerate disease in which latent tuberculosis generally plays a part are apt to be partially disabled by shortness of breath. Sanatorium treatment does them no good; in fact, to put them to bed merely increases their dyspnea.

In this country, the most acceptable compromise has been continued employment at jobs compatible with the subject's condition. Many of them are able to do regular work as miners; some can be usefully employed as pump men or at other jobs that do not involve severe physical exertion. The major difficulty is to find enough jobs of this nature to keep such old employees on the payroll.

Experience has demonstrated that most of them do reasonably well for prolonged periods. Followed in annual examination films over periods as long as ten or twelve years, their disease gradually increases in extent and their shortness of breath becomes more marked. Sputum examinations should be made from time to time to make certain that their infection has not become active and that they consequently are not a public health menace. Negative results may be expected until such time as the x-ray reveals more rapid changes in their disease. When this finally happens, hospitalization for the protection of others is indicated.

This recommendation may seem heartless but, for the most of these cases, it is perhaps the kindest treatment that can be offered. Until medical science finds some means of combating the infection, the expect-

ant treatment is as good as anything that can be offered for these tragic results of past ignorance.

Asbestosis. The pathologic changes produced by asbestos are not like those of silicosis. The asbestos fibers group about the neck of an alveolus and stimulate the formation of a diffuse fibrosis. There is no definite migration or transportation of the dust particles to the lymph nodes and no formation of the fibrous nodules shown in Fig. 16*a*. As the fibrosis increases, the reduction in lung area causes serious dyspnea. Lanza (273) suggested that the enlarged hearts noted frequently in his cases of second-stage asbestosis may be the result of the increased work of the heart resulting from this condition; it takes more work to pump blood through the asbestotic than through the normal lung.

Gardner stated (175, 170): "On grinding these fibrous minerals to a very fine state of subdivision they do not become more irritating but practically lose all power to provoke tissue reaction . . . ." Vorwald *et al.* (433) continued the animal work initiated by Gardner and concluded: "The duration of exposure required to develop the pulmonary reaction to inhaled asbestos dust is inversely proportional to the concentration of long fibers in the atmosphere; as the concentration is increased, the reaction develops in shorter time."

In silicosis it seems to be a general rule that, after a certain point, the victim's condition grows worse even if his exposure to dust has ceased. But Wood and Gloyne (455) stated that they have seen patients with asbestosis "whose condition appears to have remained stationary since stopping work in the factory," but they advised definitely that the individual with asbestosis be removed from his dusty job. Merewether (311) and Lanza were less certain on this point.

Asbestosis Bodies. In the lungs of patients who have died after prolonged exposure to asbestos dust and in the sputum of men with considerable asbestos-dust exposure are found what first were called *curious bodies* and later *asbestosis bodies* (Fig. 18) (140). While somewhat similar bodies can occur in the lungs of coal workers and even of normal persons, it is admitted that asbestosis bodies in sputum are characteristic of asbestosis. Stewart (402) gives considerable diagnostic weight to their presence as do Sparks (397) and Gloyne and Merewether (184).

Asbestosis and Lung Cancer. The British require autopsies of persons who have allegedly died as a result of industrial exposures such as cause asbestosis or silicosis. The 1947 report of the Chief Inspector of Factories (25) states that, of 235 cases of asbestosis autopsied between 1924 to 1946, 31 or 13.2 per cent were complicated by carcinoma of the lungs or pleura. This figure should

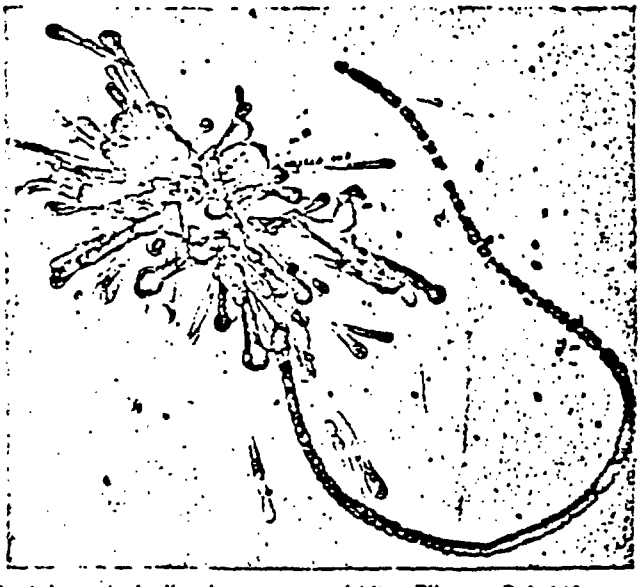

FIG. 18. Asbestosis bodies in sputum. (*After Ellman, Ref.* 140; *courtesy J. Indust. Hyg.*)

be compared with that of their cases of silicosis of whom 6884 were autopsied over the same period and in which 1.32 per cent showed cancer of the lungs. This latter is about the rate reported in the 1946 census in the United States (13 per cent of all deaths were from cancer and 1 per cent from cancer of the respiratory tract).

We do not imply that our American pathologists and our hospitals are less careful than the British in collecting data from autopsies. For example, Vorwald and Karr (434) at the Saranac Laboratories reviewed such data from their own experience and concluded that "inhaled dusts," except those containing recognized carcinogenic substances, "cannot in general be considered

as etiological factors in the development of pulmonary carcinoma." But we still are a bit envious of the tidy way in which the British assemble their industrial data on morbidity and mortality.

PREVENTION OF SILICOSIS

Suppose a manufacturer asks whether he can safely use a certain dust in his processes or a mine operator wants to know the dust risks in a new heading he is about to open up. How can the engineer best answer these questions; what will the investigation cost; and how soon can reasonable answers be given?

Chemical and petrographic analyses of the dusts enable us to reconstruct the approximate mineralogical composition (Chap. 11). Arguing that only the fine air-floated dust is breathed and causes silicosis, we can separate out the fines and determine the amount of free silica in the sizes below 5 μ. Admittedly this is a fussy job; it can be very time-consuming, but it may supply important information.

In the absence of epidemiological data on men, it may be wise to run tests on animals. We can cause small laboratory animals to breathe dust clouds in which concentration, particle size, and dispersion are controlled. This method has been used all over the industrial world, and very full descriptions of the technics have been published (172). It has worked well with quartz but has been unsatisfactory with less toxic dusts of low quartz content, which take years to produce ill effects in man. Gardner (272) pointed out that he had failed to produce silicosis in animals dusted with granite.[1]

Short cuts must be taken to compensate for the brief life span of laboratory animals. Pathological changes similar to those found in the lungs of persons with silicosis can be produced in various parts of the body. Dusts suspended in physiological saline solution can be injected intravenously and produce fibrosis in the liver (see Fig. 19, page 48), a method widely used. King (37) injected suspensions directly into the trachea of rats, Policard (251) used the cornea of rabbits, while Kettle (254) and others used subcutaneous inoculations.

[1] In retrospect we suggest that Gardner's granite dust clouds were not well dispersed and that in his animal experiments he underestimated the injurious effects he so perfectly described in man.