CHAPTER II

EFFECTS OF DUSTS AND FUMES UPON MAN

Exposure to dusts can produce four distinct types of disability: (1) the pneumoconioses, such as silicosis and asbestosis, are caused only by dust inhalation; (2) toxic dusts like lead, manganese, and cadmium can produce their effect as the result of either breathing or swallowing dust; (3) metal-fume fever is a temporary malady resulting from inhaling certain metallic oxide fumes; and (4) the allergic reaction or protein poisoning, as typified by hay fever, is the direct result of breathing pollen and other organic substances. In all four cases dust inhalation may be the sole cause of disability; only in the case of toxic dusts is there another mode of entrance.

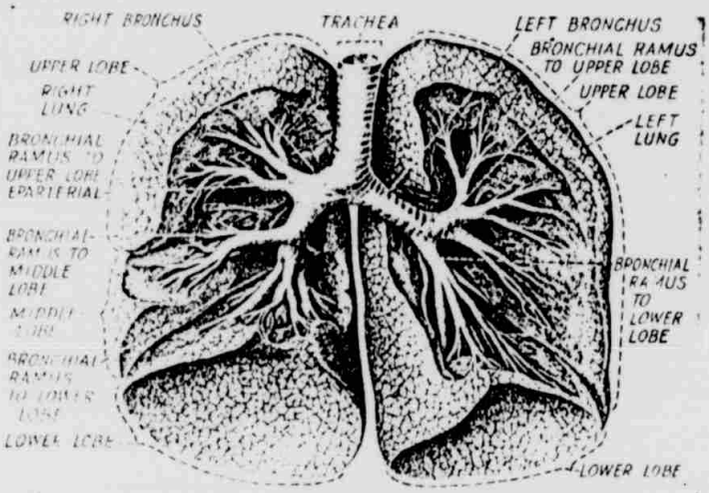


FIG. 10.—Lungs, bronchi, and trachea. (*After Sobotta and McMurrich.*)

Respiration and Dust Inhalation. The lungs are nonsymmetrical bilateral structures encased in a rather elastic cavity,

Effects of Dusts and Fumes upon Man (1954)
ACL Doc 25355 · Page 1 of 13
Published by AsbestosClaims.Law
asbestosclaims.law/library/25355/1

the chest, and they communicate with the nose and mouth through the trachea or windpipe. The left lung has two divisions or lobes and the right lung has three; the right lung is about 12 per cent larger than the left.

In the normal adult, the trachea is a tube some 1 to 2 cm. in diameter and 10 cm. long (Fig. 10); it is fortified with ringlike cartilage which gives it a strong and inelastic character. At the approximate level of the fourth rib, the trachea branches into the bronchi and these in turn subdivide into bronchioles which lead to the terminal air sacs or alveoli. It is in the alveoli that, as result of inspiration and expiration, gas exchange between blood and air takes place. Oxygen is taken up by the red blood cells and carbon dioxide is given off.

The amount of air breathed and the oxygen consumed vary with the task performed and with the individual's size. The rates at which an athletic man of 150 lb. breathes are shown in Table 1.

TABLE 1. OXYGEN CONSUMPTION AND VOLUME OF BREATHING BY MAN[1]

	Oxygen consumed at 0°C. and 760 mm. pressure, liters per minute	Air breathed at 20°C., liters per minute
Resting in bed	0.24	6
Sitting	0.30	7
Standing	0.36	8
Walking 2 m.p.h.	0.65	14
Walking 4 m.p.h.	1.20	26
Slow run	2.00	43
Maximum exertion	3.00–4.00	65–100

[1] After Henderson and Haggard (125).

The ordinary individual seldom breathes more than 50 liters per minute, the high rates being limited to trained athletes, such as long-distance runners.

The inspiratory and expiratory phases are not exactly alike, but practically we may consider each as taking half the time required for a complete respiratory cycle and we can ignore the pauses that occur at the beginning and end of each inspiration. Thus with minute volume of 50 liters the actual inspiration is at the rate of 100 liters per minute and momentarily rather high

velocities occur in the trachea. The velocity in the alveoli proper, however, is practically zero at all times because each alveolus is a dead end. The slight surge back and forth with respiration must be very close to still-air conditions. Thus there cannot be any driving or high-speed impingement of dust particles into the lung tissue. Furthermore, there is no evidence of such impingement in the upper passages where velocities are highest.

The respiratory mechanism is not strong enough to withstand for long any appreciable impediment or resistance to respiration. The muscles are easily fatigued, with resulting embarrassment and distress. Practice or training teaches the individual to minimize the effects of the resistance, but no one, strong or weak, will perform even the mildest exercise for long if the respiratory impediment exceeds a few inches, water gage. This fact is of the utmost importance in the design of respiratory protective equipment, as will be shown in Chap. XIV.

The Fate of Inhaled Dusts. A dust particle the size of a common pollen grain (15 to 25 μ) is likely to be caught in the nasal passages or at the back of the throat. If it should enter the trachea near the center line, there is no reason why it should not pass on down to the bronchi, but it is not likely to reach the alveoli. Collection of such a particle is the result solely of chance impact against the moist walls of the respiratory tubes. Obviously such impact takes place most effectively with particles large enough to have appreciable momentum and rapidly ceases to be effective as the particles approach sizes at which they move as an integral part of the transporting gas. Lining the trachea and extending down to the lower ends of the bronchioles are myriads of cells with whiplike appendages, cilia, which carry upward any foreign bodies that chance to touch the wet mucus-bathed linings of the respiratory passages. The nasal passages likewise are bathed in mucus and lined with cilia. All the mucus is moving toward the exits of the nose and mouth and is never stagnant.

Within the alveoli are other cells, phagocytes, which are brought out in vast hordes by the stimulus of foreign bodies such as dust particles, which they engulf (Fig. 11). The dust-laden cells, which have the power of independent motion, may wander off through the walls of the lung tissue into the blood capillaries

surrounding the lungs, or they may pass to the finer bronchioles, from which they are removed by ciliary action; thus they eventually reach the mouth and are spit out or swallowed. Within the alveoli there are neither cilia nor mucus.

Most of the dust-laden cells, however, migrate into the lymphatics, a system of closed vessels running through the tissue that supports the blood vessels of the lungs, the bronchioles, and the bronchi (Fig. 12). The lymph, a watery fluid which flows through the lymphatics, has its own circulation. It is derived from the blood, into which it discharges near the heart. At the various bifurcations of the trachea and the bronchi, the lymph passes through glands or lymph nodes, one of whose functions is the filtration of foreign bodies. It is at these tracheo-bronchial lymph nodes that a great deal of dust is deposited by the phagocytic cells and it is here that fibrosis of healthy lung tissue starts, following quartz-dust inhalation.



Fig. 11.—Phagocytic cells containing dust. (*Courtesy J. Ind. Hyg.*)

Dust particles can pass from the blood vessels into the lymph circulation without having been phagocytosed (60). Such particles then can be picked up by phagocytes at any point in the lymph stream or in a lymph node, the latter being particularly suited to such phagocytosis.

Fenn (78) found that all dusts are not phagocytosed with the same readiness; he gave cells an equal chance to ingest various dusts and found that quartz was among those least preferred.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25355/3

Gardner and Cummings (85) have pointed out that the motility or rapidity of migration of cells differs with different dusts.

PNEUMOCONIOSIS

It has been recognized for centuries that excessive dust inhalation generally produces a serious pulmonary disease which has been known in the various dusty industries as miners' asthma, miners' phthisis, grinders' rot, etc. Zenker (260) proposed the

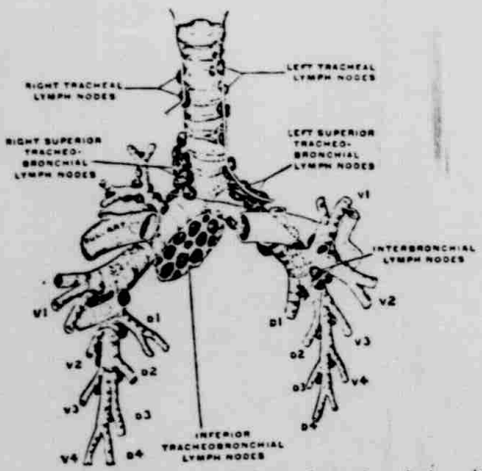


FIG. 12.—Position of lymph nodes in relation to the trachea, the bronchi, and the pulmonary artery. (*After Gray's Anatomy.*)

general name *pneumonokoniosis* (a *lung* containing *dust*) in place of the special names applied to certain industries. Recently this has been shortened to *pneumoconiosis* (134). The word originally implied that the lung had been seriously damaged by dust—enough to cause disability—but the meaning has been broadened in recent years to include all pulmonary manifestation of dust inhalation, whether the dust is injurious or harmless.

Silicosis and asbestosis are today the most important forms of pneumoconiosis. Other terms such as *silicatosis*, *anthracosis*, *siderosis*, etc., have been given to milder forms of the disease and

doubtless others will be added as studies of the dust problem progress.

Silicosis. For reasons still unexplained silicon dioxide as quartz (free silica) produces the worst form of lung fibrosis. The American definition of silicosis (3) states that it is a "disease due to breathing air containing silica (SiO_2) characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis. . . ." The International Silicosis Conference at Johannesburg in 1930 defined silicosis (133) as a "pathological condition of the lungs due to the inhalation of free silica dust. It can be produced experimentally in animals. It can be detected by clinical and radiological means, which can be confirmed with the above pathological condition with sufficient accuracy to separate it from other pneumonoconioses. It also affords a fair basis for legislative measures."

A recent note in the *Journal of the American Medical Association* (140) states that:

> Only silica (SiO_2) is capable of inducing silicosis but any other mineral dust under conditions of prolonged exposure and gross exposure may cause some increase in pulmonary fibrosis. If the relative potential harm of silica is rated as 100, these other nontoxic mineral dusts may be rated only on the order of 5 or 10. Moreover, the fibrosis is in itself not pathognomonic, since many other dusts, alkalies, acids, or vapors may induce somewhat similar if not identical X-ray markings.

In his summary of the situation in South Africa, Watkins-Pitchford (247) wrote in 1927 that dusts other than silica could "give rise to such nonpermanent and relatively harmless conditions—one can hardly call them diseases—as anthracosis, aluminosis, siderosis, etc."

In general, the damage done in silicosis (and asbestosis) is permanent; an unalterable tissue change takes place in the lungs. Of prime importance is the fact that prolonged exposure to these dusts results in a greatly increased susceptibility to tuberculosis, more so from quartz than from asbestos. Gardner has shown in the laboratory the great importance of this fact which had long been recognized in industry. Deaths from uncomplicated lung fibrosis caused by dust are infrequent.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25355/4

Free quartz possesses a unique cytocidal power; it kills phagocytic cells, the physiological scavengers, which then deposit their dust load *in situ*. The dust thus dropped may or may not be picked up again and the killed cell is said to mummify. The surrounding tissue then begins to fibrose and to appear more or

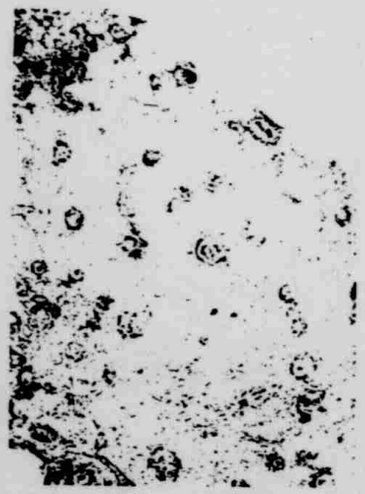


FIG. 13.—Silicosis. Isolated silicotic nodules above; fine nodules below. (*After Gardner; courtesy U.S. Public Health Service.*)

less stringy, as is shown in Fig. 13; such a fibrotic nodule contains dust particles identifiable chemically and petrographically.

Characteristics of Silicosis. For the convenient diagnosis of silicosis or of silicosis complicated by tuberculosis, the pulmonary condition is usually placed in one of three stages. In the first stage, the disease does no measurable harm (and produces no disability). The victim can work just as well as ever. But as his condition progresses to the second stage, his respiration is affected; he is bothered by dyspnea or labored breathing. If his dust exposure continues (and often even if he is kept out of dusty air), he is likely to reach a third stage in which dyspnea becomes severe and he is likely to contract pulmonary tuberculosis, generally with fatal results. Whether he contracts tuberculosis or not,

the advanced silicotic is far below normal and is inordinately susceptible to all respiratory diseases (44).

Each of these three stages of silicosis is diagnosable by X-ray, provided a reliable case history, including dust exposure, is available. A comparison of the X-ray plates of a silicotic with those of a normal person of like age and physique shows, in the first stage, distinctive shadows, evenly distributed in both lungs. These may be missed or misinterpreted by any but an expert; but even to the layman the changes in the second and third stages are obvious.

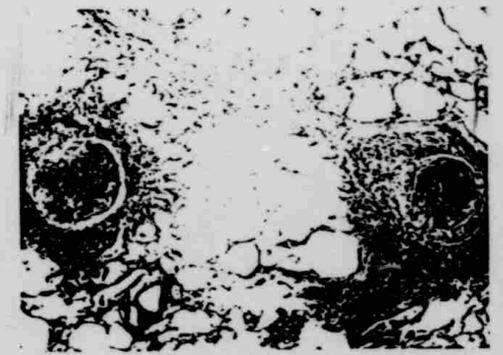


FIG. 13*a*.—Barre granite cutter. Two isolated silicotic nodules. Cellular connective-tissue borders and hyaline fibrous centers. Thickened interlobular septum extending upward to the left of the right nodule. Note dilated lymph vessels in septum. (*After Gardner; courtesy U.S. Public Health Service.*)

When silicosis first came into prominence in England, then in South Africa, and later in the United States, the cases described and the X-rays published featured advanced cases, often complicated with tuberculosis. A comparison of the X-ray pictures with those of normal chests showed striking differences that could not be missed by anyone. Granted that the layman might misinterpret what he saw in the X-ray, he could not miss the differences between the dusted and the normal chests. Today those cases would probably be called "third stage" in the American nomenclature.

The advanced third-stage silicotics described by Lanza (127), by Pancoast and Pendergrass (196), and by earlier writers in South Africa and in England seem to be disappearing in the

United States, and in their stead are being featured in the literature the cases more difficult to diagnose, which so confuse the layman. There is no doubt at all that the rapidly fatal third-stage silicosis has virtually been eliminated in the South African gold-mining district (135).

Silicosis and Duration of Exposure. Silicosis may not become disabling until some years after dust exposure has ceased. Watkins-Pitchford (247) gives examples of Welsh miners who passed the physical examination for enlistment in the British army, fought through the World War, then came back to England, and died of silicosis. Britton and Head (29) give more detailed examples of similar latent effects in the United States.

The length of exposure necessary to produce silicosis has been the subject of a good deal of controversy in the past. In Great Britain, Bridge (28) gives the following table:

TABLE 2.—SILICOSIS AND ASBESTOSIS IN GREAT BRITAIN, THROUGH 1934[1]

	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

[1] After Bridge.

Harrington (106) of the U.S. Bureau of Mines has assembled the data on length of exposure required to give definite silicosis; these show very clearly that first-stage silicosis may develop in as short a time as eight months. Among foundry workers where the silicosis risk is low, McConnell and Fehnel (178) and Pope (200) found first-stage silicosis only after long employment. But in severe quartz-dust exposure, where cases may occur in a matter of a few months, 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.

Silicosis and Tuberculosis. It is well-known that the tuberculosis mortality in the dusty trades is high. Collis and Yule (44)

TABLE 3.—RESPIRATORY DISEASES IN PERSONS EXPOSED TO SILICA DUST

Age	Ratios of mortality to that of standard population as 1000	
	Respiratory diseases	All other diseases
20	1202	704
25	1556	1055
35	2720	1160
45	3628	1474
55	4264	1550
20–65	3067	1396

showed that the mortality from causes other than respiratory diseases is also significantly higher for persons exposed to silica dust than for those without such exposure. But their figures for mortality from respiratory diseases (Table 3) are the most striking.

The experience of the Metropolitan Life Insurance Company (159) supports Collis' and Yule's data (Fig. 14). Starting at about the same level, the statistics for industrial males show a rapid increase with age, reaching a maximum at 45 to 54 years. At this point the death rate is more than three times higher than among the ordinary policy holders with the company and one and one-half times the rate for males in the United States. It is true that dust is not the only industrial factor that contributes to tuberculosis; there are other environ-

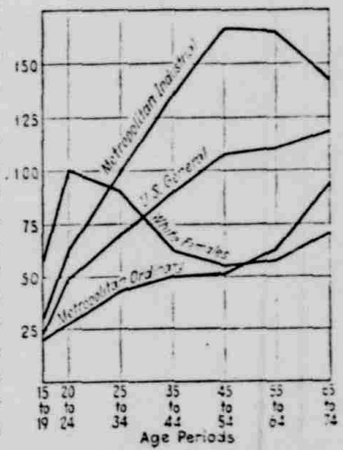


FIG. 14.—Tuberculosis—all forms—1931. Death rates per 100,000 white males by age periods. (*After Lanza and Vane; courtesy Am. Rev. Tuberculosis.*)

mental conditions of great importance which act to raise the death rate for industrial workers. Compared with female industrial workers, however, the males, who are more generally employed in the dusty trades, show a much higher tuberculosis death rate in the later years of life.

The importance of the dust hazard, in its effect upon both the tuberculosis death rate and the death rate for all causes, is shown in Table 4, in which are compared the number of actual cases and the number of expected cases (based upon general experience) among workers in the chief dusty trades. These data, which represent the combined experience of twelve life insurance companies, 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.

Disability from Dust Inhalation. There is no medical or social reason for branding a man with first-stage simple silicosis as disabled. Both industry and the man are done serious harm; the man is prevented from seeking gainful occupation, for no employer wants to hire a man whom the courts or the compensation boards have labeled as unfit or partially unfit. The employer, by hiring such a man, opens the door at once to suits involving the slightest mishap to the disabled.

In the British Empire, where silicosis legislation has been in force for some years, only the man with advanced silicosis, simple or infective, is disabled. Men with early, simple silicosis are not disabled, nor are they a menace to their fellow employees. The mere fact that a barely perceptible X-ray change is visible to the expert roentgenologist is not ground for claiming disability since most people over forty show some chest changes.

In the opinion of the special Industrial Disease Commission of Massachusetts (173), it is only when tuberculosis has become superimposed on silicosis that the victim becomes disabled or a menace to others. Thus a man with early, simple silicosis is not disabled and should not be prevented from working, an opinion seconded vigorously in the Saranac Silicosis Symposiums (211). In Canada the worker with simple silicosis is not refused further employment. The industry is enabled, by sensible legislation, to continue employing the man, and it is recognized

TABLE 4.—NUMBER OF DEATHS EXPECTED FROM SPECIFIED CAUSES AND NUMBER WHICH ACTUALLY OCCURRED AMONG PERSONS ENGAGED IN CERTAIN OCCUPATIONS EXPOSED TO SILICA DUST[1]

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	22.68	367	21	2.05	1024
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	10	1.54	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*)	58	14.14	260	4	1.30	615
Core makers and sand-molders	32	22.65	141	8	2.92	103
Iron and steel foundries:						
Molders, founders, and casters	202	130.14	155	24	13.38	179
Molders, *Bask makers, machine hands, mixers, etc*	45	31.03	145	7	3.03	231

[1] Ordinary department mortality experience of twelve life insurance companies, 1915–1926 (150).

Published by AsbestosClaims.Law
asbestosclaims.law/library/25355/7

fully that, if he has silicosis, he got it in Canada and Canada will take care of him.

In the case of a first-stage silicotic without tubercular infection, there is no method, as yet, for determining his lessened efficiency. The lung change, if any, caused by his dust inhalation is too small to measure.

McCann and Hurtado (177) have estimated disability from lung fibroses of various origins but have not yet succeeded in developing a method that is simple enough for routine use by compensation boards, insurance examiners, and the like. Their procedures involve various simple measurements of respiratory function but they have not established any methods of detecting fibrosis which are more sensitive than the present X-ray examination.

Of course, industry would welcome a diagnostic routine for detecting early harm from dust or, better, for forecasting harm. No such method is available and it seems fruitless to expect such help. The pneumoconioses are not diagnosed until harm is demonstrable by X-ray.

Collis (42) has stressed the difference between the age groupings in pulmonary tuberculosis and in silicosis, with or without tubercular infection superimposed. Tuberculosis is most prevalent between the ages sixteen to twenty-four, while silicosis rarely becomes disabling until later in life. Inasmuch as a good many years of dust exposure are required before the average case of silicosis (or asbestosis) becomes evident, it is obvious that it is not likely to be acquired early in life. However, there is an epidemiological side to the question which is of great importance and it is that which Collis especially emphasized. The average healthy individual has acquired a fairly effective immunity or resistance to tuberculosis. His chest X-ray is likely to show one or more healed scars or fibrosed areas from tuberculosis. Such an individual, Cummings emphasizes (211), is a better silicosis risk than the man who shows no evidence of past exposure to pulmonary tuberculosis. Consequently it is wise to select for dusty jobs men who are past the age of forty and not young men just taking up a trade.

Asbestosis. The X-ray picture of the typical asbestotic chest is confusing to the layman. The effect is described as a diffuse fibrosis. Characteristic silicotic nodules are absent and even

the expert roentgenologist withholds his diagnosis until the case history is complete.

The pathology produced by asbestos is not like that of silicosis. The asbestos fibers group about the neck of an alveolus and shut it off, causing what is known as *atelectasis*. There is no definite migration or transportation of the dust particles to the lymph nodes and no formation of the fibrous nodules as shown in Fig. 13. As the atelectatic areas increase, the reduction in lung

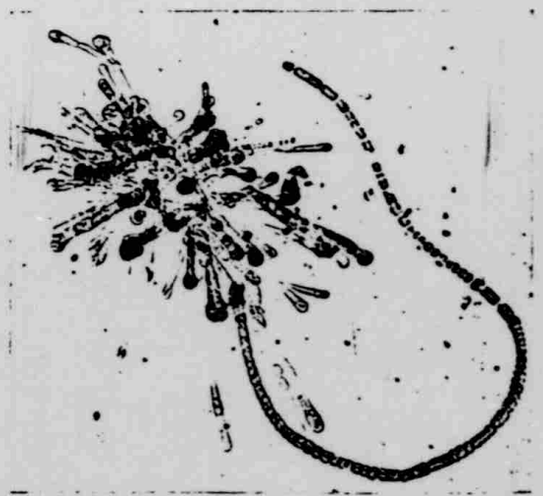


FIG. 15.—Asbestosis bodies in sputum. (*After Ellman* (75); *courtesy J. Ind. Hyg.*)

area causes serious dyspnea or labored breathing. Lanza (158) suggests 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 atelectatic than through the normal lung.

In silicosis it seems to be a general rule that, after a certain point, the victim's condition grows worse even if his dust exposure has ceased. But Wood and Gloyne (258) state that they have seen patients with asbestosis "whose condition appears to have

remained stationary since stopping work in the factory," but they advise definitely that the asbestotic individual be removed from his dusty job. Merewether (183) and Lanza are less certain on this point.

Asbestosis Bodies. In the lungs of patients who 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. 15) (75). While somewhat similar bodies 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 (223) gives considerable diagnostic weight to their presence.

Pneumoconiosis in Animals. Silicosis has been produced experimentally by quartz dusting guinea pigs, rabbits, mice, cats, and domestic fowl, thus emphasizing the specificity of quartz dust to biological tissue. In like manner asbestosis has been produced in experimental animals.

The Equidae, such as horses and mules, apparently are not susceptible to ordinary pulmonary tuberculosis but there is no reason to believe they have any special immunity or resistance to silicosis. The normal silica content of horses' or mules' lungs is not known but it would seem wholly reasonable to use the lungs of animals which have worked 5 to 15 years underground in mines as physiological dust samples. A brief report on this subject was published by Haynes (121) but data such as one needs are singularly lacking.

TOXIC DUSTS

Poisoning from inhaling toxic dusts is much more likely than poisoning from swallowing them. Dusts that reach the lungs may pass directly into the blood stream, thence to the heart, and immediately be pumped all over the body. Distribution to all the body tissue is thus brought about rapidly and effectively. But dust taken in with the food goes to the stomach and the major part passes out in the feces. Some is picked up by the portal blood circulation and moves on to the liver. That portion which causes poisoning must first pass through the liver, which is an effective filter and detoxifier; only then can it enter the general circulation.

The practical significance of this physiological distinction between the two ports of entry of dust is considerable. Hamilton (103) states,

> A great deal of money has been wasted by well-meaning employers who sought to protect lead furnacemen or oxide roasters or white lead grinders against poisoning, by providing baths and lunchrooms and clean overalls and mouth washes and such, instead of preventing the escape of lead into the air the men were obliged to breathe, and unfortunately this has sometimes been done under a physician's advice. It must never be forgotten that the great majority of industrial poisons enter the body with the inspired air and that while a workman eats only three times a day he breathes sixteen times a minute during the eight or ten hours of his working day.

Goadby (88) found that cats dusted with lead acquired lead poisoning more easily than did a control animal which was fed over ten times the total lead dosage for the dusted animals. Drinker and Shaw (61) showed how efficiently the liver removed foreign dust particles injected into the blood stream. Minot (188) emphasized the much greater danger of lead poisoning from inhaled than from swallowed dusts. Blumgart (24) showed that the absorption of lead directly in the nasal passages was rapid and might be "of a magnitude far in excess of the minimal dose by mouth."

All this experimental evidence confirms the view so often expressed by the late Sir Thomas Legge (162), but which is only now beginning to be accepted, namely, that there is far more danger of being poisoned by inhaling toxic dusts than by ingesting them in food or drink.

Metal-fume Fever. Of interest in connection with the breathing of dusts is metal-fume fever, a transient noncumulative malady that results from breathing rather heavy concentrations of metal fumes like zinc oxide, copper oxide, magnesium oxide, lead, probably lead oxide (227), and manganese dioxide.

About 2 to 8 hr. after a heavy exposure to the well-dispersed metallic or metallic-oxide fumes, the victim experiences chills followed by fever like that of malaria or the protein reaction following a typhoid inoculation. The patient's fever may reach uncomfortable heights (we have recorded 104°F.) and the next day he feels debilitated but generally can go to work. With

the fever goes an increased white-blood-cell count or leucocytosis like that experienced in any infection. By the next morning the fever has abated but the leucocytosis persists (Fig. 16). Then the victim is fairly immune; generally he can take another inhalation without experiencing a second attack (70). In industry it is a commonplace that attacks on successive days are unlikely.

The cause of this peculiar malady is probably the absorption of protein material which results from the action of the inhaled

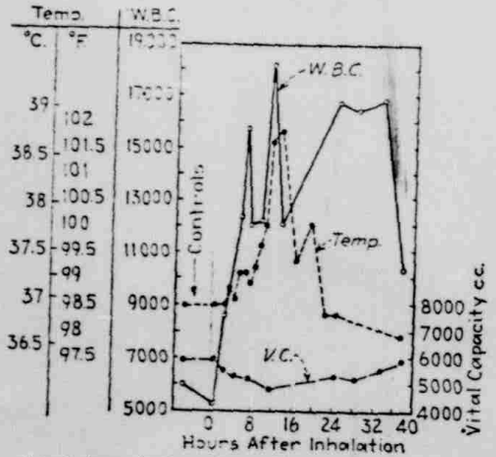


FIG. 16.—A typical attack of metal-fume fever, showing increase in leucocyte count, body temperature, and drop in vital capacity. Note that fever abates before the white count returns to normal. (*After Sturgis et al.; courtesy J. Ind. Hyg.*)

fume particles upon the tissue of the respiratory passages. In our own experiments we found that the chills and fever were acquired far more easily if one took a few deep breaths at a rate of say five breaths per minute than at the normal rate of twelve to fifteen breaths per minute. Slow, deep breathing insures penetration into the alveolar spaces.

Electric or acetylene welding in a confined space may generate metal-fume concentrations in excess of those used by Lehmann or Drinker in studying metal-fume fever. At present there are no data on the effects of breathing dense concentrations for

prolonged periods. There is reason to believe that the effects of heavy concentrations would be more severe than the ordinary metal-fume fever reaction (233).

ALLERGIC REACTIONS

An undetermined number of organic dusts produce allergic reactions like hay fever or specific kinds of skin reaction—dermatoses. Sensitivity varies greatly and unpredictably with the individual. The manifestation may take the form of a bothersome dermatitis or again may be a true poison in the commonly accepted sense. It is unnecessary for the offending dust to reach the depth of the lungs—giant-ragweed pollens, for example, which are unlikely to reach the alveoli, undoubtedly can produce their effect after being caught in the upper respiratory passages. Should the offending substance be finely ground (*e.g.*, broken-up pollens), it would reach the alveoli and as a result probably all physiologic reactions would be accelerated.

Landis (157), whose contributions to the field of pulmonary tuberculosis and pneumoconiosis are notable, stated of organic dust that it "never produces the condition known as pneumonokoniosis. That some organic dusts give rise to protein intoxication, such as asthma, threshers' fever, etc., is known."

THE DUSTY TRADES

Owing to the lack of occupational and mortality statistics on silicosis and other dust diseases in this country, it is impossible to state accurately the number of men exposed to injurious dusts. Hoffman (129) estimated in 1918 that there were approximately 4 million workers engaged in dusty occupations but of these only 1.7 million were exposed to metallic and mineral dusts. Recently Van Sicklen (244) prepared the following estimate of the number of men in various industries exposed to dust capable of producing silicosis (Table 5).

Lanza and Vane (159) carried the study further and have estimated the number within each industry actually exposed to silica-bearing dust, whereas Van Sicklen simply gave the total number employed in each industry. The data in Table 6 are compiled from the figures given by Lanza and Vane, who point out that in addition to these there are "thousands of workers

TABLE 5.—NUMBER OF MEN ENGAGED IN CERTAIN DUSTY TRADES INVOLVING (MORE OR LESS) EXPOSURE TO SILICA DUST[1]

Process	Total Exposed
Metal mining	62,268
Nonmetallic mining, quarrying, tunnel and foundation excavation	23,665
Bituminous coal mining, underground	350,513
Bituminous coal mining, open pit	8,219
Anthracite mining, underground	143,063
Anthracite mining, independent washeries	269
Smelting, nonferrous plants	13,166
Smelting, ferrous plants	24,960
Cement plants	33,368
Asbestos products	8,092
Abrasive industry	3,873
Clay products	93,336
Cutlery	14,991
Glass manufacture	67,527
Granite, slate, marble, and other stone products	28,715
Hones, whetstones, and similar products	174
Iron and steel	39,697
Mineral fertilizers	20,926
Minerals and earths, ground	1,679
Nonferrous metal alloys and products	79,183
Pottery, including porcelain ware	33,409
Sand-lime brick	566

[1] Adapted from Van Sulen.

TABLE 6.—ESTIMATED NUMBER OF MEN EXPOSED TO SILICA-BEARING DUST IN VARIOUS INDUSTRIES[1]

Process	Percentage of all employees	Number exposed
Metal mining	100	62,000
Anthracite coal mining	20	30,000
Quarrying of granite, ganister, sandstone, etc.	100	22,000
Smelting and refining	100	18,000
Foundry workers	100	200,000
Potteries, glassworks, stone products	50	70,000
Grinders, buffers, sand blasters, vitreous enamelers	?	62,000
Estimated total for chief mining, quarrying, and manufacturing industries		450,000

[1] Adapted from Lanza and Vane.

who have been exposed to silica dust in these various occupations and are now engaged in other forms of work."

Silicosis occurs in other industries as well as those listed above. Among these may be mentioned bituminous coal mining, rock excavation in connection with building and highway construction, and various public-utility jobs. The total number of men engaged in these miscellaneous industries is put by Lanza and Vane at 2,000,000, of which at least 5 per cent are said to be exposed to silica dust. Hence, one must increase the estimate in Table 6 by 100,000. Thus, the number of men exposed in United States industries to harmful amounts of silica dust is approximately 500,000.

We have no estimates of the number of workers exposed to asbestos dust in this country. In England, with an industrial population about one-third that of the United States, there are approximately 2200 asbestos workers (184).[1]

Dust Concentrations in the Dusty Trades. Clean country air contains as little as 0.1 to 0.2 mg. of dust per cubic meter, a large portion of which is organic matter. The concentration varies with meteorological conditions and with human activity. In manufacturing towns it may reach a level of 2.0 mg. or more. The air within a modern air-conditioned building should not exceed 0.2 to 0.3 mg. per cubic meter regardless of the concentration outside. In industrial establishments the dust concentration varies enormously; it will be less than 1 mg. per cubic meter in a well-controlled plant and over 500 mg. at the working face of a mine in which no dust-control measures are employed. An explosive concentration of a dust like wheat flour is about 10 to 15 grams per cubic meter. A dustiness of this concentration is very unstable because the suspension settles rapidly; an explosion usually follows a disturbance that jars dust off rafters and other dust-catching surfaces so that momentarily an explosive concentration exists.

Because of the great variation in dustiness in industry, it is impossible to summarize completely concentration values for

[1] Since writing the above we have seen a statement by Lanza from a paper presented on June 13th, 1935 at the Atlantic City Meeting of the American Medical Association to the effect that about 10,000 persons were exposed to asbestos dust. McPheeters (in *J. Ind. Hyg. and Tox.*, April, 1936) states that 9237 persons were exposed in 1929.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25355/11

the dusty trades. Figures are available from many countries but these are not directly comparable with one another because the dust-sampling instruments and methods of determination are not alike. In tabulating published data, therefore, we have been limited to figures for American industries which were determined by the impinger technique, using low-power light-field illumina-

TABLE 7.—AVERAGE DUST COUNT IN CERTAIN DUSTY TRADES

Industry	Dust Count in Millions of Particles per Cubic Foot of Air
Slate-finishing mills:	
Floormen	1598.0
Loaders	1276.0
Disk crusher operators	312.8
Talc mining:	
Jackhamer drillers	2159.8
Muckers	44.8
Talc-finishing mills:	
Crushers and cylinder men	14.0
Packers	50.1
Marble carvers	19.1
Marble cutters	32.8
Granite quarrying:	
Leyner drillers	144.4
Jackhamer drillers	112.1
Plug drillers	36.9
Cement mill, average of all operations	26.0
Granite cutting:	
Hand-pneumatic-tool operatives	59.2
Machine-pneumatic-tool operatives	35.9
Attendant labor	17.0
Anthracite coal mining:	
Mining and miners' helpers	231.5
Attendant labor	31.1
Bituminous coal mining:	
Coal cutters and coal loaders	112.3
Attendant labor	3.9
Silverware manufacturing:	
Dusty processes	5.2
Nondusty processes	1.7
Municipal dust (street cleaners):	
Congested district	4.1
Residential district	1.8
Cotton industry:	
Carding room	8.6
Weaving and spinning room	4.5

TABLE 8.—DUST CONCENTRATION IN CERTAIN INDUSTRIES; GOOD AND POOR PLANTS COMPARED

Industry and process	Control measures	Quartz, percentage	Dust concentration, Millions per cubic foot		
			Maximum	Minimum	Average
Sand pulverizing:		80–98			
General plant air	Fair ventilation		20.0	2.2	12.0
Grinding	Exhaust ventilation (fair)				21.0
Bagging	None				695.0
Bagging	Exhaust ventilation (fair)				63.0
Bagging	Exhaust ventilation (good)				7.0
Abrasive-blast cleaning:					
Sand:		42.99			
Inside room	Average ventilation		310	232	969.0
Outside room	Average ventilation		28	23	25.0
Barrel	Poor maintenance		333	1.6	33.1
Barrel	Fair maintenance		12	0.6	4.1
Table	Poor maintenance		82	41	55.0
Table	Fair maintenance		102	0.2	12.0
Cabinet	Poor maintenance		365	6	127.0
Cabinet	Fair maintenance		44	0.7	5.2
Artificial abrasive or steel:		2.5–3.6 (from sand on castings)			
Inside room	Average ventilation		3272	48	155.0
Outside room	Average ventilation		15	8	11.0
Barrel	Average maintenance				15.0
Table	Average maintenance				11.0
Cabinet	Average maintenance				8.6
Rock drilling and handling:		0.84			
Open excavation-dry drilling	None		487	0.06	58.0
	Wet drilling				
	Good exhaust hood				
Underground:					
Granite	Wet drilling				47.0
Dolomite	Wet drilling				10.0
Dry drilling	Good exhaust hood		14	5.4	2.9
Mucking	Dry, good ventilation		4.6	1.7	83.0
	Dry, good exhaust hood				8.0
Metal grinding:		80–95			
Sandstone wheels	Wet grinding		250	4.3	79.0
	Poor ventilation		62	6.3	27.0
	Good exhaust hood		2	0.2	0.7
Foundries:					
Grey iron:					
General air			76	3	17-9
Core making			48	2	11-6
Molding			475	5	39-9
Shakeout			66	5	22-17
Grinders			184	5	63-6
Sand conditioning			263	32	74
Facing mills			2608	32	193
Steel:					
General air			34	1	9
Core making			15	1	8
Molding			160	2	22
Shakeout			83	2	20
Grinders			36	1	17
Facing mills			275	4	119
Malleable iron and nonferrous:					
General air			67	4	17-3
Core making			19	3	9-4
Molding			45	3	2-4
Shakeout			97	13	5
Grinders			13	6	3
Sand conditioning					

[1] Figures in this column are for plants with good dust-control equipment.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25355/12

tion (page 117). The data in Table 7 (18) summarize the results obtained in the dust studies conducted by the U.S. Public Health Service.

In Table 8 we have brought together other data from published and private reports. An attempt has been made to indicate the range in dustiness as well as the average figure and to show the difference between the conditions in good and poor plants. These figures are characterized by the wide range in dustiness exhibited by a single process, by the variations between good and poor plants, and by the variations from one industry to another.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25355/13