

Industrial Dust

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Industrial Dust

1. The literature on hazardous effects of dust is so extensive that no attempt has been made here to cover the subject in all of its ramifications, but to assemble only the outstanding data on several of its aspects. This pamphlet is intended for employers, plant safety engineers, personnel managers, and supervisors generally. The subject has been approached from the prevention angle, giving general information on occupational diseases caused by dust and on methods of control. No reference is made to the economic and legal aspects of dust diseases in industry and none of the uncommon dusts as, for instance, might be found in the pharmaceutical industry, are discussed. There is a short section on dust explosibility.

Physiological Effects of Dust

2. The inhalation of dust may produce different types of reactions in human beings:

- Pneumoconioses (silicosis and asbestosis) which result in specific lung pathology.
- Silicosis increases susceptibility to tuberculosis.
- The systemic reaction caused by such toxic dusts as lead, radium, manganese, cadmium, and mercury compounds, when either breathed or swallowed or possibly absorbed through the skin.
- A transient disorder known as metal fume fever, from the inhalation of finely divided metallic dust or fume particles such as zinc oxide.
- A reaction, allergic in nature, caused by breathing organic dusts such as pollen, flour, certain pulverized woods.
- An increase in bronchitis and other acute respiratory infections by breathing inert, non-fibrosis-producing dust such as coal, emery, limestone, marble.

3. In each of the above cases, the inhalation of dust can be the sole cause

This pamphlet is one of more than 150 Safe Practices and Health Practices Pamphlets. It is a compilation of experience in accident prevention from many sources. It should not be assumed, however, that it includes every acceptable procedure in the field covered. It must not be confused with American Standard safety codes; federal laws; insurance requirements; state laws, rules and regulations; and municipal ordinances. Additional copies of this pamphlet are available. Member price, 35 cents each. Quantity and non-member prices on request.

of disability. With the toxic dusts, trouble may result from swallowing or more rarely from skin absorption, as well as from inhalation. Inhalation, however, is by far the most important means of absorption of industrial dusts.

4. Irritation and ulceration of skin and mucous membranes may be caused by such dusts as lime and chromium compounds.

Pneumoconiosis

5. Zenker applied the name "pneumoconiosis" to the various changes in the lungs caused by the inhalation of dust. The term has now been shortened to "pneumoconiosis." It represents three words from the Greek, which mean

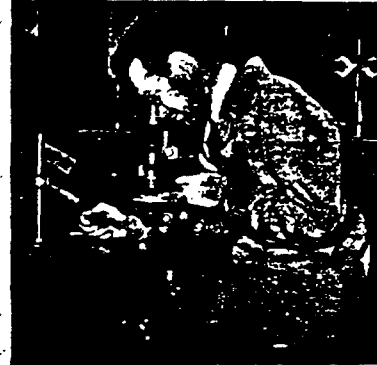


Figure 1. Dust counting by standard light field methods.

"lung," "dust," and "abnormal condition." The present generally accepted meaning of the word involves the concept merely of "dusted lung." The kind of dust inhaled determines the type of injury noted in the lungs. However, there is one pathological process which underlies lung disease caused by dust. This consists of a fibrosis or a replacement of the elastic tissue of the lungs by an impervious scar tissue.

6. Research on pneumoconioses has been carried on in the North American continent more actively in the past decade than in any other period. The hysteria with regard to silicosis and other occupational diseases has now largely subsided, and the subject is being more carefully considered and more soberly judged.

The Problem

7. Modern manufacturing methods are, in many instances, creating considerable dust. Much of this dust is often released in the immediate area where men or women are working, and this fact compels a practical solution of the problem.

8. The first step in the prevention of injury to health by dust is the control of its concentration so that workers will not be exposed to injurious amounts. In order to obtain wide spread control of dust throughout industry, it is necessary that information be developed and disseminated on the effect of various kinds and amounts of dust to which workers are exposed.

Origin and Properties

Physical

9. Dust is formed by reducing solid materials to small sizes. Processes like



Figure 2. Left: Operator in monument shed using pneumatic banker. Neither dust control nor respirator has been provided. Right: The same operation with goggles and dust-control hood in use.

grinding, crushing, blasting and drilling produce dust particles of sizes from the microscopic to the visible, their composition being the same as that of the parent materials if not altered chemically during the subdivision. Frequently the percentage of some hard mineral such as quartz in the fine dust may be less than that in the parent material. Common examples are the mineral or inorganic dusts derived from the disintegration of rock and the organic dusts like wheat and flour.

10. Smoke from burning carbonaceous fuels—coal, oil, wood, etc.—contains droplets as well as dry particles. Tobacco, for instance, produces a wet smoke composed of minute tarry droplets. The particle size of tobacco smoke is about 0.25 microns.

11. When a solid is broken into finely divided particles and released in the air, one of the important changes that take place is that the space occupied by the broken material and also the surface areas are increased many times from that of the original mass. For example, if one cc. (.061 cu. in.) of quartz is crushed into particles one cubic micron in size, there will be 10^{12} (1,000,000,000,000 or one trillion) particles with total surface areas of six square meters (9300 sq. in.) as compared with six square centimeters (.930 sq. in.) for the original block. If we assume a dust concentration of 100 million particles to each cubic foot of air, the one cc. of material will occupy an air space of 10,000 cubic feet.

12. With the exception of such fibrous material as asbestos, the dust particles must be smaller than 10 microns (one micron equals one twenty-fifth

thousandth of an inch) in their longest dimensions in order to enter the inner recesses of the lungs where the damage is caused. It was formerly believed that the hardness and sharpness of quartz particles were deciding factors in the injurious properties of dust, but this view has been generally disproved. (See paragraphs 20 and 29). Hay fever and other allergic types of disease from organic dusts or larger particle sizes can be caused by breathing particles into the nose and not having them reach the lungs at all. Ragweed pollen is from 18 to 25 microns in diameter.

13. By comparison a healthy red blood cell is about 7.8 microns, and a white cell up to about 16 microns. A person with normal eyes can see an object 50 microns in diameter. However, the reflection of light from a shiny particle of quartz may be seen when the particle is as small as 20 microns in diameter. It is to be realized, therefore, that individual dust particles small enough to reach lung tissue are essentially invisible to the naked eye.

14. Fumes may result from any of several chemical or physico-chemical changes such as zinc oxide, formed when burning zinc vapors arise from molten brass, or lead occurring in the exhaust of a motor car burning gasoline containing tetra-ethyl lead.

Chemical

15. The chemical and mineralogical make-up of the dust are the deciding factors in determining its injurious properties. It has been shown that silicon dioxide (free silica) in the form of quartz may produce a disabling fibrosis, whereas little or no fibrosis is produced by the equally hard and sharp cornered aluminum oxide (emery).

Classification of Dusts

16. The following classification of

dusts, according to their physical characteristics and physiological effects, has been arranged by Dr. R. R. S. V.

ORGANIC DUSTS

A. Non-Living Organic Dusts
The name implies these are composed of non-viable particles which may or may not be either toxic or irritant but nevertheless have effect on the human organism.

1. Toxic or Irritant Dusts. These are organic dusts which produce in toward symptoms either systemic or local. Those producing local symptoms are described as irritants, while those producing general symptoms are termed toxic. A dust may be both toxic and irritant.

2. Allergic.
(See paragraph 59.)

B. Living Organic Dusts

These dusts contain particles capable of reproduction or multiplication, such as bacteria and fungi. They are usually found in low concentrations and associated with non-living dusts in the air.

1. Bacteria. One of the most important is the anthrax bacillus, which is found in the dusts from skins, furs, wool and animal hair, horns, hoofs, bones, etc. Diphtheria, tuberculosis, typhoid and other bacillus-produced diseases may result from exposure to infected dusts. Bacterial-sensitized dust can also be the cause of allergic diseases, as asthma, hay fever, eczema, migraine headaches, etc.

2. Fungi. Dusts containing parasitic fungi may cause annoyance and discomfort. Mould on straw, hay, grass, vegetable debris is typical of this type of dust.

C. Inorganic Dusts

These are of mineral origin not requiring a living organism to produce them. Many dusts not classed as toxic come under this classification. Classified under inorganic are toxic and/or irritant, fibrosis-producing and non-fibrosis-producing dusts.

1. Toxic and/or Irritant. These are inherently toxic when inhaled, ingested or otherwise absorbed. Among them are the dusts from heavy metals and their compounds, such as lead, mercury, arsenic, cadmium, zinc.



Figure 3. Granite surface in operation, without and with dust control provided.

2. *Fibrosis-Producing Dusts*: The most important of these are the inorganic, slightly soluble dusts, which cause fibrosis in the lungs, of which quartz is the outstanding example.

3. *Non-Fibrosis-Producing Dusts*: These are inert, and by themselves seldom if ever cause fibrous tissue, but may lie free in the tissues or be absorbed. Included are aluminum oxide, coal, corundum, emery, limestone, magnesite, marble, plaster paris, polish of rouge, etc.

The Respiratory System

Retention of Dust

17. In the human respiratory system, there are complicated moist passages, on the walls of which dust particles will stick. Also, the nose contains hairs which catch some dust. However, the respiratory system can become overloaded with dust, and when this happens, the dust catching mechanism functions poorly.

18. C. E. Brown has estimated the average amount of dust a normal man will retain. Breathing at the rate of 20 respirations a minute, for example, he will inhale about 10 liters a minute and retain 60 per cent of the inspired dust. It is estimated roughly that the retention of common silica dust is about 50 per cent, but the way this dust may be divided between the upper and lower respiratory passages has not been clearly defined. Drinker has observed in the case of metal fume fever that slow, deep breathing, possibly five or six breaths a minute, is much more likely to produce a fever than the normal rate of about 15 breaths a minute. This is supposedly due to the higher retention of dust resulting from deep breathing. However, we cannot select by any known examination the men best suited to dust exposure, although some men show themselves obviously unfitted for dusty atmospheres.

19. The way in which dust particles are removed from the respiratory tract has been described by Dr. A. E. Barclay, British specialist on X-ray. The bronchi or respiratory passages, are covered with a number of tiny, hairlike cilia or microscopic whip lashes. These cilia make a fast stroke in one direction and a slower return stroke. They cover the passages leading to the lungs, and all keep in time with each other. This tends to push any foreign particles of dust upward in the direction in which the cilia bend, so that the particles may be expectorated. Dust particles which reach the inner recesses of the lungs are also

removed by means of dust valves (phagocytes) which ingest the dust particles and carry them to a system of fine drainage canals called the lymphatics. Both the cells and their contents are transported through these canals to sedimentary reservoirs known as lymph nodes.

Sizes of Particles

20. Microscopic dust particles are, of course, attracted by gravity, but because of the high resistance of the air, the smaller dust particles settle out more slowly. The settling motion of a dust particle through still air will vary with the size and shape of the particle.

21. In order that dust particles be inhaled, they must be small enough to float about in the air or be carried by air currents. It has been shown by research that dust particles must be smaller than 3 microns in diameter to cause silicosis. However, since all atmospheric dusts include a majority of particles smaller than this size, the particle size distribution of dust in the air is of little significance. McCrae showed that 70 per cent of the particles in silicotic lungs were found to be less than one micron and that the largest particles did not exceed 10.5 microns in their greatest dimensions. Results since obtained by other investigators have served to substantiate this comparatively early statement.

22. In silicotic lungs, dust particles under 3 microns greatly outnumber those of larger dimensions. Some have believed that the human respiratory mechanism is responsible for this size grading. It must be realized, however, that considerable size grading is done in the air before the dust comes into contact with the respiratory organs. An excess of small particles may be found in the lungs simply because smaller particles remain suspended longer and in greater amounts. Particles above five microns will not remain floating in the air for any great length of time. The air cells of the lungs are large enough to admit particles possibly up to 200 microns in length. However, according to Clark and Drinker, the most representative particle size in the lungs of men who have died of silicosis is one micron.

22a. Fine asbestos dust particles have been shown to produce only the inert reaction on the lungs characteristic of other silicate dusts. It is, consequently, believed that the asbestos



Figure 4. Ground silica dumped onto the apron of a conveyor. Note the enclosed conveyor exhaust hood and the use of an approved respirator by the operator.

fibers are the cause of the physical irritation resulting in asbestosis.

23. The finer particles of dust remain suspended in a still atmosphere for relatively long periods of time. Their chances of being inhaled are, therefore, greatly increased. The smaller dust particles also travel farther away from their point of origin, and inasmuch as the larger particles settle out very quickly, the farther away from the dust source, the greater will be the percentage of smaller particles.

24. Neither upper nor lower size limits have been suggested for toxic substances, as lead, etc. It is the opinion of Drinker and Hatch that no physiological size limits exist for these substances.

Silicosis

25. Undoubtedly the most important kind of lung disease caused by the inhalation of dust is that which has been named "silicosis."

Definition

26. Dr. Gardner's definition of silicosis is as follows: "Silicosis is a chronic disease of the lungs resulting from prolonged inhalation of fine particulate silica. It is manifested anatomically by formation of sharply defined fibrous nodules not over four to six mm. in diameter, which in most cases are uniformly distributed throughout all portions of both lungs; and clinically by a paucity of symptoms and physical signs that usually appear only in the late stage of the disease and by a tendency to become complicated by tuberculosis. In some cases, nodulation is concentrated in certain parts of the lungs, in which cases symptoms may be marked."

27. The International Silicosis Conference in 1930 defined silicosis as a

"pathological condition of the lungs due to the inhalation of free silica (SiO_2). Other authorities have given various definitions. That given by the American Public Health Association includes the statement that silicosis is "a disease due to breathing air containing silica." Sayers and Jones at the Saranac Symposium on Silicosis in 1935 stated that, "From the viewpoint of etiology, the harmfulness of a given dust containing free silica is directly influenced by the number of particles of free silica less than 10 microns in diameter that it contains."

Factors of Influence

28. Silicosis becomes noticeable after widely differing periods of exposure to silica dust, apparently depending on:

- The amount of dust inhaled.
- The percentage of free silica contained therein.
- The size frequency, or fineness of the particles inhaled.
- The nature and source of such other substances (including vapors and gases) as may be inhaled simultaneously or otherwise.
- The powers of resistance of the individual concerned.
- The presence or absence of a complication by an infective process.
- The presence of complicating conditions such as high temperatures or humidity, and unfavorable postures.

Physiological Effects

29. In earlier years it was believed that the physiological action of quartz on the lungs was caused by the hardness of the material. In fact, it was claimed that men who worked on sandstone were harmed not only by the silica particles, but also by sharp bits of steel. These ideas were contradicted by Dr. L. U. Gardner, of Saranac Laboratory, who showed that silicon carbide and fused aluminum oxide, both of which are harder than quartz, had practically negative physiological effects when inhaled. Dr. Gardner also showed that dust from the diamond, the hardest substance known, was practically inert when inhaled.

30. Silicosis is considered in three separate stages by medical authorities:

First Stage: The disease here produces no disability and does no appreciable harm. The affected man can carry on his operations as well as ever.

Second Stage: Respiration is affected. The victim may be bothered by labored breathing.

Third Stage: This stage may develop

following the second stage even if the victim has been removed from the dusty atmosphere. Here labored breathing becomes severe and the injured person is susceptible to pulmonary tuberculosis, frequently with fatal results. The worker is now far below normal and a possible victim to respiratory diseases.

31. Silicosis may be detected by X-ray in each of the three stages. However, X-ray appearances alone are not sufficient for a diagnosis of silicosis. The complete occupational and medical history of the employee should be carefully evaluated and related to the X-ray findings before a conclusion is reached.



Figure 5. An automatic bag loader handling finely powdered silica dust. An effective local exhaust system is in operation.

32. From most industrial experience the development of silicosis may not be anticipated short of five years. A few cases have been claimed to develop in as short a period as one and one-half years, but these were under extreme and unusual conditions. There has been evidence that alkaline materials, such as soap powder, accelerate the development of silicosis, but this has not been substantiated in recent experience.

33. Silicosis is a disease of which the victim may be unaware in the early stages. Its development is usually slow and unperceived. Inasmuch as silicosis cannot be cured by any means yet known and in its advanced stages is frequently complicated by tuberculosis or other infections, the importance of prevention is apparent. Also, in many industries, new developments have greatly increased the dust output. In granite stone cutting, for instance, and in rock drilling, dust production increased tremendously with the introduction of pneumatic tools.

34. Men with early first stage sili-

cosis, in the opinion of the Saranac Symposium, are not disabled and are not a menace to their fellow employees. The fact that barely perceptible X-ray change is visible is no grounds for assuming disability, since most persons over 40 show some chest changes regardless of dust inhalation.

Action of Silica on the Lungs

35. The theory of the action of silica on the lungs is as follows: Where quartz dust is inhaled and is passed through the upper respiratory tract, it reaches the terminal air sacs of the lungs where the exchange of gases between the blood and air takes place. At this point, as the dust is a foreign matter, it is ingested by what are known as "scavenger" cells which carry it through the walls of the air sacs to the lymph drainage canals located outside the air sacs. This results in a rapid increase of tissue cells which narrows the lymphatic channel, hampering elimination, as it is along these canals that foreign substances are removed. Also, when silica is being removed, the particle may kill the scavenger cell by which it is being transported, with the result that all dust particles are not removed from the lung. Instead, they may remain along the course of the drainage canal. Apparently, these small particles of silica dust are toxic in this location and the tissue cells in the immediate vicinity are altered and replaced by what is known as scar or fibrous tissue. The ultimate result is larger areas of tissue or lung volumes in which capillary action is definitely reduced—through which a normal exchange of gases between the blood and inhaled air cannot take place.

36. As the inhalation of silica continues, the amount of fibrous tissue will, of course, increase, with the ultimate result that the lungs will not readily oxygenate sufficient blood for the body need. This produces a shortness of breath in the affected individual in proportion to the amount of scar tissue in his lungs.

Free Silica vs. Silicates

37. With the exception of asbestosis, the silicate dusts have not been shown to cause a disabling lung condition such as is produced by free silica. Experiments conducted both by causing animals to inhale dust over a long period of time and by injecting dust in their peritoneal tissues have shown that sili-

...are inert and do not cause the characteristic nodulation produced by free silica. In addition to the animal experimentation, men who are exposed to silicate dusts containing no free silica have been x-rayed, and in a number of cases there has been opportunity for examination of their lungs. The nodular appearance on the x-ray and the actual nodules on the lungs, both of which are characteristic of silicosis, have not been found when exposure has been limited to silicates. Gardner has stated that evidence so far available indicate that, where a disabling lung condition is alleged to result from exposures to silicate dusts (other than asbestos), there is either an unsuspected presence of free silica in the dust, the victim has had a previous undisclosed exposure to free silica dust, or there has been inadequate interpretation of the x-ray. On the other hand, Dreeson et al. have reported pneumoconiosis in workers exposed to high concentrations (150 million particles per cubic foot of air) of clean washed mica. Greenburg and his co-workers found fibrosis associated with such symptoms as dyspnea and chronic cough in 14.5% of 221 tremolite talc miners and millers. These men were all employees of 10 or more years exposure and the reported dust concentrations are as high as 1300 million particles per cubic foot of air. In two mines in this area, Gardner had found quartz ranging from 12 to 20 per cent, but in the mills, where all rock is mixed and crushed, the dust showed low quartz percentages both in Gardner's studies and those of Greenburg. This subject of the possible action of silicate dust on the lung tissue is being further investigated, and any indications that certain silicate dusts may be causing a disabling lung condition should be given thorough study before conclusions are reached.

Mixed Dusts

37a. Where free silica is mixed with other dust, the greater the percentage of free silica, the less the dust exposure required to cause silicosis. Since some dusts such as gypsum have an inhibiting effect, there is an increasing tendency to evaluate the extent of the hazard presented by mixed dust on the basis of the individual dust exposure. More data are constantly being accumulated on the actual effects of various types of mixed dusts.

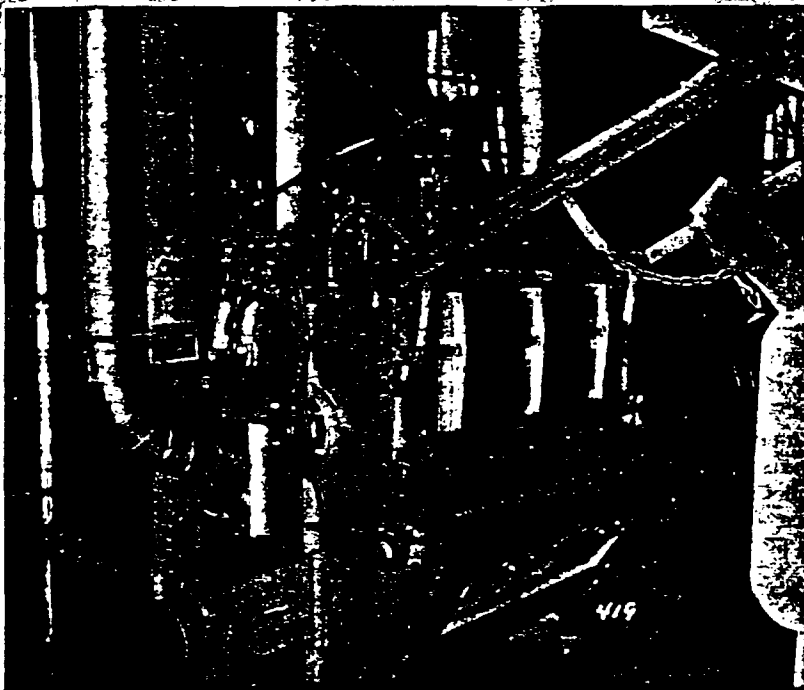


Figure 6. A tumbling barrel used for finishing small castings. The barrel is in operation, but note the absence of dust due to a good exhaust system.

Asbestosis

38. Asbestos is the only silicate at present recognized as causing pathology, and even its reaction is definitely different from that due to silica alone (Drinker). Its behavior is the exception to the rule that silicates are inert, at least in uninfected tissues. Asbestos, when inhaled, produces fibrous tissues in the lungs of both men and animals. Asbestos is made up of hydrated silicates of magnesium with variable amounts of iron, calcium, sodium, potassium, and aluminum replacing portions of the magnesium. The property common to all of this group is the fibrous structure. It has been shown that the presence of fibers of asbestos are necessary for the production of asbestosis and this fact, coupled with the observation that fine asbestos dust is analogous in its inert reaction on the lungs to that of other silicate dusts leads to the conclusion that asbestosis is the result of physical irritation of the lung tissue and not of chemical action as in the case of silicosis.

39. Following a study by The United States Public Health Service of the asbestos textile industry, it has been recommended that the dust concentration be kept at less than 5 million particles per cubic foot of air if asbestosis

is to be avoided. The use of a standard based on the number of fine particles result from the use of dust determination technique developed for the study of silica dust exposures. Although future research will undoubtedly set up a standard based on the number of asbestos fibers in air, keeping the fine dust at less than the suggested standard will keep the concentration of injurious fibers within safe limits.

Infection

40. In the opinion of most investigators, the factor of infection is most important. It has been observed in guinea pigs with chronic pneumonia that in an infected area which developed before exposure to dust, the local branches of the lymphatic system were not so efficient as those in the rest of the lung. Silica inhaled into tissue thus damaged appeared to accumulate in unusual amounts.

41. Since men with silicosis are more susceptible to tuberculosis than are normal men, there is often an excessive incidence of tuberculosis among such workers. Usually disability and death of a silicotic, when due to pulmonary conditions, results from the complicating tuberculosis. For this reason, it is especially important to have a re-em-

ployment and periodic x-ray program among workers exposed to silica dust.

42. Gardner states as follows: "At the present time the treatment of tuberculosis in silicotic subjects is comparable to that for ordinary consumption 50 years ago. The prospects are far from hopeless. Silicosis is gradually being brought under control. The time is not far distant when dangerous concentrations of silica dust will no longer be permitted in American industries. Periodic examinations of those already employed will detect new infections in persons now silicotic. Prompt institution of treatment will eliminate most of the hopeless cases of silico-tuberculosis. Pre-employment examinations will insure placement of men in positions that they can fill without damage to their health."

Coal Mining

43. Anthraco-silicosis is a term used for a form of pneumoconiosis commonly called "miners' asthma." According to Public Health Bulletin No. 221, "Anthraco-Silicosis Among Hard Coal Miners," it is a chronic disease due to breathing air containing dust generated in the various processes involved in mining and preparing anthracite coal. It is characterized anatomically by generalized fibrotic changes throughout both lungs and with the presence of excessive amounts of carbonaceous and siliceous materials.

44. Symptoms found in early stages are shortness of breath, cough, pain in the chest, and possibly physical weakness. In the advanced stages of the disease there is loss of weight and decreased capacity for work, due partly to pulmonary infection.

45. In studies conducted by the United States Public Health Service in Pennsylvania, no cases of anthraco-silicosis were found in the controlled group of hard coal mining employees whose dust exposure averaged less than five million particles per cubic foot of air. However, the prevalence of anthraco-silicosis among the entire group of employees was found to be about 23 per cent. Among all except rock workers, less than two per cent of the men were affected when the duration of employment was less than 15 years, regardless of the amount of dust in the air. Among rock workers, who were exposed to dust averaging 35 per cent free silica the prevalence of anthraco-silicosis va-

ried from 10 to 72 per cent depending on length of exposure and dust concentration—the higher percentages of incidence being found among workers exposed more than 25 years to concentrations greater than 300 million particles per cubic foot of air.

46. According to A. E. Russell, the disability rate among anthracite miners from respiratory tuberculosis is about



Figure 7. A method of receiving dust into trucks and auxiliary collecting equipment.

4½ times the rate for general manufacturing. The pneumonia rate is less than one-third of general manufacturing. Bronchitis, however, is a rather common complaint.

47. S. L. Cummins and S. F. Sladden conducted an examination of a large number of coal miners' lungs in South Wales. As a result, they made the statement—"Coal is retained in large amounts only when there is a really high silica content." They added, "We believe that in the absence of the silica factor, there would be, under modern mining conditions, no serious degree of anthracosis." E. L. Collins and J. C. Gilchrist have claimed that workers who have had considerable exposure in trimming coal ships where there is practically no silica exposure, but nevertheless a very heavy dust condition, showed some fibrosis which, however, was not considered disabling.

48. A study of 2500 post-mortem examinations made in Pittsburgh hospitals shows that city air which may contain an unusually large amount of coal dust may cause lungs to become darkly pigmented but yet produce no pathology of importance other than perhaps a predisposition to colds, pneu-

monia, bronchitis and similar ailments.

49. Also, Dr. Greenburg reminds that there is an important factor other than the one of environment, which is that of individual susceptibility. Investigators have noticed that men working in the same plant, at the same time and in the same work place, frequently do not develop silicosis to the same degree. It is possible for one worker to be an early victim, while the man working beside him remains comparatively free. This angle of the problem indicates a need for further research.

50. It is well, nevertheless, to consider all coal mining as involving a risk to some degree, depending upon the type of rock being drilled. While the hazard may not be as severe as in most metal mining operations, it is of importance at least because of the number of workmen concerned.

51. The hazard of silicosis seems to be practically absent in the manufacture of cement. A recently completed survey of 2000 cement plant employees in all sections of the country by Dr. LeRoy U. Gardner and staff of the Saranac Laboratory for the Study of Tuberculosis, has failed to demonstrate that the inhalation of cement plant or quarry dust has a significant effect on the respiratory tract. Neither did it appear that men who had spent their industrial lives in such atmospheres were unusually susceptible to respiratory infection. The incidence of influenza, disabling colds and pneumonia was not high. No cases of clinical tuberculosis were discovered, and the incidence of healed or latent foci as revealed in the roentgenograms is the same as that found in the general adult population; namely, 4.5 to 5 per cent. Dr. Gardner has stated:

"Although the medical examinations reveal no evidence of respiratory injury from cement plant dust, even after periods of long exposure, maximum concentrations in dusty areas should be reduced to 100 million particles per cubic foot (light field count, impinger technique) as rapidly as economically feasible. High concentrations may not be dangerous, but they are not compatible with good housekeeping standards.

"An exception to the general rule is indicated by the discovery of two cases of non-clinical dust reaction in this group of 2,000 persons. These men had been exposed to quartz dust (usually in the form of sandstone) used in the manufacture of special cement. While possible inhibitory effects of

...elements of cement mill dust... against poisoning, by providing bath and lunch rooms, clean overalls, mouth washes, and such, instead of preventing the escape of lead into the air the men were obliged to breathe. 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 16 times a minute during the eight or ten hours of the working day.

Toxic Dusts

Breathing vs. Swallowing

Generally, workers may be poisoned by toxic dusts much more quickly through inhaling them than by swallowing them. Dust swallowed with food goes into the stomach from which the major part of it is directly eliminated. Some may be picked up in the blood circulation and then moved on to the liver. The liver, however, is an effective filter and detoxifier, and it is only the poison that gets beyond this point that enters general circulation. On the other hand, dusts that reach the lungs can pass directly into the blood stream, to the heart and then to all parts of the body.

53. Alice Hamilton, in "Industrial Toxicology" (Macmillan Company) points out the distinction between the two ways in which poisonous dust can be taken into the body in the following paragraph—"A great deal of money has been wasted by well-meaning employers who sought to protect lead fur-



Figure 8. View in an iron foundry showing bench grinders with exhaust hoods.

nacemen, white lead grinders, etc., against poisoning, by providing bath and lunch rooms, clean overalls, mouth washes, and such, instead of preventing the escape of lead into the air the men were obliged to breathe. 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 16 times a minute during the eight or ten hours of the working day.

54. Numerous authorities give evidence that there is far more danger of being poisoned by inhaling toxic dusts than by eating them with food or taking them in liquids. But all precautions should be taken to prevent toxic dusts from entering or even coming into direct contact with the body.

Lead

55. The practical problem in connection with lead poisoning is the prevention of serious lead exposure. The problem consists of two phases: one, the determination of the limits of safe exposure, (see paragraph 68) and, two, the reduction of lead exposure below these limits.

56. We must recognize however that lead is a normal constituent of a living animal and that there is a level of lead absorption which is safe. Lead intoxication and lead absorption can be prevented by engineering methods. (See Health Practices Pamphlet No. 3 and Safety Instruction Card No. 116 on this subject.)

Metal-Fume Fever

57. Metal-fume fever may be one of the results of breathing dust from metal sources. This malady usually follows contact with a heavy concentration of magnesium oxide, copper oxide, lead, zinc oxide, and possibly lead oxide and manganese dioxide.

58. The symptoms are chills followed by a malaria-like fever which usually passes off within 24 hours, allowing the worker to return to his job on the following day. The first attack usually immunizes the victim so that attacks are not experienced consecutively. However, after a lay-off or an absence from contact with the dust, a further attack is likely. Slow, deep breathing, according to Drinker and Hatch, seems to bring about the symptoms more quickly.

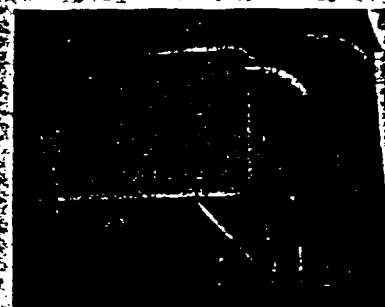


Figure 9. Dust collector units mounted on the roof.

Allergic Diseases

59. Conditions arising from allergic disturbances of an occupational origin are coming more into prominence, from the compensation point of view.

60. Persons said to be allergic are those who are particularly susceptible to certain substances with which they come in contact. These substances may include foods, drugs, and vapors and dusts of various kinds. A wide variety of substances may, therefore, be responsible for allergic reactions in various individuals, which substances may have no effect on others or, in some cases, may even be of positive benefit. The types of reactions experienced by affected individuals to given substances may also vary greatly both in nature and in degree. A single substance may, in one case, cause intestinal disturbances, in another case develop asthmatic attacks, and in a third case cause a skin eruption. Still another person may be affected in all three ways.

61. Dusts of various kinds sometimes cause attacks of asthma. Asthma may be suffered by stablemen from exposure to horse dander and by furriers from exposure to fur dust. Other persons cannot work near animals or, in fact, keep pets or sleep on feather pillows without suffering acute attacks. Hay fever is another type of reaction to dust on the part of certain individuals. These dusts are frequently in the nature of pollen, as from plants. Hay fever, it claimed, however, may also be caused by other types of dust. In any event, the precise manner in which an allergic individual may react to a substance to which he has become sensitized cannot be predicted.

62. Many believe that allergic individuals are born with their particular

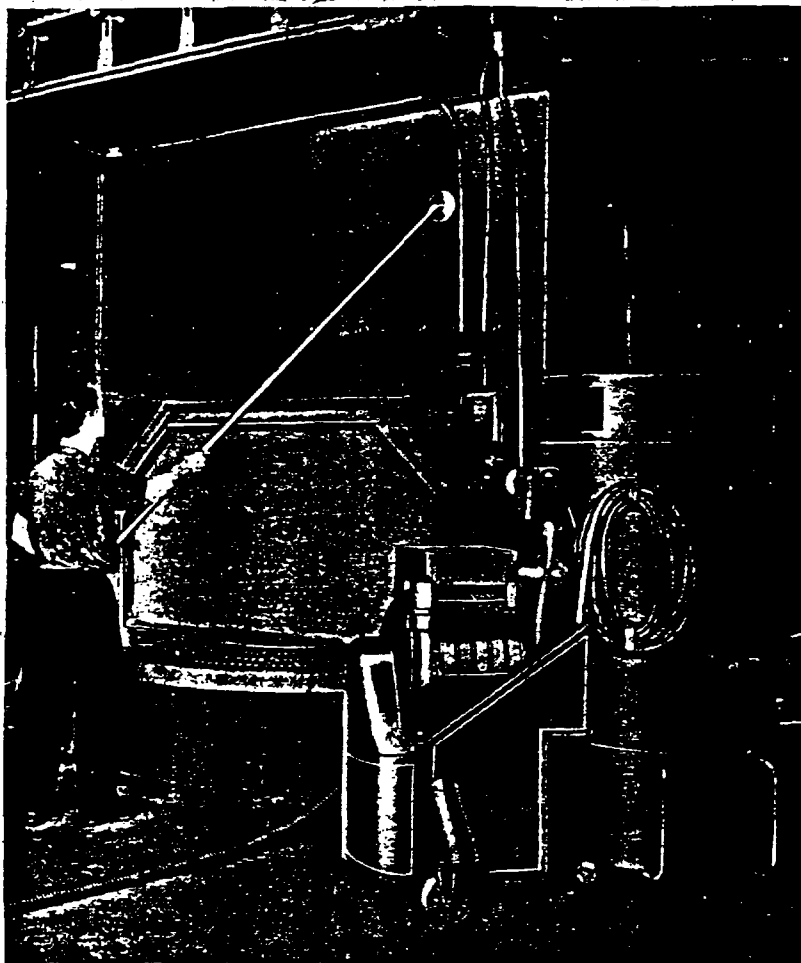


Figure 10. Portable industrial vacuum cleaner.

types of hyper-sensitivity. While this is probably so, according to Dr. May R. Mayers, individuals do not become sensitive to substances which later cause the trouble until they have had sufficient contact to become sensitized. When a person who is hyper-sensitive first comes in contact with the substance, he is not aware of his condition. An "incubation period" is required, during which time repeated contacts perhaps sensitize him to a point where subsequent contacts may result in immediate allergic reaction. This waiting period may vary from a week or two to several months or even years. However, the allergic tendency must have been in the individual to begin with, and then there must be continuous or intermittent contacts with the substance in question in order that the latent allergic tendency can be brought out.

63. It has been claimed that once a person has become sensitized to a given

substance, he may, more readily than before, become sensitive to other substances. Occasionally, too, it has been suggested certain individuals are capable of developing a certain degree of immunity as time goes on. However, from the viewpoint of prevention, it seems logical that workmen who have become allergic to certain substances in the course of their occupations should be encouraged to go into other lines of work, for in most cases the tendency is to become more sensitive as exposure continues.

Permissible Dustiness

64. Generally, we lack data for defining rigidly permissible dustiness. The point has not been reached where a manufacturer can be told the exact concentrations at which, for instance, his men will start developing cases of silicosis, asbestosis, or lead poisoning. He can only

be told that the maintenance of certain degrees of air cleanliness represents the best known practice. Moreover, while such standards would undoubtedly be of value for a single industry under different conditions and in other industries there would be a question of their applicability.

65. As the result of many years of practical experience in dust control in the South African gold mines, a concentration of one mg. of dust per cubic meter of air was accepted as standard and is now known commonly as "the South African standard." In 1934, L. G. Irvine stated that no miner who had entered the industry since this low concentration had been maintained had, as yet, contracted silicosis.

66. It was stated by D. E. Cummings in a paper presented at the Second Symposium on Silicosis at Saranac Lake—

"Information from field studies made in many parts of the world indicated that a normal man might work in pure crystalline silica dust for many years without impairing his health if the concentration did not exceed five million particles per cubic foot of air."

67. Dr. L. U. Gardner states, concerning the permissible dustiness where silica is present:

"The evidence now available makes it seem improbable that a silica hazard is defined solely by the number and size of the silica particles in industrial atmospheres. The other components of a dust modify its action. Some may inhibit, others retard, and perhaps some will be found to prevent its injurious effect. Because these possibilities are recognized, it becomes difficult to set up definite standards of permissible dustiness in industrial atmospheres."

Dr. Gardner continues further:

"Not enough is known about the action of protector substances to warrant the recommendation that they be employed to prevent silicosis in industry. Our aim should be to reduce existing dust concentrations rather than to increase them by adding more dust. I would very strongly oppose the recommendation that protector dust be used to attempt to prevent silicosis at the present time. But I do believe that we should attempt to discover more about their action. Some, like gypsum, seem to combine with silica in the atmosphere, forming clumps which are too heavy to remain suspended in air and too large to pass the barriers of the nose and upper respiratory tracts. With such information at hand, it may be

possible, to apply the knowledge in a practical manner.

68. Other threshold limits for specific dusts have been stated as follows:

Asbestos—(Dressen, et al.) United States Public Health Service) five million particles per cubic foot.

Coal dust—(R. R. Sayers, et al.) 50,000,000 particles per cubic foot for coal dust containing not over 5% quartz, 10,000,000 to 15,000,000 particles for dust with 13% quartz, and 5,000,000 to 10,000,000 particles for hard rock workers exposed to dust containing 35% free silica.

Lead—(Legge and Duckering) more than .5 mg. per cubic meter is hazardous and (A. E. Russell et al.), less than .15 mg. per cubic meter is safe.

Zinc oxide—(P. Drinker, et al.) 14 mgs. of zinc oxide per cubic meter for 8 hours of exposure or 45 mgs. for short exposures.

69. Of the metals, the following suggestions have been made:

The American Public Health Association Committee on Lead Poisoning state in their comprehensive 1943 Report that "when the air of workrooms regularly contains more than 1.5 milligrams of lead per 10 cubic meters of air, cases of disabling lead intoxication do not occur among men who work regularly in such workrooms, and cases of questionable or minor intoxication are rare. In practice the attempt is made to maintain the lead content of the air within such limits as will yield an average of not more than 1.5 milligrams of lead per 10 cubic meters throughout the working day, while preventing the occurrence of materially higher concentrations (5 milligrams per 10 cubic meters or more)."

Zinc Oxide: To avoid metal fume fever the concentration should be kept below 14 mg. per cubic meter for 8 hours' exposure or 48 mg. for short exposures (P. Drinker et al.).



Courtesy, Kirk and Blum Manufacturing Company

Figure 11. Exhaust system for removal of lead dust and fumes.

Manganese: 50 mg. per cubic meter is safe (Drinker & Hatch).

Control of Dustiness

70. There are various ways in which dust diseases and annoyances may be avoided*:

1. The avoidance of exposure furnishes the first and most important defense against industrial disease. Jobs should be performed in clean air as far as possible. It is not necessary, however, to prohibit all exposure to a toxic substance, as the human system can protect and cleanse itself to a certain degree.

2. Exposure can be limited by reducing working hours where dust concentrations cannot be sufficiently reduced to constitute an otherwise safe condition.

3. Safe processes may sometimes be substituted for those of an unsafe nature. An example of this is the substitution of abrasive wheels made from synthetic abrasives containing no quartz, for wheels made from natural sandstone, which is about 95 per cent quartz.

4. The substitution of wet methods for original dry methods has exerted considerable control over dusty atmospheres. Dry drilling is the cause of considerable dustiness in mining, which may be greatly reduced by wet drilling. This is also true in many construction and quarry jobs.

5. An effective method for preventing the escape of undesirable dust into the working atmosphere is the local exhaust hood. It should be placed close to the source of pollution to be most effective. The air should be discharged through a dust collector to prevent contamination of neighboring areas or return of the dust through open windows into the plant.

6. The routine recording of dusty concentrations is a valuable check on the existing exposure. Periodical counts of air samples are a great help in showing whether or not there is need for further control.

7. In some cases, the application of general rather than local exhaust ventilation is desirable. There are instances where the source of polluting materials is constantly changing position and local exhaust becomes difficult and impracticable. Here a general ventilating system may be the only answer.

*The eight methods of prevention given here are listed in this order in "Industrial Medicine" by Clark and Drinker, published by the National Medical Book Company, Inc., New York, N. Y.



Courtesy, Kirk and Blum Manufacturing Company

Figure 12. Adjustable exhaust hoods for polishing wheels.

8. A final method of protection is the wearing of special protective equipment by workers. This step is frequently the last resort and is used only in an emergency or for jobs of short duration, but it is nevertheless of considerable importance. (See American Standard Safety Code for the Protection of Heads, Eyes and Respiratory Organs.)

71. In many localities codes or ordinances for the control of dustiness are in effect which should be investigated and followed by employers seeking to improve working conditions.

Local Exhaust Systems

72. Local exhaust ventilation for the control of industrial dusts and fumes consists of four principal parts:

- Exhaust hoods
- Piping or ducts
- Air cleaning plant
- Source of suction

73. While each of these parts should be designed and installed to perform its required function with respect to the system as a whole, the exhaust hood probably demands the most careful design, inasmuch as the degree of control secured depends to a larger extent upon the shape and location of the hood—and the rate of air flow to it—than upon any other single factor.

74. Hoods have to a considerable extent followed the design first employed in such industries as woodworking and metal grinding. However, as noted by Theodore Hatch, in recent years the industrial dust problem has assumed hygienic and economic aspects entirely beyond the limitations of those industries in which the production of dust has been

a nuisance rather than a serious health hazard. Occupational diseases have come to mean so much to industry in medical costs and compensation as well as the health of workers, that there is ample justification for extensive research and investigation in the development of the best possible design in control equipment.

75. To maintain a safe concentration of dust in the vicinity of dust generating equipment it is necessary to introduce sufficient clean air from the outside to dilute the dust-filled atmosphere to the degree necessary for safety. In the utilization of general ventilation systems, care must be taken to avoid excessive exposure of workers as dust is removed from its source. Especially where local dust systems are being supplemented by general ventilation, it should be seen that eddy currents are not set up to interfere with proper functioning of the local exhaust.

76. Undoubtedly, the best method of ventilation is the control of contaminated air at its source. To do this, there must be sufficient movement of air toward the exhaust opening to counteract any tendency of the dust-laden air to escape into the surrounding atmosphere. Proper hood design will make this possible with the least possible flow of exhaust air. The object of an exhaust system is to trap the contaminated air and not to remove the harmful dusts from it. It is suggested that the scattering of large particles may be prevented by the erection of suitable barriers or, of course, by causing them to flow into the collecting duct. For efficient operation of dust collecting equipment, all air motion around the source

of dust production must be under absolute control.

77. Proper design and location of the suction opening and the rate of air flow into it is a matter of utmost importance. Laws governing the flow of air into exhaust openings have been given in "Industrial Dusts" by Drinker and Hatch:

1. Enclose the process as completely as possible and provide internal baffles to guide the air flow where it is most needed.
2. An exhaust hood which does not enclose the process should be placed with its opening as close as possible to the point of dust generation since the air velocity in the zone of hood influence decreases approximately with the square of the distance from the face of the hood.
3. Shape the hood to conform with the shape of the area of dust production so as to secure reasonably uniform air velocity over this area. For a given required air velocity at the point of dust generation, the velocity at the hood opening should be as low as possible. This is contrary to the common idea that the hood suction should be high.
4. Provide flanges wherever possible to reduce the air flow from ineffective areas where no dust is produced.
5. Locate the hood opening, or part of it, so as to receive directly any dust that is thrown off along a well-defined path, thus utilizing the directional energy of the material for its own capture.

"Design of Exhaust Hoods for Dust-Control Systems" by Theodore Hatch, Harvard Graduate School of Engineering and School of Public Health, Boston, Mass. The Journal of Industrial Hygiene and Toxicology, November, 1936.



Figure 14. Sweeping with the use of coiled sawdust to allay dust.

Wet vs. Dry Drilling

78. On the matter of drilling, Harrington says:

"Dry drilling is, of course, very much more of a dust producer than wet drilling—possibly in the ratio of 10 to 1. Wet drilling produces some dust, but if reasonable precautions are taken with wet drilling, and suitable precautions are also taken as to ventilation, as to the wetting of the muck pile, as to the time in which blasting is done, as to the precautions which are taken after going back to the face after blasting . . . there need be no particular harm to workers in those places, as far as dust diseases are concerned."

79. Prof. Philip Drinker, in commenting on the adoption of the New York State Code governing dustiness in rock drilling, stated:

"This code divides all rock formation into two classes, class 1 being those with less than 10 per cent free silica, by weight, and class 2 being those with more than 10 per cent free silica. If drilling is done in class 1 rock, dust counts must be below 100 million per cubic foot, and below 10 million particles if the rock falls in class 2."



Figure 13. Dust from rock drilling operations, uncontrolled and controlled by dust trap

This means in effect that dry drilling can only be done with local exhausts and adequate cleaning will happen; also, that dry drilling without ventilation will not achieve adequate air cleanliness in the case of the high quartz or class 2 rock.

80. The situation is well summed up in an editorial that appeared in *Chemical Engineering and Mining Review* (Melbourne).

Whatever the ultimate findings as to the cause and development of pneumoconiosis, the task of the mining engineer is indicated clearly: the first essential is to minimize the formation of dust, and the second is to collect or remove unavoidable dust as rapidly as possible. In this latter respect, a logical forward step has been taken in the adoption of means to collect the dust at certain points of production instead of relying upon the general ventilation current to dilute and transport the injurious material. Small units are now being installed in some mines at loading stations and orepass tipplers, the installation and maintenance costs of which are low and a high efficiency is developed.

More efficient rock-drilling practice and improved general standard of health, reduced working hours, provision of change houses and other facilities, and many minor but important factors can also contribute to a reduction in the incidence of silicosis.

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