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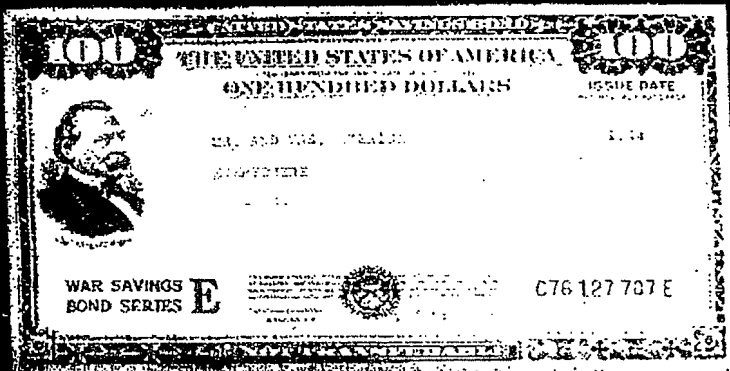
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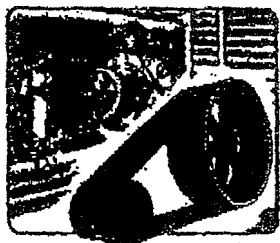
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Dust as an Industrial Health Hazard

F. W. HUTCHINSON

Assistant Professor of Mechanical Engineering,
University of California, Berkeley, Calif.

THE health problem arising from dust in industry is one of increasing seriousness. Socially, economically and legally it has become evident that immediate steps must be taken to combat the hazard represented by wholesale industrial exposure to the pneumoconiosis-producing dusts. Pneumoconiosis, in many of its forms, is incurable. Thus consideration of the problem which it represents is based largely on methods of dust reduction and removal and is therefore as much a responsibility of the engineer as it is of the physician.

Fortunately, medical knowledge and engineering equipment have progressed to such a point that recognition of hazardous conditions and availability of methods for correcting them will now permit a complete, practical and economical solution of the problem. Unfortunately, prejudice and fear have surrounded the entire subject with such an aura of suspicion and distrust that neither workmen nor employers have until recently been willing to acknowledge the hazard or recognize the need for preventive measures. In addition, the slow action of the disease coupled with failure on the part of many physicians to acquaint themselves with the proper diagnostic methods has made it difficult for the worker to establish his claim for compensation in the event of lung injury and equally difficult for the employer to defend himself against fraudulent claims; it has been said that silicosis claims constitute one of the most widespread of legal rackets.

Workmen have bitterly opposed adoption of the exacting employment qualifications and periodical physical examinations of those exposed to dust which are essential to medical control. Yet, it is essential that persons having a predisposition to tuberculosis or a low nasal filtering efficiency be excluded from the dusty trades. Similarly, protection of the worker demands that he be removed from exposure at the first indication of lung damage.

Employers, on the other hand, have adopted the point of view that most claims of silicosis are unfair and an unscrupulous attempt to take advantage of them. The explanation behind an employer's unwillingness to acknowledge the hazard is not unreasonable. The dust diseases are almost invariably of long duration and frequently are not discovered until many years after exposure has ceased. Thus many employers are now in the difficult and alarming position of facing claims for disease which is a result of exposure to conditions that existed in their plants 10 or 15 years ago; at that time they were unaware of the health hazard associated with dusty atmospheres and consequently, though they might have had every

desire to furnish their employees with full protection from occupational injury, they did not take the "special care" which is now known to be necessary. Wilson states: "Most well-intentioned employers may be said to have used 'reasonable care' in past years and—considering their lack of knowledge—this is now their best defense in damage suits. However, from now on, with the spread of . . . available knowledge . . . only 'special care' will safeguard the interests of employer and workman alike."

Recent interest in dust diseases has built up an extensive literature; theories have been confirmed with experimental evidence and innumerable case histories of individuals and of entire groups are available. In contrast to the widespread ignorance of ten years ago, it would probably be difficult at the present time to find a worker in the dusty industries who has not at least heard of silicosis, or an employer who is unaware of the crippling damage suits which will face him if his employees, through failure on his part to take the necessary preventive measures, contract the disease.

The cost of dust control is often far less than the cost of one damage suit. Jury awards in silicosis cases of \$10,000, \$17,000, \$22,500, \$25,000 and like sums are matters of record. In the St. Louis area alone there were, some time ago, over 2000 suits pending. A Missouri mining company, as another example, faced claims aggregating \$5,000,000 and spent \$500,000 in out-of-court settlements of a few of them.

The purpose of this article is to review the literature on the physiological effects of some of the hazardous industrial dusts and to summarize present-day criteria acceptable as a basis for the design of dust control systems.

Silicosis

Silicosis is a disease of the lungs caused by the inhalation of dust containing free silica. Some authorities believe, but this fact has not been established, that the inhalation of silicates is also a causative factor.

Incurable and Progressive. The important facts about this disease are that, for practical purposes, it is progressive and incurable. The qualification "for practical purposes" is necessary because recent investigations suggest that uncomplicated silicosis is not progressive; however, the continued existence of a case of silicosis without the onset of complications is so rare that, from the standpoint of social consequences, the disease can in itself be regarded as progressive. In the medical literature the "progressive"

viewpoint is the one which has been most widely adopted but the possible future significance of the recent findings is promising.

With reference to the "incurability" of silicosis, recent work has also indicated the possibility of a solution but it is as yet too soon to hold out hope for satisfactory medical treatment. One promising method consists in the administration of thyroxene; the investigators report that:

"Thyroid therapy may possibly not only prevent the development of silicosis, but may also arrest its further progress and possibly reduce to some extent the degree of fibrosis already existing."

The above remarks on the "progressiveness and incurability" of silicosis have been included because they show how very far the medical profession has yet to go to achieve a solution of the silicosis problem. In terms of concrete present-day gain, the sum total of the best that medicine can now offer is not of much value. When dealing with contagious diseases such as smallpox and Spanish influenza, it has been possible to achieve a satisfactory treatment as a first step toward the elimination of the disease itself; in the case of silicosis, a non-contagious disease, the only advance of real significance which is possible is an advance which must be brought about by the application of basic engineering principles—the reduction of dust concentrations at the breathing zone to a point such that a health hazard no longer exists; only in this way can silicosis be eradicated.

If permitted to run its course without complication, silicosis will lead to death by suffocation—the lungs becoming incapable of maintaining a gas transfer sufficient to sustain life. Usually, however, termination occurs as a result of complications such as tuberculosis or heart disease; a definite correlation has been established between silicosis and tuberculosis, but an explanation is, as yet, lacking.

With reference to tuberculosis a study of axe grinders and polishers showed a death rate of 1,900 per 100,000 from tuberculosis as contrasted with 150 per 100,000 for the general population of the same state. In this study the death rate of all workers in the dusty trades investigated was used. More striking figures and more significant ones were obtained from a Massachusetts investigation in which the tuberculosis death rate of 961 silicotic quarry workers was compared with that for the general population and found to be forty times as great.

Background and Extent. The name silicosis is of relatively recent origin, but the existence of the health hazard associated with dusty work has been recognized by medical men for many centuries. From the literature of the period we find that the Ptolemies knew of it. Pliny the Elder referred to the "stone cutters' disease" and devised a crude respirator for the protection of miners; Aristotle warned Greek workmen against the inhalation of dust. Hippocrates, three centuries before Christ, noticed that his surgical knives were dulled by dust in the lungs of stone workers. In the 16th century Georges Agricola, a mining engineer, wrote "De Re

Metallica" and, with reference to dust, warned his readers to beware because it "penetrates into the windpipe—eats away the lungs—implants consumption in the body." He called attention to the fact that the death rate among Carpathian miners was so great that a wife could often outlive six husbands.

The passage of time has done little to alleviate the hazard. The development of pneumatic tools has led to a great increase in the dust concentrations attending the grinding, polishing and cutting of stone. The death rate among Barre granite cutters, for example, increased from 1.5 per 1,000 in the year 1890 to 19.6 per 1,000 in 1924, an increase attributable to the introduction of pneumatic equipment. When one realizes that a threshold concentration of 5 million particles per cubic foot is required to constitute a silicosis hazard (for dust made up of 100% free silica), it is then evident that there are many industrial operations today which represent health hazards that did not exist prior to the introduction of mechanical equipment.

Many estimates have been made of the number of persons exposed to hazardous concentrations of silica dust, but such estimates are usually based only on industrial exposure. When account is taken of the fact that 60% of the earth's crust consists of silica the likelihood seems great that such estimates are conservative. Lanza and Vane estimated that the number of industrial workers exposed to the silica dust hazard is in excess of half a million in those industries in which the processing or handling of siliceous material is of basic importance; this estimate does not take account of the many workers exposed as a result of the industrial use of silica as a filtering agent.

In one other respect the silicosis hazard has not yet been evaluated. As already mentioned there is a definite relationship between silicosis and tuberculosis. This fact leads to the probability that in any community in which there is a large number of silicotics there will be a decided familial tuberculosis hazard. Fortunately, however, this danger is somewhat lessened as a result of the long incubation period which is found with silicosis—by the time a workman's lungs have reached the stage at which tuberculosis is most likely to set in it is more than probable that his children will have grown past the age at which the danger of their contracting the disease is greatest.

That silicosis is definitely related to a particular kind of occupational risk is demonstrated by the data from examinations (X-ray) of 2500 workmen from all departments of a typical "heavy industry"; 5.8% had silicosis, but 90.0% of those affected were foundry workers. The great importance of the silica hazard is clearly indicated from the following comparative mortality figures (from a 1933 publication of Hollis and Yule):

1000 for standard population (all causes for
ages 20-65)
1984 for silica group
967 for non-silica group

The silica group (above) showed only three causes of death (diabetes, appendicitis, digestive diseases) which did not differ significantly from the general group. The significance of these figures is at once apparent when it is considered that an estimated 110,000 workers in the United States have silicosis in some degree.

Development of the Disease. The time required for incapacitation due to silicosis has been the subject of a great deal of attention, but as yet no generally accepted conclusions have been reached. Apparently the period of exposure preceding contraction of the disease is a function of the dust concentration, nature of the dust, nature of the work and individual physical idiosyncrasies. From a study of 9,800 stone-cutters in the Ruhr it is concluded that 13 years exposure are required to show slight silicosis and 20 years to cause incapacitation. This same study reveals that 63% of the deaths among silicotics occur from tuberculosis complications. In porcelain workers silicosis develops slowly but progressively; the second stage is usually not reached until after 10 years. Among sandstone workers the minimal exposure has been found to be 11 years while for iron miners an exposure of 4 to 5 years may be responsible for the appearance of lung changes 4 to 8 years after the exposure has ceased. In contrast to the above relatively long incubation periods are those found among workers engaged in sand-blasting and in the manufacture of abrasive soaps; in these occupations only 3 or 4 years may precede the onset of the disease. Likewise, an examination of 175 men engaged in rock drilling showed that 50% were affected in less than one year.

There is a good reason for believing that under particularly heavy dust concentrations silicosis takes an acute form and may appear after an exposure of only a few months. Not soon forgotten will be the case of the Hawk's Nest Hydro Plant on the New River. Some 4000 workers, mostly negroes, were employed for wages of \$3.00 for a 13-hour day; in a relatively short time after completion of the tunnel work 475 of them were dead and an estimated 1500 others incurably ill. This case was investigated by a committee in the House of Representatives before whom witnesses testified that, "In the tunnel 10 drills were in operation at once and you could not see ten feet ahead of you even with the headlight of the donkey engine". Workmen were so thoroughly covered with a silica dust that "you could not tell a white man from a colored man, 15 feet away". It was this case that a senator from West Virginia described as "the most horrible industrial disaster in the history of the world". It was of this case that Representative Marcantonio of New York charged that 169 workers had been buried unidentified because the undertaker had "lost" his records. On the basis of all evidence which has yet been brought forward it would seem to the impartial investigator that the Hawk's Nest Tunnel should remain a tragic reminder to the engineer of the need for consider-

ation of human values in the solution of engineering problems.

The above case is only one of a number of similar but less sensational cases which have shown that there is a real possibility of contracting an acute form of silicosis if work is in an atmosphere having an extremely high concentration of siliceous dust. Experimentally the possibility of acute silicosis has been demonstrated by laboratory tests on animals; for exposures of eight hours a day to concentrations of 200 million particles per cubic foot or more it has been shown that the last stage of silicosis may be expected in a relatively short time.

Size of Hazardous Dust. The importance of particle size in determining the hazard associated with dusts has received a great deal of attention. Particles 25 to 30 microns in size will usually settle out rapidly and are therefore not likely to be breathed in quantities sufficient to constitute a hazard. However, particles of this magnitude (such as pollen of the Giant Ragweed or wood dust in carpenter shops) may be responsible for hay fever or for various types of asthma. In general, particles greater than 10 microns are caught in the trachea, large bronchi and mucous membrane. Particles around 5 microns go to the respiratory tract, but few of them reach the alveoli where the 1 micron particles are most numerous. Policard made a detailed examination of the lungs of two silicotic miners and found that 50% of the particles were less than .4 micron and 75% less than 1 micron; his findings should be used with care, however, because it is very probable that the dust distribution in the lungs is a function of the distribution of particle size in the inhaled dust—thus Policard's findings may be applicable only for the conditions under which the men whose lungs he examined had worked. The smaller particles are obviously the most dangerous since, on a weight basis, they offer a much larger surface than do the larger ones. For this reason dust counts are of more value in evaluating the hazard associated with a dusty environment than are gravimetric dust determinations.

The dust particles that are most effective in the causation of silicosis are of a magnitude so small that they are not perceptible to the human eye. A micron is 1/1000 of a millimeter or about 1/25,000 of an inch. As has been shown, the particles most important in silicosis are in the neighborhood of .5 micron or 1/50,000 of an inch—this represents a dimension of the same order of magnitude as the wave length of that form of radiant energy which we recognize as light. If one cubic inch of quartz were broken into cubes .5 micron on a side enough particles would be formed to create a silicosis hazard in a 250,000 cubic foot space.

The determination of mean dust sizes was the purpose of a series of comprehensive studies conducted by the U. S. Public Health Service: examination of 18,000 outdoor dust particles showed the median size to be .5 micron—nearly all were less

than 1.0 micron; in contrast, only 21% of 6,000 industrial dust particles were less than 1.0 micron, the majority being between 1 and 3 microns and the mean 1.5 microns.

Hatch and Moke investigated foundry dusts and found that composition is a function of particle size: the proportion of combustible matter and acid soluble iron, carbonates, etc., increase with decreasing size; the proportion of clay and other silicates varies without a definite trend with size; the quartz becomes less as the size decreases and for particles under 2 microns (which comprises 90% of the total number) it is from 1/5 to 1/25 of that found in the particles greater than 10 microns. These findings emphasize the necessity of taking dust samples directly from the contaminated air and not utilizing "rafter" or any other kind of settlement sample which does not contain a representative size distribution.

Hazardous Concentration. Permissible concentrations of dust vary with size, composition, and the dust retention characteristics of the individual. The latter factor is one which offers some hope from the standpoint of preventive medicine. Practically all that is known of the dust retaining characteristics of the human nose is attributable to the work of one man—G. Lehmann. This experimenter has shown that the dust fixation in the nose varies from 2% to 75% and is almost completely independent of such factors as the condition of the nasal secretion, the rate of air currents, the degree of concentration or chemical composition of the dust, the width or narrowness of the nasal passages; seemingly, fixation is a function of the vortex formation and rebound after passage through a restriction.

A correlation between fixation and silicosis would seem to follow from the following tests: Of 46 stone cutters with fixations above 40% only two were found to have silicosis; of 31 stone cutters with fixations less than 29% only 2 were healthy. After examining the above data Lehmann concludes that, "applicants for work with poor dust fixation capacity should not be allowed to work where a menace of silicosis exists".

As a result of 418 experiments with magnesium oxide and calcium carbonate, Carlton E. Brown gives the following relationships between dust retention and other factors:

Per Cent Retention is inversely proportional to

1. Respiration rate for rates below 20 per minute. An increase above 20 per minute is apparently followed by no changes in per cent retention.
2. Minute-volume of air breathed. This probably results from increase in respiration rate with minute-volume.

Per Cent Retention is unaffected by

1. Volume per respiration.
2. Vital capacity.
3. Relative humidity.

The same investigator found that the percentage of

magnesium oxide retained during normal breathing while at rest varied little from about 60% at a concentration of 10 mg. Above 10 mg. the retention changed but little, but as the concentration dropped below 10 mg. the retention rapidly approached 100%.

The relation between dust concentration and the incidence of silicosis has been studied by many investigators, but no general empirical relationship has as yet been established. One commonly used rule (proposed by Drinker) recommends the acceptance of 5 million particles per cubic foot as the minimal permissible dust concentration for pure silica and 50 million particles per cubic foot for dusts that are very low in silica. Some evidence suggests that this rule is unduly conservative for dusts which contain little silica and an alternative which is widely used is to limit the total dust count to a value such that the product of the dust concentration by the percentage of silica be less than 5,000,000; this rule gives 100,000,000 particles per cubic foot as the minimal concentration for non-siliceous dusts.

Physiological Effect. The lung condition known as silicosis results only from the inhalation of silica-bearing dusts. Why silica possesses the ability to bring about this condition has been the subject of much investigation and theories of silicosis are at present responsible for considerable controversy. Physiologically—excluding the effect of complications—silicosis affects the normal functioning of the body in the same way that the formation of scale affects the efficiency of a heat transfer device. In the heat exchanger the hot and cold fluids are separated by an exchange surface and the deposit of scale on this surface reduces its effectiveness as an exchange medium. If the scale becomes sufficiently heavy it will be impossible to secure the transfer of enough heat to accomplish the purpose for which the exchanger was installed; similarly, the lung is a surface through which oxygen is transferred from the air to the blood. The formation of scar tissue reduces the effective transfer area and eventually the lung is unable to accomplish its purpose—death from suffocation is the result.

Asbestosis

Asbestos dust is second only to free silica in the magnitude of health hazard which it represents. Asbestos is a fibrous material of varying composition, but is usually made up of approximately 43% magnesia, 40% silica and 14% water. Oxide of iron is frequently present and appears as dark particles among the translucent fibers—the fibers are of such length and fineness that cloth weighing less than 8 oz. per square yard has been made from them.

The lung condition resulting from the inhalation of this dust is known as asbestosis and resembles silicosis in its main clinical aspects, but differs due to the enhanced rate of development. Under average industrial conditions the length of employment in fatal cases is only about one-half that which is usual

for silicosis. It is estimated that there are in the United States approximately 10,000 men exposed to this hazard as a result of work in the insulating, asbestos cloth, and similar industries. As is true of the silicosis exposure estimates, this figure does not take into account the number of persons exposed as a result of employment in the process industries where asbestos finds use, under various trade names, as a filter aid. In one respect asbestosis is less deadly than silicosis—that is, there is seemingly a lower susceptibility to the supervention of pulmonary tuberculosis. The danger of contracting the disease is apparently not influenced by either age or sex. Like silicosis, it is incurable and progressive; a case is reported of a patient who was exposed to asbestos dust for one year and whose sputum showed the presence of asbestosis bodies fourteen years later.

Very few data are available on the relation of dust concentration to the incidence of the disease. No minimal safe concentrations have yet been set up and information is scant as to the conditions in those plants where a hazard is known to exist. It has been determined, however, that the asbestos dust encountered in fabricating plants is free of silica when air is floated at the breathing level and that the danger of the dust increases as the quality or length of the fibers increases.

Miscellaneous Siliceous Dusts

Many siliceous dusts other than quartz and asbestos have been carefully investigated. Talc—a silicate containing 62% silica—has been shown to be responsible for a form of pneumoconiosis when inhaled in large quantities. One group of investigators report that 50% of the talc workers whom they examined were affected and about 12% had tuberculosis. The same report shows that talc containing 10% tremolite is much more dangerous than talc containing 40% tremolite, but an explanation is lacking.

The action of cement on the lungs is not definitely known. German investigators say that it produces only an inert deposit in the lungs, but American reports show that the frequency of disability from respiratory diseases in cement plants is about double that found among employees in non-dusty manufacturing plants; absence from work was also twice as frequent. Yet animal experiments show that cement like limestone and gypsum produces an adsorptive reaction; the dust disappears from the peritoneal cavity without the production of scar tissue.

Slate (1/3% quartz) in concentrations of 700 million particles per cubic foot produces a fibrosis and pneumoconiosis, but few cases have been known to progress seriously. On the other hand, there is reason for believing that clay and slate dusts are responsible for a high incidence of tuberculosis.

Italian investigators have studied the dust hazard in cotton spinning mills and report that the silicosis hazard is apparently negligible. Mineralogic analy-

sis of such dusts brought to light the interesting fact that their composition is similar to that of the soil in which the cotton was grown.

Non-Siliceous Dusts

Apparently there is a great difference of opinion as to whether or not non-siliceous dusts are harmful either to normal or tubercular persons. The data are so scant that a review of the literature must consist essentially of a catalogue of the few and scattered investigations that have been made of particular dusts. General conclusions can not be drawn and an extension of available data to dusts of even the same mineral family is hazardous.

German investigators have studied a number of different carbon dusts and rate them in the order of hazard which they represent: lignite, coal, graphite, charcoal, soot.

A statistical study of the health hazard in a large American company which manufactures artificial abrasives led to the conclusion that persons who have had pulmonary tuberculosis should not be allowed to work where they will be exposed to dust from abrasives. The dust in this particular plant was aluminum oxide combined with silicon carbide and the incidence to tuberculosis was 1 1/2 times as great in the dusty departments as in the non-dusty. In this connection it is interesting to note that other investigators had previously reported aluminum oxide as not being a factor in the production of silicosis—thus a distinction must be drawn between the fibrosis-forming ability of a particular dust and its danger through increased susceptibility to pulmonary tuberculosis. This leads to the conclusion that the claim is unwarranted that a dust is not harmful merely because its reaction on lung tissue is inert.

The U. S. Public Health Service has grouped dusts (siliceous as well as non-siliceous) in three classifications:

1. Those causing an inert reaction, the amount of dust in the peritoneal cavity remaining approximately constant: soapstone, carborundum, jeweler's rouge, anthracite coal, bituminous coal and precipitator ash.

2. Those causing a proliferative reaction, nodules continuing to increase in size: quartz, chat and flint.

3. Those causing an absorptive reaction, dust disappears from the peritoneal cavity, but scar tissue is not formed: calcite limestone, precipitated calcium carbonate, gypsum and cement.

A similar list of dusts has been prepared by Haynes who carried on a long series of animal experiments. He arranges them in order of toxicity as follows: precipitated silica, flint, slate, hydroxide, precipitated chalk, magnesium carbonate, carborundum, calspar, emery, colloidal coal (in massive amounts).

All of the respiratory disorders discussed above, together with many others which are caused by the inhalation of fibrosis-forming dusts, are grouped under the one broad term Pneumoconiosis.

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