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THE JOURNAL OF INDUSTRIAL HYGIENE AND TOXICOLOGY

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# THE EFFECTS OF HEAT AND HUMIDITY UPON THE HUMAN BODY*

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CONSIDERATION of the effects of heat falls into two natural divisions:

1. *The acute effects of encountering very high temperatures such as are met in many industrial processes and under natural conditions in such places as the desert regions of southwestern United States.* The temperatures with which we are concerned in this division of our inquiry are necessarily high. Brief exposures to temperatures of 200°F. are encountered in the steel industry. One hundred and fifty degrees Fahrenheit is frequently found in the stokeholds of coal-fired steamers. Outdoor temperatures of 100° to 125°F. were met at Boulder City, Nevada, and under such exposure men worked through regular shifts during long periods of time.

2. *The effect of residence in tropical climates.* By this is meant the gradual deterioration experienced by most northern white people who reside in the tropics. A tropical climate of the sort that interests us is characterized by little change. There are no seasons such as we have in the temperate zone. Monthly temperatures and temperature differences between day and night are slight. In the Marshall Islands for example, islands lying in the equatorial belt, the daily maximum temperatures for a year were between 88° and 91.5°F. and the daily minima between 75° and 77°F. It has been said of such places that "they possess more climate and less weather", but in spite of such qualification they are not so suitable for human residence as are the changeable regions of the temperate zone.

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# THE CAUSATION OF PNEUMOCONIOSIS*

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THERE are four different types of reaction produced in man by the inhalation of dust. The first and most important are the pneumoconioses, such as silicosis and asbestosis, which cause specific lung pathology and often are followed by pulmonary tuberculosis. The second type of reaction is caused by toxic dusts like lead, cadmium, and radium. A third type of malady follows the inhalation of finely divided metallic fume particles such as zinc oxide and is known as metal fume fever. Finally, the fourth reaction, allergic in character, is caused by breathing organic dusts such as pollen and certain types of pulverized wood and flour. In all four instances dust inhalation can be the sole cause of the disability but with the toxic dusts characteristic reactions result from swallowing as well as from inhalation. The latter route, however, is much the more important.

In the present instance we are concerned only with the pneumoconioses which result solely from the action of inhaled dust upon the lung tissue. Of necessity then, the dust particles must be small enough to float about in the air and be carried by rather slight air currents; otherwise they cannot be inhaled.

The modern word, *pneumoconiosis*, is a shortening of Zenker's original *pneumonokoniosis*. Zenker (1) depicted lungs definitely damaged by dust particles, but today pneumoconiosis is generally used to describe any lung which has been dusted to more than the normal degree—there need not necessarily be demonstrable lung pathology. Silicosis, asbestosis, anthracosis, anthraco-silicosis, and similar terms indicate the different causative agents (silica, asbestos, and coal) in various kinds of pneumoconiosis.

The International Silicosis Conference in 1930 (2) defined silicosis as a "pathological condition of the lungs due to the inhalation of free silica ($SiO_2$)". The American Public Health Association (3) described it as a "disease due to breathing air containing silica." Sayers and Jones (4) state 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". Collis has claimed for years that free silica, especially quartz, was the all-important

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| COAL-MINING |

FIG. 1.—Mortality fr

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factor. In summarizing his South African experience, Watkins-Pitchford (5) wrote in 1927 that dusts other than silica could "give rise to such non-permanent and relatively harmless conditions—one can hardly call them diseases—as anthracosis, aluminosis, siderosis etc".

In figure 1 are shown data from Collis indicating the etiological importance of free silica. The U. S. Public Health Service's data (6), shown in table 1, emphasize still further the importance of free silica.

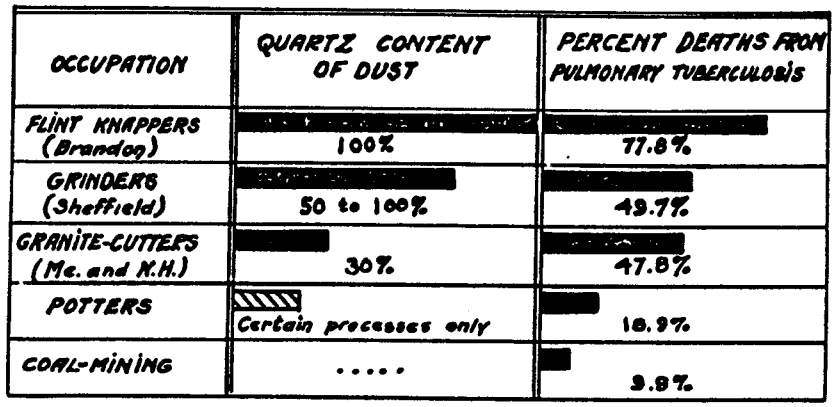


FIG. 1.—Mortality from pulmonary tuberculosis in various dusty trades. (After Collis)

*Silica.*—Silica occurs most commonly as the mineral, quartz, which is a natural contaminant of most ores, occurs in many rocks, and is found in a fairly pure state as beach sand, chert, flint, sandstone, gritstone, ganister, quartzite, and jasper.

Quartz is a hard crystalline mineral which is weakly birefringent, that is, its two indexes of refraction are near together. Chemically it is very inert and inactive. In fact its hardness and inertness are the two properties which make it especially useful in industry. Other varieties of pure silica such as opal, which is non-crystalline, have not as yet been appraised hygienically and there are no statistical studies to show the relative potency of the various forms of pure silica.

*Silicates.*—Silicatosis is a word suggested by Badham (7) to describe a lung fibrosis caused by dusts in which silicates and not free silica predominate. The distinction between free and combined silica is best shown by a simple example: A granite dust (8) might have the following mineral composition:

| | Per cent |
|---|---|
| Feldspar (orthoclase) | 70 |
| Quartz | 25 |
| Mica (muscovite) | 5 |
| | 100 |

Chemical analysis of this granite would give the following result:

| | Per cent |
|---|---|
| $SiO_2$ (total) | 72.55 |
| $Al_2O_3$ | 14.80 |
| $K_2O$ | 12.42 |
| $H_2O$ | 0.23 |
| | 100.00 |

TABLE I

SUMMARY OF THE SIX DUST STUDIES BY THE U. S. PUBLIC HEALTH SERVICE SHOWING THE DUST CONCENTRATION, COMPOSITION AND THE RESULTING HAZARD*

| INDUSTRY | AVERAGE DUST COUNT IN MILLIONS OF PARTICLES PER CUBIC FOOT | AVERAGE PERCENTAGE OF FREE SILICA (QUARTZ) | OTHER CHARACTERISTICS OF DUST | DEGREE OF HAZARD UNDER CONDITIONS AS OBSERVED IN EACH STUDY |
|---|---|---|---|---|
| Granite cutting: | | | | |
| Hand-pneumatic tool operators | 59 | 35 | Balance mostly combined silica | Great excess of pulmonary tuberculosis after 15 years or more exposure; silicosis in from 2 to 10 years. Silicosis after prolonged exposure; no excess of tuberculosis |
| Surface-machine operators, etc. | 36 | | | |
| General air | 20 | | | Negative except for occasional nondisabling silicosis |
| Less than general air | 9 | | | |
| Anthracite coal: | | | | |
| Rock drillers | 82 | 31 | Siliceous rock | Data insufficient; other studies show severe hazard |
| Miners and miners' helpers | 23 | 1 5 | Carbon and inorganic matter | Dyspnea and other signs of pneumoconiosis; excess sickness from respiratory conditions; excess mortality from influenza, pneumonia and possibly tuberculosis |
| Bituminous coal: | | | | |
| Rock drillers | 78 | 54 | Sandstone | Data insufficient; other studies indicate severe hazard |
| Loaders and machine men | 112 | 1 2 | Carbon | Generalized fibrosis chiefly linear in character; excess mortality from influenza and pneumonia |
| Cement | 26 | 6 8 | Primarily lime | Some early pneumoconiosis; excess of disease of upper respiratory tract and of influenza |
| Cotton-cloth manufacturing | 7 | ? | Vegetable and silica | Negative |
| Silverware manufacturing | 5 | 1 7 | Metal and other | Negative |
| Municipal | 4 | ? | Not determined | Negative |

*After Thompson, et al.



Recently Jones (9) ... cite was the outst... mineral constituent ... series of lungs he ex... is a variety of mica w... $K_2O \cdot 3Al_2O_3 \cdot 6SiO_2 \cdot 2H$... rence in nature is wid... quantities actually f... less than those of quar... been shown that s... quartz, will produce ... ogy; all investigations ... have been negative.

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Asbestos is not a ty... a name applied to a ... is easily separable ... flexible fibers. In ... commercial asbesto... riety of serpentine a... mineral chrysotile $3M... $4SiO_2 \cdot H_2O$, which, ... chemically, shale, kao... $2H_2O$, feldspar, and ... been studied in both ... laboratory. The pati... duce is much less sig... from quartz and the ... disabling.

*Carbon.*—An exce... of coal miners' lung... Cummins and Stadd... sult of which they ... is only retained in lar...

| | | | | |
|---|---|---|---|---|
| Loaders and machine men... | 112 | 1.2 | Carbon | Generalized fibrosis chiefly linear in character, [illegible] tality from influenza and pneumonia [illegible] |
| Cement..... | 26 | 6–8 | Primarily lime | Some early pneumoconiosis; excess of disease of upper respiratory tract and of influenza |
| Cotton-cloth manufacturing... | 7 | ? | Vegetable and silica | Negative |
| Silverware manufacturing..... | 5 | 1.7 | Metal and other | Negative |
| Municipal..... | 4 | ? | Not determined | Negative |

*After Thompson, et al.



Recently Jones (9) showed that sericite was the outstanding common mineral constituent of a considerable series of lungs he analyzed. Sericite is a variety of mica with the formula $K_2O \cdot 3Al_2O_3 \cdot 6SiO_2 \cdot 2H_2O$. Its occurrence in nature is widespread but the quantities actually found are much less than those of quartz. It has not been shown that sericite, without quartz, will produce silicotic pathology; all investigations along such lines have been negative.

Asbestos is the only silicate at present recognized as causing pathology which is definitely and distinctly different from that due to silica alone. The condition known as asbestosis, like silicosis, predisposes to tuberculosis but to a much less degree. Since, however, asbestos is handled by far fewer persons than are dusts containing free silica, asbestosis is much less common than silicosis.

Asbestos is not a true mineral but is a name applied to any mineral which is easily separable into more or less flexible fibers. In this country, the commonest asbestos is the fibrous variety of serpentine in the form of the mineral chrysotile, $3MgO \cdot 2SiO_2 \cdot 2H_2O$.

Other silicates such as talc, $3MgO \cdot 4SiO_2 \cdot H_2O$, which resembles asbestos chemically, shale, kaolin, $Al_2O_3 \cdot 2SiO_2 \cdot 2H_2O$, feldspar, and pure mica have been studied in both the field and the laboratory. The pathology they produce is much less significant than that from quartz and the fibrosis rarely is disabling.

*Carbon.*—An extensive examination of coal miners' lungs was made by Cummins and Sladden (10) as the result of which they wrote that "coal is only retained in large amounts when there is a really high silica content" and "we believe that in the absence of the silica factor there would be, under modern mining conditions, no serious degree of anthracosis. . . ." Men who were engaged in trimming coal ships with virtually no quartz exposure but undoubtedly with very heavy dust exposure, showed some fibrosis but it was not considered disabling (11).

The study made by the U. S. Public Health Service (12) in the anthracite mining district of Pennsylvania adds much weight to statements quoted from Cummins and Sladden, namely, that the harmfulness of a coal dust varies with the silica which contaminates the coal.

Haldane considered that coal dust might even reduce the severity of a quartz dust exposure; he suggested actually blowing coal dust into a mine with high quartz content as an antidote for the quartz dust (13) but no serious attempt apparently was ever made to test the validity of Haldane's claims. The recent statistical analysis of lungs autopsied in the Pittsburgh district (14) shows beyond doubt that city air, contaminated by an unusual amount of coal dust, does not produce a disabling fibrosis. Many of the lungs were markedly pigmented but the pathology found was not, in general, significant.

*Calcium and magnesium carbonates.*—These substances occur in nature as the minerals calcite, $CaCO_3$, magnesite, $MgCO_3$, and dolomite, $CaCO_3 \cdot MgCO_3$. Limestone and marble contain high percentages of calcite while the bulk of the rock from which cement is made consists of these carbonates. All three minerals are a great deal more soluble in water and in body fluids than

is quartz. Furthermore the solubility of all three substances increases greatly if the solvent is saturated with carbon dioxide, a condition which occurs in the lung fluid which wets inhaled dust.

The several studies which have been made on calcium carbonate dusts indicate that the dusts are not harmful probably because they are so readily dissolved (8).

*Gypsum.*—This mineral, $CaSO_4 \cdot 2H_2O$, is very common and is mined and milled all over the civilized world. It is an essential ingredient of ordinary plaster and is now used extensively as a wall board. When partially dehydrated it again takes up water readily, but in neither that state nor as the native substance has it been shown to be harmful (15). It is an interesting fact that pure limestone (quartz free), dolomite, and gypsum are recognized universally as the dusts safest (hygienically) to blow into soft coal mines to prevent explosions.

*Iron Oxides.*—The mining of iron, next to coal, is perhaps the mining industry in which the greatest number of men are employed throughout the world. The ore generally is hematite, $Fe_2O_3$, but other oxides, and sometimes the sulfide, pyrites, $FeS_2$, are handled on a considerable scale. As a result of the improvements made in machine-tool steel, it is possible today to see iron dust created by work on lathes, drills, and the like. Yet there are no data to indicate that iron, in the absence of silica, causes pathology in any way comparable to silicosis. However, lungs which have been heavily dusted with iron in any form are generally colored distinctively and were called by Zenker (1) *siderotic*.

## Estimation of Dust Exposure

The effect of any inhaled dust varies more or less directly with the duration of the exposure, the dust concentration and the volume of air breathed. In the case of the gas, carbon monoxide, our knowledge of the relationship of these factors is sufficiently exact to permit the use of a simple rule (16) for predicting the effects of breathing various gas concentrations under various conditions. Unfortunately, one cannot estimate or predict the severity of dust exposures with any such nicety.

In cases where men work for a number of years at different jobs with differing degrees of dustiness Bloomfield and DallaValle (17) compute exposures by averaging dustiness in these various jobs over the total period in question. In their anthracite coal dust study they found that computations so made agreed well with the results of the physical and x-ray examinations of the men. A typical example of their method of computing dust exposure is reproduced in table 2.

The advantage of these estimates lies in their simplicity and the fact that they have proven useful in correlating dustiness with physical examinations. The error in such calculations is that the effects of dust do not vary exactly, but only very approximately, with the dust concentration and with the exposure. Thus, Mavrogordato (18) writes that "Lesions of silicosis in a mild degree can be produced in an animal by 30 hr. exposure to intense dust clouds, and one is inclined to suspect that it is intermittent exposure to relatively dense clouds that is the deciding factor in producing the disease in susceptible human subjects." These momentary excesses of dusti-

### *Methods of Sampling*

Dustiness is given gravimetrically if the dust is to be determined chemically and by counts if the chemical determination is especially difficult. Thus, lead is always recorded in milligrams per cubic meter but dust which may cause silicosis is given in particles per cubic foot or per cubic centimeter.

Sometimes one may wish to convert one set of readings into the other. If the dust particles are perfectly uniform and of definite shape such as spheres or cubes and of known composition the conversions can be made with precision. In such a case, it does not matter how dustiness is recorded. But in practice the dust particles are never uniform in size and rarely in composition. Conversions are, therefore, apt to be misleading. For practical work, 1 mg. of fine quartz as collected by the impinger contains 300 million particles (by the light-field counting technic).

A great deal of energy is being wasted in deciding which dust sampling method is best. The rapidity with which new methods are appearing, each claiming unusual points of merit, indicates that standardization is unlikely. An international agreement on a reference standard would be welcomed by all working in this field at present, such an agreement seems to be far off.

In the United States the impinger technic is most used, largely because our only extensive field data come from the Public Health Service whose workers favor this instrument. If one wishes to compare his results with those of the Public Health Service he must copy their technic. But copying the Public Health Service's technic for the sake of the legal prestige thus gained has its drawbacks. We know of one case where a gasoline motor-driven generator and electric pump mounted on a mine car and hauled by tractor, with three men in attendance, resulted in obtaining only four samples in one day.

In South Africa where dust sampling has been done on a scale far beyond anything attempted elsewhere the konimeter and sugar tube both have been used. In England great hopes for the new thermal precipitator have been advanced but the results published so far have added nothing significant to the data already available from methods which are less accurate. In Australia several different instruments including Owens' counter have been used while the results coming from Germany were obtained by filter methods and by a modification of Owens' counter.

In our own studies we use several different methods according to the problem. There is every reason to encourage rapid methods which are particularly applicable to routine control and to discourage the widespread use of the impinger technic except for occasional check-ups. The impinger has little to recommend it in control work while a light portable instrument, such as the konimeter, has already proven its value in practice.

### Composition of Air-Floated Dusts

It is rarely that one meets exposures to pure silica; generally the dust is a mixture of which the original composition either is fixed, as in granite, or variable as in most foundry and mining operations. But the composition of



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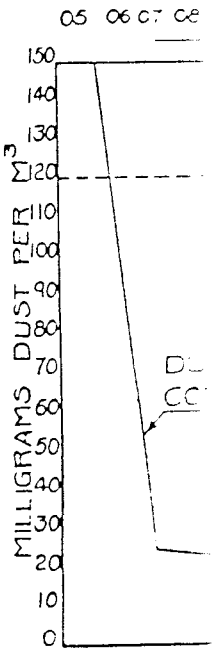


Fig. 2.—Dust ... Mavrogordato).

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Many examples ... composition of air-... found in the litera... statements but it ... the facts generally ... Thus, in this cou...

the dust breathed from a mixture is seldom the same as that of the parent material.

If a dust arises from the grinding or comminution of a comparatively pure substance, such as beach sand, the resulting material must have substantially the same chemical and mineralogical analysis in all stages of particle subdivision. But if a complex material like granite is ground and the dust then scattered in the air, samples of different particle size will have very different composition.

Many examples of the changes in composition of air-borne dust can be found in the literature to prove these statements but it is interesting that the facts generally have been ignored. Thus, in this country, investigators frequently sample and count air-borne dust by the impinger technic and then for chemical and mineralogical analyses take samples of material which has settled on rafters which will have a quite different composition. To make bad matters worse, samples for mineralogical and chemical analysis usually have not been graded into various sizes.

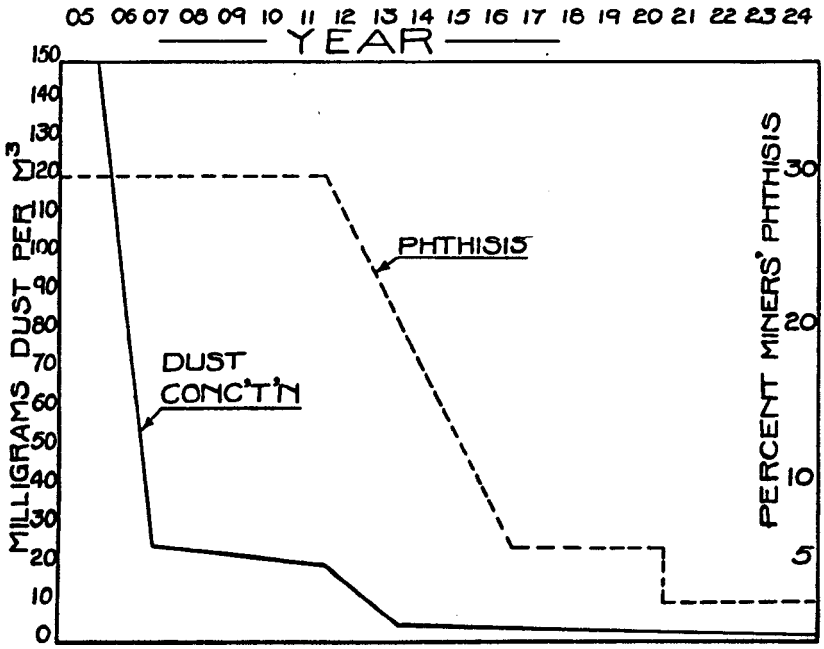


FIG. 2.—Dust control and silicosis in South African gold mines (adapted from Mavrogordato).

Jones (19) has given indisputable proof of the carelessness of which most of us have been guilty in recording the composition of air floated dusts. In support of Jones' contentions an interesting example is given by Drinker and Hatch (8) for foundry dusts. "A good molding sand is made up of about 80 per cent quartz in the shape of coarse particles; the bulk of the re-

maining material is clay in the form of fine particles which coat the coarse quartz grains. The chemical composition of the finer fraction (say below 10 microns) of material in foundry sand is markedly different from the composition of the coarse fraction." Thus, the original material contains about 76 per cent quartz, while particles above 10 microns have 85 per cent quartz and those below 10 microns have only 19 per cent (see table 3). Under the present methods of estimating dust composition and dust hazards a large error obviously would be made by assuming that the original material represented the fine air floated dust.

TABLE 3

Composition of Coarse and Fine Fractions of Unused Foundry Sand (Containing Natural Bond)

| Constituent | Percentage by weight | | |
|---|---|---|---|
| | Total sample | >10μ | <10μ |
| Combustible | 2.3 | 0.7 | 12.7 |
| HCl soluble | 5.5 | 1.6 | 30.5 |
| $H_2SiF_6$ soluble (clay) | 16.0 | 12.7 | 37.5 |
| Residue (quartz) | 76.3 | 85.0 | 19.2 |
| Total | 100.0 | 100.0 | 100.0 |

Another example is given by Jones (19) from the South African "banket" —large crystals of quartz held together by fibrous sericite. In figure 3 are illustrated the changes in dust composition which he noted as the dust was allowed to settle, as it would in practice. Obviously, the quartz content decreases as the sericite increases. Jones remarks that "when a wall built of quartz boulders is pulled down, the bulk of the dust comes not from the quartz boulders but from the mortar."

It would be interesting to know what changes in the composition of Barre granite, of 35 per cent original quartz composition, and of various coal-silica mixtures take place on settlement in air. Obviously, changes will occur (probably great changes) but they have not been measured as yet although we have all welcomed the figures of reasonable dustiness suggested by the U. S. Public Health Service for the two industries in question, granite cutting and coal mining. Their analyses were all made from samples which had settled out on rafters, and no samples were separated into various particle size fractions.

*Particle Size*

It is well known that in silicotic lungs dust particles under 3 microns vastly outnumber those which are larger. It has been alleged that the respiratory mechanism, the lungs, and the phagocytes are mostly responsible for this size grading. However, it is easy to show that the size grading is done in the air before the dust is breathed and not later in the human body. That is, we find an excess of small particles in the lungs simply because that is the way that they occur in air. It is physically impossible for any but particles below 5 microns



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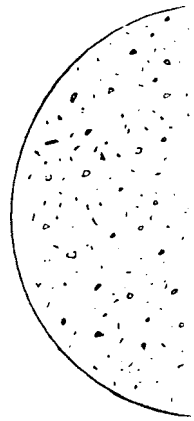

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to remain afloat in air long enough to be carried about by gentle air currents and to be inhaled. It is perfectly true that the alveoli are large enough to rarely remain in air more than momentarily. Bloomfield (21) has recorded hundreds of measurements of air floated dusts. His average sizes

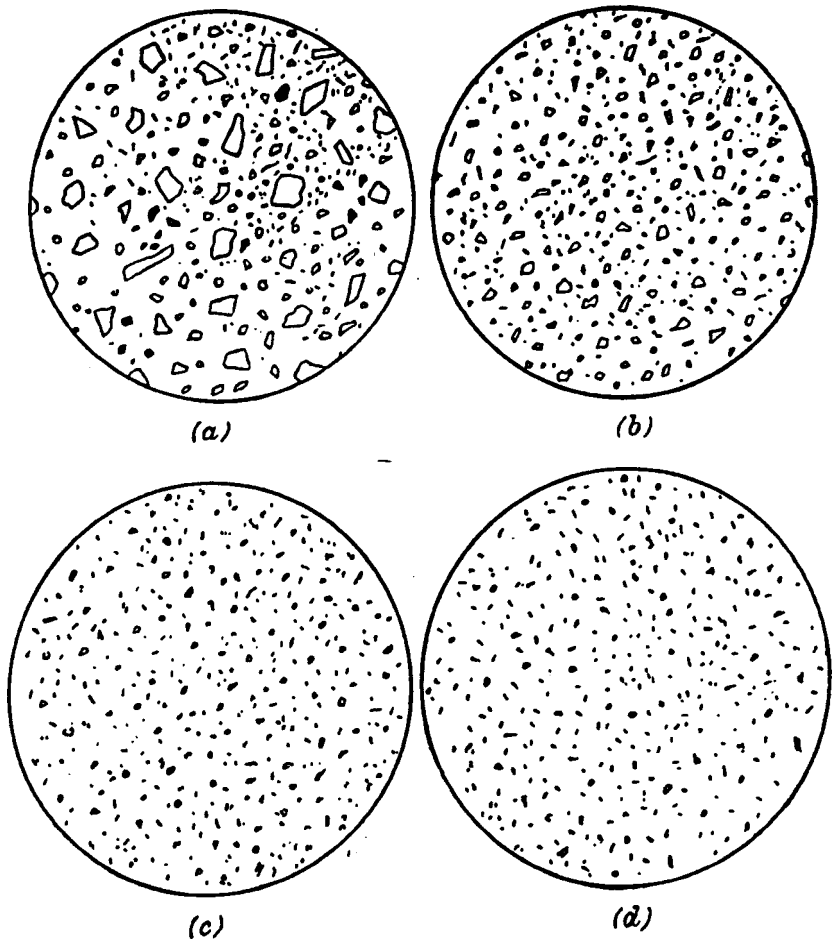


FIG. 3.—Sketches illustrating the increase in the ratio of fibers of sericite to quartz particles at different periods after blasting in a Witwatersrand gold mine. (*a*) about 15 minutes after blasting; (*b*) about 1 hour after blasting; (*c*) about 2½ hours after blasting; and (*d*) about 3 hours after blasting. (After Jones, courtesy Inst. Mining and Metallurgy.)

admit particles 100 or even 200 microns in length and that such particles are occasionally found in lungs (20). But the reason that they are found in lungs so infrequently is that they are all of the order of those found in lungs and in phagocytes.

Gye and Kettle (22) showed that colloidal silica was extremely toxic and that it could initiate fibrosis. It

is claimed occasionally that the damage done by quartz particles is due entirely to those which approach the colloidal in size. Usually particles possessing colloidal as distinguished from crystalloidal properties are considered to be less than 0.1 micron in size. All colloidal particles are so small that they can be measured only ultra-microscopically.

There is no evidence indicating that appreciable or significant quantities of ultra-microscopic quartz can be made by any known process of grinding or comminution, including blasting. In fact, the difficulty of preparing even small specimens of quartz below 0.5 microns is considerable. Furthermore, there are no published data indicating the relative potency of quartz particles in various sizes below 3 microns. Under the circumstances, then, there is no good reason to blame colloidal particles as the initiators of the silicotic nodule when it has been demonstrated again and again that such nodules can be produced by particles approximating the common bacteria in size.

### *Standards of Dustiness*

Under one name or another the plant or mine manager always wants an objective for his dust control program. It serves no useful purpose to evade the issue on the ground that precise figures are not available. Probably they never will be. The practical man argues, very properly, that if dust is the cause of silicosis there must be some degree of dustiness or of air cleanliness which is safe. Having been told that silicosis is a disease caused by breathing silica, the practical operating man naturally expects of the physician or hygienist some objective of air cleanliness, call it by whatever name one likes.

In this country the U. S. Public Health Service studies have furnished the only published data we have from which we may suggest dust standards. In the case of Barre granite it was pointed out that a dustiness of 10-20 million particles per cubic foot was reasonably certain not to cause disability. The coarse dust, in this case, contained about 35 per cent quartz. In their recent anthracite coal study the Public Health Service found that counts of 50 million per cubic foot, with 5 per cent quartz in the coarse dust, seemed safe.

In the case of pure quartz Cummings (4) suggests a figure of 5 million particles per cubic foot which is not far from the South African figure of 1 milligram per cubic meter (figure 3). We have then 50 million for dust with less than 5 per cent quartz and 5 million for pure quartz. It is very questionable if one has any right to interpolate for the quartz percentages between 50 and 5 but the figures certainly invite such interpolation.

These standards do not answer the question of the plant which handles dust of no proven pulmonary significance. What standard should the manager of such a plant take as his objective or need he take any precautions at all? We cannot give him any standards but we can only suggest that he investigate one of the many plants which has reduced dustiness without waiting for any physiological justification. Generally the manager and workmen of a clean plant will uphold eloquently the advantages of dust control.



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It is only in South Africa that dustiness and silicosis have been correlated routinely over a considerable period. There they realized at the outset that dustiness would not be controlled properly unless measured and recorded routinely. Concerning the results of their procedure, Irvine (23), Chairman of the Miners' Phthisis Medical Bureau in 1934, stated that "No 'New Rand Miner' who has entered the industry since August, 1923, i.e. 10½ years ago, has as yet contracted silicosis. These facts demonstrate that the engineering and medical measures which have been directed against silicosis have achieved a very significant degree of success." It would be hard to devise a more eloquent or complete proof of the advantage of dust sampling and of dust standards.

Silicosis and asbestos are definite diseases; in anthraco-silicosis and various silicatoses one sees modifications of normal lung conditions. The effects of carbon, or carbonates, of gypsum and of iron oxides are also discussed.

Methods of estimating dust exposures of a workman includes the taking of dust samples from the work place and interpreting the results during the man's total period of exposure. Errors which have been made from estimating the composition of dust from samples which have settled upon rafters instead of using samples taken from the air, are emphasized.

Permissible dust concentrations need not exceed 50 million particles per cubic foot for dust with very low quartz content or 5 million for pure quartz. In defense of the use of standards of dustiness the experience in South Africa where such standards have been applied successfully for many years is cited.

## Summary

The various pneumoconioses and their causative agents are discussed.

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# CLINICAL ASPECTS, DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS*

W. IRVING CLARK

*From the Norton Company, Worcester, and Harvard School of Public Health, Boston, Mass.*

THERE are two types of dust which may cause some degree of pneumoconiosis; these have been termed inert and active dusts. The determination as to whether or not a dust is active or inert has been reached by pathological examination of the lungs of those dying of pneumoconiosis, by clinical studies, especially x-ray, and by animal experimentation.

## REVIEW OF ANIMAL EXPERIMENTS

The study by animal experimentation has been particularly valuable as it provides a method of determining the effect of a given dust upon the living tissues and a comparison with the effect of similar exposure to other dusts. The animals used have been white rats, rabbits, guinea pigs, monkeys, fowl, cats, and, for some experiments, tadpoles and fish (1, p. 44).

The methods used have been:

1. *Dusting.*—This consists in exposing the experimental animal to a "high concentration of dust (up to 10 or 12 billion particles per cu. ft. of air, dark-field) to produce maximum effects in short time." This method might be called the normal breathing method.

2. *Injection of suspensions of dusts.*—This consists in injecting suspensions of dusts into various parts of animals to detect the capacities of different dusts to provoke reaction in the tissues. Results are obtained more rapidly than by dusting but do not correctly show the natural development of disease in the lungs.

The dust suspensions may be introduced into the animal by:

1. Intratracheal or bronchoscopic injections.
2. Intraperitoneal injections (Miller-Sayers method (2)).
3. Subcutaneous injections.
4. Intravenous injections—showing the effects of the dust on the extrapulmonary viscera.
5. Intracutaneous injections—for gross reaction.
6. Intra-lymphatic injections—for reaction of lymph node cells (3).

All animals tested either by dusting or by injection showed a tissue reaction to silica. This reaction was typified by the formation of fibrotic nodules except in the cold blooded animals where there was necrosis followed by some fibrosis. Changes in susceptibility of animals to tuberculosis as a result of the effects of various dusts have also been studied, and the

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When presented, the paper was illustrated by lantern slides showing x-rays and specimens of pathological conditions.

activating effect of silica demonstrated.

The effect of the inert dusts upon the animal tissue was found similar to that of any foreign body. There was reactionary enlargement of lymphatic nodes, slight fibrotic reaction immediately surrounding the injected dust but no continuation of this fibrosis and no formation of nodules. The exact pathology noted was scattered or clumped cells containing the inert dust lying in the alveoli, slight inflammation or no inflammation of the adjacent walls, a slow accumulation of dust-containing cells in the lymph nodes with enlargement of the nodes, and a deposition of dust about the lymphatics of the lung or pleura. When inert substances were injected intravenously, intraperitoneally or subcutaneously, there was no reaction beyond that of any foreign body (2).

As a result of such experiments which have been repeated in many parts of the world, it is believed that of all suspected dusts only silica has a specific action upon animal tissue which results in fibrotic nodulation. Asbestos fibres produce a peculiar reaction in the lung which is different from silica. Following the inhalation of asbestos dust there are formed "cuffs of more dense connective tissue about the terminal bronchioles" (1, p. 46). Contraction and collapse of the alveoli supplied by the affected bronchioles occurs. This is followed by induration and fibrosis of the collapsed alveoli, presenting the picture of diffuse fibrosis of parts of the affected lung with persistent foci of normal air spaces.

## Silicosis

The Committee on Pneumoconiosis and the Committee on Standards of the American Public Health Association (4) adopted in 1932 the following definition: "Silicosis 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, (some or all of which symptoms may be present) and by characteristic x-ray findings."

### *I. Pathology of Silicosis*

Dust particles after reaching the lung alveoli may remain quiescent for a considerable period as is the case with the inert dusts, or they may penetrate the lymph spaces in small numbers. The bulk, however, are phagocytosed, that is engulfed, by wandering endothelial cells which at first, lining the wall of the alveolus, become detached, and then, having taken up the dust particles, pass by ameboid movement through the walls of the alveolus into the lymph spaces. The cells now slowly migrate to the minute lymph islands which guard the entrance to the small lymphatic vessels, pass through these, thence to the lymph vessels, and finally are caught by the large group of lymphatic glands which form part of the hilus or root of the lung. These wandering cells are commonly called dust cells.

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lation of lymph so that the dust cells tend to move slowly toward the lung roots, blocking the lymph vessels and later the minute lymph masses which guard the entrance to the lymph vessels. The dust cells which are killed by the silica disintegrate and free the silica dust for further injury to the neighboring tissues. The reaction of the tissues to silica is the production of fibrous tissue. Thus fibrous tissue forms along the lymphatic vessels which accompany the blood vessels of the lung, invades the septa between the lobes, and spreads its shoots into the lung tissue itself. The minute masses of lymphatic tissue become fibrotic nodules and these, as time goes on, increase in number to such an extent that, combined with the fibrous tissue of the former lymph channels, conglomerate masses of fibrous tissue are formed, blocking off large portions of the lungs. The blocking of the lymph flow toward the hilus increases the spread of dust cells to the pleura and eventually leads to fibrosis of this structure.

The fibrotic nodule is characteristic of silicosis. It is a small, discrete hyaline mass surrounded by apparently normal lung. It does not exceed 6 mm. in diameter. "Occasionally some of these nodules may show microscopic foci of central necrosis" (1, p. 146).

This increasing amount of fibrous tissue obliterates the alveoli and provokes a compensatory enlargement of the alveoli elsewhere. This condition of enlargement is called emphysema. As the amount of air space is insufficient to permit the normal oxygen-carbon dioxide interchange of the lungs required by the body, the effort of the heart to pump blood fast enough may promote enlargement of that vital organ. This, however, if it occurs, is a terminal condition infrequently observed, as the patient usually develops pulmonary tuberculosis or dies of an intercurrent disease before this stage is reached. It must be remembered that only one-fourth of one lung is necessary for life, and that the amount of fibrosis must be enormous to restrict the lung capacity to this extent.

### *II. Symptoms*

The effect of the underlying pathology upon the workman is at first too slight to be detected by physical examination and the worker shows no symptoms. While there is a pathological condition present, it is to all intents and purposes harmless, in that if it does not progress rapidly it may not provoke symptoms during a worker's life, or only during the last decade. Even respiratory disease of another nature, such as bronchitis or even pneumonia, may occur with recovery during this period.

As the condition progresses, however, more and more of the lung tissue is converted into fibrous tissue, and the patient begins to show certain symptoms. He complains of shortness of breath when he goes up-hill and says that he notices his heart beat on exertion. He also mentions a cough which is at first dry and infrequent, but which gradually becomes moist and frequent. With the cough he raises scanty, stringy sputum which is sometimes discolored. The sputum is never large in amount unless he catches cold and develops bronchitis. At this

stage an attack of bronchitis does not disappear but becomes chronic. During this period the silicotic may complain of pain in the chest due to pleurisy or to epigastric pain, anorexia, and morning vomiting. While he is able to work, the victim cannot carry on his usual tasks and seeks lighter work.

Slowly the shortness of breath increases, the patient has poor lung expansion, and his color becomes pale and his lips bluish.

At this stage pulmonary tuberculosis is a frequent complication. When this occurs, the cough becomes more severe and continuous, the sputum free and moist, areas of dulness to percussion are noted, râles are heard over the chest on physical examination, and cavitation may be detected. Tubercle bacilli may or may not be present in the sputum. Many of these cases are able to carry on light work for years before finally succumbing. Hemorrhage from the lungs occasionally occurs.

During the early stages when perivascular, peribronchial, lymph node reaction is present there are only indefinite physical signs. Irvine (5) describes these as follows:

> 1. A certain lack of elasticity of the chest wall during movements of respiration together with
> 2. A somewhat reduced air entry, and
> 3. A characteristic alteration of the inspiratory murmur from the normal vesicular character to a higher pitched or "harshened," thinned and commonly somewhat shortened type, the expiratory murmur although somewhat prolonged remaining fainter than the inspiratory.

There is little change in these signs as the disease progresses. "The percussion note is somewhat flattened without being definitely dull especially posteriorly. Breath sounds have more definite characteristic thinning, the expiration being longer and fainter" (6). With the advent of tuberculosis the physical signs are those of that disease.

### *III. X-ray*

The most important evidence of silicosis is obtained by x-ray. While a flat film gives valuable information, an accurate diagnosis requires a stereoscopic set of films. In many cases a lateral film is desirable in order to determine the amount of emphysema present and the size of the heart. Fluoroscopic examination is of interest in cases where diaphragmatic adhesions are suspected or shown on the film.

The old classification into first, second and third stages has been recently changed to a more descriptive nomenclature. The conditions noted on the film are now called:

1. Stage of perivascular, peribronchial, lymph node proliferation. Irregular exaggeration of linear markings.
2. Stage of nodulation.
3. Stage of fibrosis with conglomerate masses.
4. Any of above stages complicated by shadows characteristic of tuberculosis.

The first is not considered as diagnostic of silicosis as it may occur in persons who are in good health or in a number of pathological conditions which have nothing to do with silicosis.

The stage of nodulation is pathognomonic of silicosis but may be confused with films of miliary tuberculosis.



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### *IV. Diagnosis*

Diagnosis is made upon:

1. Occupational history with estimated length of exposure, quality of dust and quantity of dust.
2. Physical examination.
3. X-ray examination.
4. Presence or absence of tuberculosis as a complication.

Of these measures the first and third are most important. Probably the most difficult thing to decide is whether or not tuberculosis is present. Absence of fever and maintenance of weight, with absence of physical signs suggesting active inflammation in the chest, is indicative of simple silicosis.

The absence of tubercle bacilli from the sputum is inconclusive, as many cases of silico-tuberculosis fail to show bacilli in the sputum.

### *V. Progress*

Silicosis once established in the lung has a strong tendency to progress. This appears to be owing to the toxic properties of the silica particle. What causes this toxic action is still in doubt. "Experiments and human pathological material indicate that in high concentrations silica is toxic and kills tissue; prolonged action of lower concentrations cause proliferation and fibrosis" (1, p. 46).

In spite of the fact that quartz is generally recognized as relatively insoluble, there is great rapidity of development of body reactions to silica. "Possibly unexplored properties of silica, electrical charge, adsorption etc., are concerned" (1, p. 48). This progressive tendency has been noted by Irvine, Böhme, Russell and others (1, p. 28).

The question is not what is the present condition of the silicotic, but how rapidly it will advance and how far. This is a serious problem in industry, for a worker having no symptoms and an early stage pathology by x-ray, may develop a disabling silicosis or a complicating tuberculosis. It is, therefore, possible for a worker to contract his disease while working for one employer and to develop symptoms many years later while working for another. If the work he is doing for the second employer involves exposure to an inert dust the employer may have difficulty in proving that this was not the cause of disability.

One of the striking differences between the effects of the inert dusts and silica dust is the lack of progress of pathology in the former as compared with the latter. Böhme (7) found that silicosis progressed after removal from exposure in 20 per cent of the cases diagnosed as having silicosis grade 1, in 40 per cent of the cases in grade 2 and in practically all the cases in grade 3. The experience of the State of Wisconsin, however, suggests that silicosis detected at an early stage in many cases will not progress if the worker is transferred to non-dusty work (8) though the reports of Watkins-Pitchford (9) and Britton and Head (10) make this somewhat doubtful.

Infection may play a part in the development. In fact, it is the frequent and serious complication of silicosis, and of the common infections, pulmonary tuberculosis is by far the most serious.

Tuberculosis may develop early or late in the disease. In those who have an inactive focus in the lung, the silica dust may change the condition to one of activity, or in cases of well-developed silicosis, tuberculosis may be superimposed upon the silicotic fibrosis.

Thus Kettle writes, "harmful dusts if inhaled into the lung may excite to activity a latent tuberculosis infection; they may exaggerate an active tuberculosis lesion or a coincident infection; and they may render the lung less able to cope with a superimposed infection" (11).

The course of silicosis with a secondary tuberculosis is usually slow and without the usual symptoms of intoxication and may be carried for years without serious impairment of working capacity.

While tuberculosis is the most frequent complication and cause of death, other infections do occur. On the Rand pneumonia is a common cause of disability and death. Pope and Zacks found pneumonia occurring with great frequency among foundry workers in Massachusetts. Proske and Sayers have confirmed Cummings' discovery of fuso-spirochetal organisms as a cause of infection among miners in Pieher, while chronic bronchitis with asthma is fairly common (12, p. 47).

Collis and Yule (13) compared the mortality experience of an occupational group exposed to silica dust with that of the general population, and with that of an occupational group exposed to dust not containing silica. As a result of the study, they concluded that silica is a body poison like lead. Even though it exerts its primary injurious effects on the respiratory tract, the silica slowly invades the body, involving the circulatory system, the nervous system, the digestive organs, the kidneys and liver, and eventually causes death through its harmful effect upon one or another of these organs.

## Asbestosis

"Asbestosis is a pneumoconiosis caused by the inhalation of asbestos dust. It is distinct from silicosis both in its pathology and clinically. Asbestos is a hydrated magnesium silicate containing no free silica but about 44 per cent of combined silica, 43 per cent magnesium, nearly 13 per cent of water and traces of iron and nickel" (14).

### *I. Pathology of Asbestosis*

Asbestos dust differs from other inhalable dusts in that it exists in thread-like fibres in which the diameter may be very small (5 microns or less) but which may be many microns in length.

These dust fibres do not appear to enter the alveoli. They are stopped at the neck of the alveolus where they are phagocytosed or penetrate the tissues. Acting specifically or merely as a foreign body they become surrounded first by mononuclear phagocytes, later by giant cells and last by fibrous tissue. There is no migration in dust cells to the lymphatics and lymph nodes as is seen in silicosis. The newly formed fibrous tissue contracts, constricting the neck of the alveolus so that no air can enter. Collapse of the alveolus follows and subsequent fibrosis.

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The asbestos fibre after it has become fixed at the neck of the alveolus frequently develops into a "curious body," the asbestosis body of Cooke (15). This is a long, narrow, cylindrical body, golden yellow and translucent, often with rounded ends like a dumbbell. These asbestosis bodies are found on autopsy and occasionally in the sputum. As similar bodies may occur in lungs not exposed to asbestos dust, the finding of these bodies is not diagnostic of asbestosis unless accompanied by a definite history of exposure to asbestos dust.

### *II. Symptoms of Asbestosis*

The symptoms of asbestosis, like those of silicosis, are cough and dyspnea. With the cough there is sputum which may or may not contain asbestosis bodies. Hemoptysis is rare. There is loss of weight in the advanced cases, and marked reduction in the vital capacity and in chest expansion. The patient's face has an "earthy" look.

In advanced cases the dyspnea is out of proportion to the physical signs, being very severe, and is accompanied by blueness of the lips and occasionally by clubbing of the fingers. The fingers frequently show "asbestos corns" from the penetration and irritation of fine spicules of asbestos which are detached from the asbestos in handling.

### *III. Physical Examination*

The patient, if an advanced case, shows loss of weight. The chest is emphysematous, and respiration is shallow, expansion being frequently less than 1 inch. The percussion note is one of dull tympany but with no definite dullness. The breath sounds are distant and expiration prolonged. The heart is normal in size, but lateral x-ray may show an anteroposterior enlargement.

### *IV. X-ray*

This shows a fine mottling of a "ground glass" quality over the lower half of the chest. There is often an obliteration of the costophrenic angle and pleural thickening, shown particularly in the interlobular pleural on the right. The lateral view may show well-marked compensatory emphysema. A spot of tuberculosis at either apex is occasional but rare.

Still later in the course of the disease the appearance is that of a very fine stippling that obliterates most of the natural markings. The pleural shadow is definitely thickened. In some of the advanced cases the heart is enlarged and radiating from it into the lung fields is a series of heavy fibrous bands. This picture has been referred to as "porcupine heart."

## Anthraco-Silicosis

This disease occurs among hard coal miners and is commonly known as miners' asthma. It is caused by the inhalation of large amounts of dust consisting of a mixture of anthracite coal and quartz, the quartz coming from the rock in which the coal is imbedded.

The pathology consists of a clogging of the lymph spaces with carbon particles which invade the upper lobes especially. Accompanying this is a linear or nodular fibrosis due to the

inhaled silica particles. When large areas of the lung are involved there is a compensatory emphysema.

*I. Symptoms of Anthraco-Silicosis*

The cardinal symptom of anthraco-silicosis is shortness of breath, which explains the term "miners' asthma." With this there is cough and sputum. The cough may be dry and the sputum scanty but if infection in the form of bronchitis is present, the sputum is muco-purulent and colored with coal dust. As in silicosis, tuberculosis is a frequent complication.

The physical signs are similar to those of simple silicosis or of silicosis with infection. Diagnosis is made largely by x-ray which gives a picture very similar to that of simple silicosis. The U. S. Public Health study on this problem (16) suggests that the true lung injury is due to the silica inhaled rather than to the carbon particles. Carbon has been found to be harmless when inhaled in the form of smoke and by animal experiment.

## Inert Dusts

*I. Pathology*

The inert dusts are relatively harmless. They may increase the frequency of respiratory disease when inhaled in large amounts, but this has not as yet been proved statistically. Workers inhaling these dusts develop a mild fibrosis which follows the course of the lymphatics along the broncho-arterial tree and is accompanied by enlargement of the trachial lymph nodes. This reaction which is very slow, after a number of years may progress to a moderate amount of interstitial thickening. The pleura may be thickened and there may be diaphragmatic adhesions. These later manifestations only appear after many years of constant exposure to a high concentration of dust.

*II. Symptoms Caused by Inert Dusts*

The symptoms of the pneumoconiosis of inert dusts are negligible. If the exposure has been long and the dust discharge abnormally heavy, there may be some dyspnea on moderate working. This is not usually severe enough to interfere with the worker's normal activities and may be caused by degenerative changes of the heart due to advancing age, as much as to the pathology of the lung. There is no evidence that the inert dusts, unless mixed with an active dust, can produce disability.

*III. X-ray Picture, Physical Examination and Diagnosis*

The x-ray picture is that of perivascular, peribronchial, lymph node thickening which does not progress. Some diaphragmatic adhesions may show in cases with long exposure to heavy concentrations of dust. Serial pictures fail to show nodulation or the massive areas of fibrosis which characterize silicosis.

The physical examination of those exposed to inert dusts is usually negative. In a few cases where there has been prolonged exposure to great quantities of dust there may be some restriction of chest expansion and signs of emphysema.

The diagnosis is made by physical examination, occupational history, and x-ray.

A typical inert dust is that of the artificial abrasive, aluminum oxide. This material is widely used in grind-



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ing wheels and for polishing, sand paper, etc. In the year 1929 over 71,000 tons were sold or used in plants in the United States and Canada (17).

The use of aluminum oxide creates a certain amount of dust. The effect of this dust has been studied by Clark (18) and Simmons clinically and by Gardner (19) on the experimental animal.

In a factory where artificial abrasives and grinding wheels are manufactured, Clark has studied the effects of inhalation of this substance for 25 years. He believes that in factories which provide proper dust removal, the continuous inhalation of artificial abrasive dust extending over many years of work does not produce the symptoms or present the x-ray findings of crippling fibrosis of the lung; that the number of cases of pulmonary tuberculosis does not greatly exceed the number normally present in the community; and that workers using wheels made of artificial abrasive or using this substance for polishing or other purposes run but slight risk of pneumoconiosis if excessive dust is removed by exhaust systems.

In spite of the fact that clinical and experimental evidence points to the relative harmlessness of the inert dusts, an effort should be made to keep the dust count around 20 million particles per cubic foot of air. This will make working conditions much more satisfactory to the worker and will reduce the hazard of respiratory disease.

## Treatment of Pneumoconiosis

The only method of treatment of the pneumoconioses is prevention. Dust in quantity must be eliminated from industry wherever possible, and in most operations this is feasible. In a few where it is not, protective devices such as respirators or positive air pressure helmets must be used by the worker.

When a worker is found upon examination to have lung fibrosis it is wise to keep him at work in a non-dusty department. While his lung condition, if it is due to silica or to asbestos dust, will progress slowly, many years of profitable work are before him unless infection intervenes. As dyspnea increases, lighter work must be provided. If tuberculosis complicates the picture and the patient develops tubercle bacilli in the sputum, he must be isolated from other workers, but even then may be able to do light outdoor work. In more severe cases where there is temperature, all work must, of course, be stopped.

### *Exposure*

The rapidity of development of silicosis and of asbestosis depends upon the amount of dust inhaled and the percentage of free silica ($SiO_2$) or of asbestos in the dust.

In men exposed to heavy dust clouds with a high concentration of silica, silicosis has developed in as little as 2 years, but under the usual exposures of mining and of industry where the free silica usually varies from 13 to 35 per cent, the development is slow and symptoms do not appear for 15 or more years.

In the case of asbestosis, the development of symptoms is more rapid, 5 to 8 years being the usual required time of exposure.

Efforts are now being made to correlate the exposure with the pathology, as shown by x-ray, and with the physi-

cal signs. In other words, an effort is being made to determine standards of permissible amounts of dust in the working air for both the inert and the harmful dusts. Such standards will be of the greatest value to industry in its effort to reduce its dust hazard to a minimum.

## Epidemiology

### *I. Silicosis*

The danger of inhaling inorganic dust has been recognized for centuries, but careful study of the effect of such inhalation was first instituted in South Africa and culminated in the International Conference on Silicosis held in Johannesberg in 1930.

In 1933 Van Sielen (20) published an estimate of the health hazard from dust in the mines and allied industries of the United States. In his study he found that of 7722 men examined in the Tri-State zinc-lead district of southwest Missouri in 1927–28, 5704 were classified as negative for lung disease, 1362 had signs of first stage silicosis, 253 had second stage, and 32 had third stage. The remainder showed signs of tuberculosis with or without silicosis.

In Butte mines (Montana), of 1018 miners 42.4 per cent showed definite signs of dust injury to the lungs.

In the Lead-Deadwood district mines in South Dakota the sickness rates per thousand for respiratory disease was two and a half times greater than in general industry, while the tuberculosis rate was almost ten times greater.

Grouping the various industries studied, Van Sielen found that in metal mining about 62,228 workers were exposed while among those engaged in non-metallic mining or quarrying, 23,665 had a respiratory hazard.

The prevalence of silicosis in the general population was studied by Lanza and Vane in 1934 (21). They cite the following occupations as constituting a definite silicosis hazard:

1. Anthracite-coal and metal mining, quarrying.
2. Certain manufacturing industries such as potteries, glass works, and plants manufacturing granite, sandblasting.
3. Construction work—rock drilling, handling sand and gravel.

Their rough estimate of the number of workers exposed to silica dust to a harmful degree in the United States is upward of 500,000.

A careful study of 2,600 granite and foundry workers has recently been made by Pope and Zacks (22) in which correlation of the duration of exposure to dust and the incidence of silicosis was determined. Their conclusions are as follows:

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

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

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

In 1907 Summons of the Miners' Phthisis Committee of Australia reported that gold miners there who contracted silicosis died of tuberculosis



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Russell found an excessive number of deaths from tuberculosis in his Barre, Vermont study of the health of granite workers (24) and Gardner (25) believes that at least 75 per cent of those who develop silicosis contract tuberculosis.

berculosis is so often associated with asbestosis that it seems probable that the association is more than accidental".

Gardner (1, p. 51) says that many autopsies show a combination of tuberculosis with asbestosis and other silicate dusts but "that surveys of living American workmen show no great excess of this infection", while Lanza (28) in a study of dust conditions in asbestos mines and mills in Canada and in fabricating plants along the Atlantic seaboard found in his study

TABLE 1

SILICOSIS AND ASBESTOSIS IN GREAT BRITAIN, THROUGH 1934*

| | NUMBER OF DEATHS | AVERAGE AGE AT DEATH | DURATION OF EMPLOYMENT IN YEARS | | |
|---|---|---|---|---|---|
| | | | Maximum | Minimum | Average |
| Silicosis | 261 | 55.4 | 60 | 2.3 | 34.8 |
| Silicosis with tuberculosis | 315 | 52.5 | 67 | 2.0 | 32.0 |
| Asbestosis | 41 | 41.0 | 27 | 1.5 | 12.9 |
| Asbestosis with tuberculosis | 26 | 38.0 | 29 | 0.8 | 9.9 |

* J. C. Bridge: Report of Chief Inspector of Factories and Workshops, London, 1934, chapter 3.

### *II. Incidence of Asbestosis*

The health hazard of asbestos dust has only been recently recognized. The Regulations for the Asbestos Industry in England have been in force for only 4 years. Bridge (26) says: "Over a number of years" (number not specified) "the deaths from asbestosis reported to the Department and compiled in 1934 numbered 41 while those from asbestos and tuberculosis numbered 26". (See Table 1.) Wood and Gloyne began their studies on pulmonary asbestosis in 1928 and published results of the study of 100 cases in 1934 (27). Most of these cases worked in the same factory. According to Wood, "pulmonary tuberculosis in 126 workers by x-ray that 67 were diagnosed as having asbestosis in some form, but that no predisposition to tuberculosis due to asbestos dust was indicated. Clark and Drinker (29) in summing up present beliefs say that while a secondary tubercular infection is not uncommon in asbestosis, it is far less frequent as a complication than in silicosis.

### SUMMARY

1. Pneumoconiosis is a disease resulting from the inhalation of inorganic dust.

2. The two pneumoconioses which produce disability are silicosis and asbestosis.

3. The pathology of silicosis is characterized by the presence of fibrotic nodules scattered through both lungs, and that of asbestosis is characterized by an interstitial fibrosis involving the lower half of both lungs.

4. The symptoms of silicosis and asbestosis are similar, the most important being dyspnea.

5. The most common complication is pulmonary tuberculosis which in silicosis is a frequent cause of death.

6. The diagnosis of both silicosis and asbestosis is made largely by a correlation of history and symptoms with the x-ray examination.

7. The treatment of pneumoconiosis is preventive and symptomatic.

8. The course of silicosis and of asbestosis is slow but eventually leads to incapacity due either to the disease itself or to a complication, frequently chronic pulmonary tuberculosis.

9. The number of workers exposed to harmful mineral dusts in mining and in industry in the United States has been estimated as 500,000.

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### THE VALUE OF DUST SAMPLES

The purpose of dust sampling is to make possible the control or elimination of dustiness rather than to obtain a precise measure of dust concentration. Conditions may vary greatly almost from moment to moment and only occasionally are they reasonably constant during a working shift. In general, then, it is advisable to give the results of the final estimate with an indication of the expected variation. "Thus, as a means of classifying processes according to their respective health hazards, it appears to be unnecessary to arrange them in concentration groups closer than 100 per cent, *i.e.*, 0 to 5, 5 to 10, 10 to 20, 20 to 40, etc." (8). Such an arrangement is consistent with the few American data we now have on standards of dustiness and is in reasonable agreement with Mavrogordato's "figures of merit" as obtained by konimeter samples.

A great deal of time can be saved by ignoring samples which are obviously too dusty—it is as well to take the sample for a matter of record but it is absurd to work long over it if it is certain to be vastly in excess of the objective. Mavrogordato dismisses such samples with the laconic symbols "T.M.C." (too many to count).

On the other hand, it is well to have records which indicate that the environment is as clean as is desired. In our own experience the ignoring of such samples has raised difficulties in proving in court that adequate dust control had been enforced. There is no doubt whatever that, in the United States at least, dust samples now have a very definite place in depicting working conditions to compensation boards or to a court. Under the circumstances, it is very unwise in making surveys to ignore the clean places.

TABLE 2

EXAMPLE OF METHOD USED IN DETERMINING AN EMPLOYEE'S TOTAL DUST EXPOSURE*

| OCCUPATION | YEARS IN OCCUPATION | AVERAGE DUST CONCENTRATION, MILLIONS PER CUBIC FOOT | MILLIONS OF PARTICLE YEARS PER CUBIC FOOT |
|---|---|---|---|
| Slate picker (dry breaker) | 2 | 380 | 760 |
| Patcher (dry mine) | 2 | 71 | 142 |
| Mule driver (dry mine) | 3 | 71 | 213 |
| Miner's laborer (chamber) | 3 | 480 | 1440 |
| Miner (chamber mining) | 3 | 480 | 7200 |
| Section foreman | 5 | 7 | 35 |
| Totals | 30 | | 9790 |

$$\frac{\text{9790 millions of particle years per cubic foot}}{\text{30 years}} = \text{326 millions of particles per cubic foot}$$

* After Bloomfield and DallaValle.

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