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October 3 to October 7, 1932

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Although the National Safety Council endeavors to eliminate from discussion at its conventions matters which are not pertinent to its purposes or which are contrary to its policies, the Council accepts no responsibility for the views expressed either in the papers presented or in the discussions thereon.

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NOTE: The sessions of the Street and Highway Traffic Section, the Child Education Section, and the Home Safety session are published in a separate small volume of these Transactions, available on request.

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a proper staff, and not by some enthusiastic person who has not been trained for the work. Why not use more registered nurses in the smaller plants? The registered nurse is much better qualified to interpret a physician's plan of medical service than any lay attendant.

Much depends upon the physical condition of the employees. I am of the opinion that the physician, by reason of his professional attainments, is best qualified to handle some of the problem cases among employees. Mental symptoms, brought about by worry over home affairs, quite often produce accidents. In many cases the fear and worry of sickness in the family can be greatly allayed by a talk with the doctor or the nurse. Many of these cases have come under my personal observation.

It should also be absolutely the part of the physician to say when a man shall return to his regular employment after injury, and this decision should not be influenced in the slightest by its possible affect on any "no-lost time accident" contest or record. These contests are proper and have brought about outstanding results in the reduction of lost-time frequency figures. However, there have been cases of injured employees being urged unduly to return to some form of work merely to prevent the marking up of a lost-time accident. Nothing should be allowed to interfere with the employee's welfare. The physician has every motive to return the employee to his regular work as promptly as possible, but his higher duty is to conserve both the physical and economic welfare of the employee as well as the employer.

The safety director has need of the medical service, and the two should earnestly co-operate and seek to bring about a mutual feeling among the employees. Both departments are striving by all possible means to prevent economic waste chargeable to the disabilities and hazards of industry.

TUESDAY AFTERNOON SESSION

October 4, 1932

The delegates first attended an informal luncheon, and then gathered for the afternoon meeting in a Joint Session with the Metals Section. This session was devoted to a symposium on the dust problem in industry. Chairman Greenburg immediately introduced the first speaker.

The Effects of Inhaled Mineral Dusts

By LEROY U. GARDNER

Director, Saranac Laboratory for the Study of Tuberculosis, Saranac Lake, N. Y.

As long as men have worked in stone it has been appreciated that an unusually large proportion of them suffer from disease of the lungs. It was natural to assume that such disease would be caused by the dust generated, and this belief was strengthened by the discovery of black, grey or red pigments in the lungs, sometimes accompanied by the formation of scar tissue.

The term, pneumonokoniosis was introduced to describe the lung pigmented in this manner. With further observation pathologists attempted to classify various forms of pneumonokoniosis on a basis of the type of dust inhaled and such names as anthracosis, siderosis, chacicosis and even byssinosis and tobaccosis made their appearance. It was recognized that some forms of the disease were attended by severe symptoms and result in death but there was no correlation between the clinical and pathological pictures and the causative dust. Finally, statistical study demonstrated that of all kinds of industrial dusts silica generally produced a definite type of disease with a characteristic pathological lesion and complex of symptoms.

Today the clinical entity known as silicosis is one form of pneumonokoniosis which is quite clearly defined. Moreover, within the last three or four years asbestos dust has also been shown to produce another specific type of reaction attended by symptoms differing in some respects from silicosis.

The other forms of pneumonokoniosis still lack complete definition. The widespread use of radiography has brought together a mass of descriptive data from individuals exposed to many kinds of occupational dusts but these findings have not yet been correlated with pathological anatomy, chemical studies of the tissues and detailed analyses of industrial atmospheres. There is a tendency to assume that many of these pulmonary changes are due to the action of silica perhaps modified by the chemical components of the dust. We shall only be in a position to speak with assurance about them when the pure types of dust and their various combinations have been studied in human beings or in experimental animals.

For the sake of clarity no mention has been made of the infections which so frequently complicate some forms of pneumonokoniosis. In the prebacteriological era of medicine the use of such terms as "grinders' consumption" and "miners' phthisis" reflects the popular association of pneumonokoniosis with tuberculosis. The names were justified both by clinical and pathological observation for it has been subsequently shown that, in silicosis at least, a super-imposed tuberculosis may cause death in perhaps 75 per cent of the cases. Some observers even go so far as to state that silicosis itself does not develop unless the lungs are previously damaged by a latent tuberculous infection. This is probably an exaggeration, for typical silicosis can be produced in the experimental animal by the inhalation of silica without infection.

The role of asbestos dust as an excitant of tuberculous infection is not as clear cut as that of pure silica. While there are numerous reports of death in asbestosis due to a terminal tuberculosis, nevertheless surveys of living workers have failed to demonstrate any excess of such infection. Simple anthracosis is said to prevent the progression of tuberculous infection. Statistics from coal mining districts do show a tuberculosis rate much lower than that "normal" for the age group. As yet corroborated experimental proof of protective action of coal against tuberculous infection is lacking.

Not only the tubercle bacillus but other bacteria seem to develop with special facility in the tissues previously damaged by silica. For example, the pneumonia rate among silicotic native laborers of South Africa is many times that in non-silicotic natives living under similar conditions. In coal miners, likewise, pneumonia is a common and often fatal complication.

What has been said of the effects of inhaled inorganic dusts would indicate that the reaction of the tissues is not merely a response to mechanical irritation by particulate matter. Today it is generally accepted that the injury is chemical in nature and that only certain of the common types of industrial dusts possess properties capable of exciting reaction. In the cases of silica and the silicate of magnesium or asbestos the slightly alkaline body fluids probably effect a slow solution of the dust particles liberating silica in colloidal form. This substance irritates the connective tissue cells which respond by multiplying. The result is an overgrowth of the supporting framework elements at the expense of the more delicate cells specialized for specific functions.

For these two types of dust the character and form of the pathological changes has been carefully worked out. In *silicosis* the essential tissue change consists of nodules of connective tissue which develop first in the lympoid tissues situated in the drainage apparatus of the lungs. These nodules interfere with the physiological removal of foreign bodies and subsequently inhaled particles accumulate in the framework of the lung itself. Such reaction gradually decreases the normal elasticity of the organ and encroaches upon its functional elements. As a result the cardinal symptom of the disease, dyspnoea or shortness of breath develops. Because of the excessive amounts of scar tissue formed in the lungs the right side of the heart

encounters increasing difficulty in forcing blood into the organs. In an attempt to compensate the overworked heart muscle becomes thicker and heavier and unless infection intervenes the time eventually arrives when the heart fails and death occurs from this cause.

The reasons for the prevalence of infection in the silicotic lung are not altogether clear. It is obvious that when the drainage system is so obstructed by nodules of scar tissue that more dust particles can only be removed with difficulty, the same must be true of bacteria which may be inhaled. As a consequence they remain in the lung and set up progressive infections. But this is not the whole story. The silicotic tissue apparently offers a peculiarly favorable soil for the continued multiplication of tubercle bacilli and perhaps other bacteria. Experiment has shown that even attenuated organisms of this type will produce rapidly fatal tuberculosis in a silicotic guinea pig. Furthermore, inhaled silica dust will cause a pre-existing latent tuberculous focus, harboring a few attenuated bacilli, to become progressive and spread.

The cause of this effect upon tuberculous infection has not yet been ascertained but probably it is in some way associated with the death of tissue caused by the toxic silica. Associated with this factor is an increased activity of the phagocytes which are stimulated by the irritating silica so that these cells tend to migrate rapidly and carry tubercle bacilli into previously uninvolved portions of the lung.

Asbestos dust, perhaps because of its fibrous structure, is not transported very far within the lung. It tends to lodge along the walls of the finer tubes and little of it is removed by the lymphatic drainage system. Tissue reaction, probably initiated by the solution of the fibers, occurs in their immediate vicinity. As in the case of silica this reaction consists of an overgrowth of the connective tissues which is later transformed into leather-like scars.

Aluminum oxide will serve admirably as an example of a non-siliceous dust whose particles are as hard and sharp as those of crystalline silica. When a measured quantity of such particles, 1 to 3 micra in diameter, are injected into the ear vein of a rabbit the majority of them come to rest in its liver and spleen. There they remain, apparently harmless, collected in large phagocytic cells and producing no reaction of the connective tissues for at least two years. Injection of the same quantity of the same sized quartz particles excites the formation of so much scar tissue that the functional elements are almost completely destroyed and the organs are reduced to nodular masses of leather-like consistence.

Carborundum, the carbide of silicon, when inhaled by guinea pigs for periods as long as four years, likewise fails to excite significant overgrowth of the connective tissues. It produces only a low grade inflammatory change with no nodules.

Instances such as those cited constitute the basis for believing that the cellular response to dust particles is not due to their hardness and sharpness but to chemical substances liberated by the action of the tissues upon them. The case for solubility has not been proven directly, for the detection of soluble silica in the tissues offers technical difficulties which today are unsurmountable. Nevertheless this substance is known to be a cell poison and in weak concentrations it will provoke an overgrowth of the connective tissues.

Mention should be made of the effect of inhaled coal dust. Most city dwellers breathe in sufficient quantities of this material during an average life-time to pigment considerable areas of their lungs. Deposits of grey or black dust will be found along the course of the lymphatic drainage system but this pigmentation is associated with little or no new growth of connective tissue. The coal miner's lung is much blacker but in most instances it also develops no deforming scars. When such reaction does occur analysis usually reveals appreciable amounts of silica in the lungs. The presence of excessive quantities of carbon dust apparently influences the localization of moderate amounts of silica so that the latter is not deposited in nodules as in pure silicosis but in streaks along lymph vessels as pure coal would localize.

Coal miners' fibrosis, due to relatively small amounts of silica, is therefore dif-

ferent from that of the pure quartz worker. When the amount of inhaled silica is excessive, the effect of the coal is negligible and the reaction approximates that in pure silicosis. There is said to be more reaction to hard coal than to the bituminous variety but there is evidence to suggest that this is due to the greater amount of siliceous rock which must be worked in mining anthracite coal.

Finally, consider the silicosis of granite cutters. This develops somewhat more slowly than that of the pure quartz miner and it has been maintained that this is due to the relatively small amount of uncombined silica in granite (25 to 40 per cent). But it is believed that other components of the glomerate granite may neutralize or at least inhibit the effects of the silica so that only after prolonged exposures to high concentrations does significant reaction occur. While typical silicotic nodules develop in guinea pigs inhaling pure quartz for a year, not even the earliest suggestion of nodules have appeared in the lungs of animals inhaling comparable concentrations of granite (40 per cent silica) for four years.

This brief discussion of some of the problems involved in pneumonokoniosis draws attention to the limitations of our present knowledge. It indicates that the reaction to an inhaled dust is determined by the chemical and probably physical composition of that dust. It emphasizes the need to further study of different types of dust both in the pure state and in measured combinations. The ultimate aim of such a study should be the neutralization of the toxic action of such dangerous substances as silica.

The Dust Content of the Atmosphere in Various Dusty Industries

By J. J. BLOOMFIELD

Sanitary Engineer, United States Public Health Service, Washington, D. C.

The speaker said in part: The properties of a given dust which determine its capacity to produce pulmonary pathology are, the nature of the dust, that is, its chemical and mineralogical composition, its particle size, and finally the quantity of the dust dispersed in the atmosphere.

One of the outstanding results of the last 20 years of research in the field of dust inhalation is the demonstration of the fact that, in general, the degree of health hazards associated with the inhalation of any dust, all other factors remaining constant, is dependent upon the mineralogical composition of the dust. For example, it is now well established that the inhalation of certain types of dust, such as granite dust, will in time produce fibrosis of the lungs, at times associated with tuberculosis. In other cases exposure to dust may result in the production of much less fibrosis without notable tendency toward subsequent tuberculosis; this is true of cement dust. And finally, there are certain types of dust, as typified by marble dust, which in the quantities and lengths of exposure so far observed produce little lung fibrosis. In general, it has been found that those dusts which are high in quartz content are the ones which produce a disabling fibrosis of the lungs most readily.

So far as the size of the dust particle is concerned, it is apparent that in order for any given dust to produce injury to the lungs, it must gain access to the parenchyma of the lung, the site where the harmful effects of the dust take place. It is known that not all of the particles of inhaled dust gain access or are retained by the human lung. In this connection it is of importance to have regard to the size of the dust particles present in the industrial atmosphere.

With reference to the quantity of dust present in the air of a workroom it is apparent that when the dust concentration is high the exposed person will inhale a greater quantity in a given period of time than he will when the dust concentration of the atmosphere is relatively low, and since the rate of production of the fibrosis

is partially dependent upon the rate in which the dust is inhaled, this latter item plays an important role in predicting the relative danger of different environments. Hence the need for the evaluation of the quantity of dust in the industrial atmosphere is obvious.

Research on the problem of industrial dust inhalation has indicated that so far as their fibrosis producing qualities are concerned, dusts may be divided into three groups: (1) those composed completely of combined silica, that is silicates, such as pure asbestos; (2) those containing free silica in the crystalline form known as quartz, (granite contains approximately 35 per cent of quartz); and (3) dust containing free silica in a non-crystalline form such as diatomaceous earth. In general, it has been found that the harmfulness of a quartz containing dust is in direct proportion to its quartz content. For this reason, in attempting to evaluate the harmfulness of a dust, it is of the utmost importance to ascertain its exact mineralogical composition.

It has been our experience that to determine accurately the exact mineralogical composition of a dust one should resort to a combined chemical and petrographic analysis. By no other method have we found it possible to determine the amount of quartz present in a given sample. In certain instances, such as when one is dealing with a mixture of quartz and pure potash feldspar, it is possible to determine the amount of quartz present in such dust merely by a chemical analysis. However, most dusts which come into question are mixtures of quartz and silicates. Take granite for example. The average granite is made up chiefly of three minerals in about the following proportions: feldspar 60 per cent, quartz 30 per cent, and mica 15 per cent. Chemical analysis shows that this average granite contains 70 per cent of silica. Of this 70 per cent, 30 per cent is present as quartz (free silica) and the other 40 per cent is present as combined silica, in chemical combination with the other minerals that make up granite; it is possible to determine these proportions only with the aid of the petrographic microscope.

We find in practice that samples of dust settled out of the asmosphere at the breathing level of the worker serve admirably for both chemical and mineralogical determinations. Table 1 presents the quartz content of dusts obtained in various industries which we have studied.

TABLE 1

Percentage of Quartz Present in Various Industrial Dusts

Kind of Dust	Percentage of Quartz
Rock drilling dust (bituminous coal mine)	54.0
Granite cutting dust	35.2
Rock drilling dust (anthracite coal mine)	31.0
Brass foundry dust	19.0
Dust from raw mills in cement plant	6.5
Slate mill dust (Vermont red slate)	3.0
Silverware polishing dust	1.7
Anthracite coal dust	1.5
Bituminous coal dust	1.2
Cement dust	1.0
Slate mill dust (Vermont green slate)	trace
Talc mill dust	none
Marble cutting dust	none

It is evident from this table that rock drilling occupations in the coal mining industry and certain occupations in the granite cutting industry and in brass foundries would be in the hazardous class as judged by the proportions of quartz in the atmospheric dust.

It has been demonstrated that particles of dust of a size greater than 10 to 12 microns in longest dimension are very seldom found in the lungs. This absence of larger particles is partly due to the fact that the numbers of such particles greater than ten microns in size present in industrial air is, as compared with the lower sizes, comparatively small; furthermore, these larger particles do not penetrate to the terminal portions of the respiratory tract. Hence we need only concern ourselves with those dust particles that are less than ten microns in longest dimension.

In order to ascertain whether or not an industrial dust is capable of gaining access to the lungs, it is necessary to know something of the size of the particles in the dust under consideration. In practice the samples for particle size studies may be obtained by the use of the Owens jet dust counter. The advantage of this instrument over other devices is that the Owens apparatus projects the atmospheric dust in unaltered condition directly on a microscope cover-slip. This cover-slip may then be properly mounted and examined by any one of several methods. In another method a microphotograph of the dust is made at a high magnification; the particles revealed on an enlarged print or screen may be measured by means of a millimeter scale. No matter which method one uses for particle-size measurements the results may be treated in the customary manner. The particles may be grouped in classes according to size, from which a percentage distribution curve is easily obtained.

As pointed out earlier, a knowledge of the quantity of dust dispersed in the atmosphere is very important, since with any given dust the rate of production of the injury will be dependent upon the total quantity inhaled.

From the practical hygienic viewpoint, the particle count is at present the best quantitative index of the degree of atmospheric pollution. The decision as to the size range of the particles which should be included in the dust count is a question requiring careful consideration. Obviously the size of the smallest visible particle will depend on the magnification and type of illumination used in the microscope, the refractive properties of the dust and to some extent on the visual acuity of the observer. We must bear in mind that our chief interest in this problem is in the industrial hygienic aspect. Primarily we are interested in differentiating between the dust content in the ordinary normal atmospheres, not yet known to be harmful, and certain industrial dusts which are known to be associated with lung damage. This difference is sharply marked so far as the dust particles between approximately one-half and ten microns in diameter are concerned; but the difference between such normal and abnormal air is masked and lost when we include in our determination the particles of ultra-microscopic size which are present in vast numbers in all air.

So far as the upper limit of particle size is concerned it has been demonstrated by the South African studies that particles greater than ten microns in longest dimension are, as a rule, of negligible importance. The data concerning the lower size limit of potentially hazardous dust is not so conclusive. Moir of South Africa, who examined microscopically 120 dust particles obtained from two specimens of silicotic lung, found that only 13 per cent of the particles were less than 0.5 microns and about 36 per cent of the particles were less than one micron in diameter. The majority of the particles, 60 per cent, were between one and three microns in size. The median size of the dust was found to be 1.2 microns in diameter. Drinker, in comparing the size frequency of the particles measured by Moir with the particles measured by him of the dust found in the sputum of men employed in ore mills, found a close correspondence. These findings have also been corroborated by Navrogordato.

In connection with the lower limit of particle size of dust of pathological significance the following pertinent question arises: Aside from the evidence direct or indirect of the non-retention of minute particles of dust by the lungs, what

TABLE 2
Average Dust Counts in Certain Dusty Trades

Industry and Occupation	Dust exposure in millions of particles per cubic foot	Average per cent of quartz in dust
Talc Mining and Milling:		None
Jack-hammer drillers	2,160	None
Packers	50	None
Muckers	45	None
Crushermen and cylindermen	14	None
Slate Finishing Mills:		3
Floormen	1,598	3
Loaders	1,276	3
Disc crusher operators	312	3
Quartz Grinding Plant:		99
Mill operators	173	99
Laborers	83	99
Packers	55	99
Granite Quarrying and Finishing:		35
Leyner drillers	144	35
Jack-hammer drillers	112	35
Hand pneumatic tool finishers	59	35
Machine pneumatic tool finishers	36	35
Plug drillers	37	35
Attendant labor (indoors)	17	35
Anthracite Coal Mining:		1.5
Miners and helpers	232	1.5
Attendant labor	31	1.5
Bituminous Coal Mining:		1.2
Coal cutters and loaders	112	1.2
Attendant labor	4	1.2
Marble Cutters	33	None
Cotton Cloth Manufacturing:		None
Carders	9	None
Weavers and spinners	5	None
Silverware Manufacturing:		1.7
Dusty trades	5	1.7
Non-dusty trades	2	1.7

evidence is there that appreciable percentages of ordinary industrial dusts ever fragment into those minute sizes less than 0.5 microns in diameter? The best answer to this question would be data of actual measurement of such dust. Unfortunately we have but scant published data on the particle size frequency of dusts in the air of industrial establishments. In 1929 Fehnel made some particle size measurements in connection with the dust study of hard rock drilling in New York City. He reported the findings on three samples, which showed the dust which was less than 1 micron in size to vary from 1 to 15 per cent. Most of the dust in these hard rock drilling operations was between 2 and 5 microns in size. Badham, in studying the dust hazard among sandstone workers in Sydney, Australia, measured some 16,000 particles of dust in the air of work places and found that 67 per cent of these particles were about 1 micron in size. In a particle size