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Are All Dusts Hazardous?

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DUST has always been considered an important industrial hazard, but within recent years European experts have believed it to be an outstanding industrial problem. Although we have not made as much progress in these matters in this country, most authorities agree that our problems are really becoming apparent.

This brief survey of the various phases of dust as an industrial problem will be confined to five different headings: (1) kinds of dusts; (2) what happens to dust when it is breathed; (3) silicosis; (4) methods of protection; and (5) legislation.

Kinds of Dusts

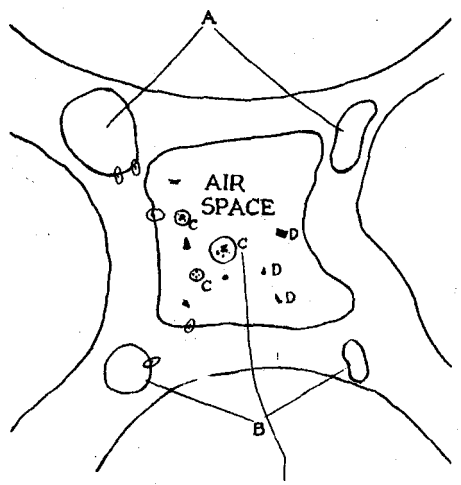
It is important for us to remember that there are harmless as well as harmful dusts. An explanation of the word "harmful" is in order; when we say that a dust is harmful, we mean that it produces a fibrosis in the lungs. By fibrosis of the lungs, we mean that an irritation is set up by the quality of the dust breathed in, this irritation producing fibrous tissue, or scars, which take the place of the normal lung structure.

Of course there are chemical dusts of various kinds, which if breathed into the lungs will produce systemic poisoning. There is no more rapid way to contract lead, mercury or arsenic poisoning, than by breathing in finely divided particles of these metals, because the poisonous material is immediately taken into the circulation through the very thin and microscopic partitions in the air sacs of the lungs.

In addition to poisonous chemical dusts there are the so-called non-poisonous organic or vegetable dusts, such as cotton, jute, hemp, also sugar and flour, and others which could be included under this category. Such dusts as these may cause functional disturbances in the lungs, but as far as we know, do not produce structural or organic changes. It is also well known that these dusts can be irritant to the skin and produce characteristic irritant eruptions in certain instances.

Dusts classed as "harmful" are those which are known to cause fibrosis of the lungs or systemic poisoning. Non-poisonous organic dusts are not considered causes of structural damage

Of the inorganic or mineral dusts we have three types, the non-silica-containing dusts, such as coal and lime; (of course there is a certain amount of silica in anthracite coal, but in most bituminous coals there is relatively a very small amount of free silica); the combined silicates, as the name signifies, are those compounds in which silica is in combination with other substances, typical examples of these being clay, talc, feldspar, and mica (up to about five years ago, most authorities agreed that no combined silicates produced fibrosis in the lungs; within recent years, however, there have been reports in the literature of cases of so-called asbestosis caused by the breathing of finely divided abestos dust which is chemically calcium-magnesium metasilicate. Also within very recent years, due to research of Dr. Leroy Gardner of Saranac Lake, it has been shown that carborundum or silicon carbide is also capable of producing a fibrous reaction when breathed in finely divided particles): finally, under the heading of inorganic or mineral dusts is the classification known as harmful or free silica, the best known types of this class being silicon dioxide or sand, granite, quartz, and the different varieties of sandstone.



Schematic diagram of lung cell showing engulfing cells migrating to blood and lymph vessels. "A", blood vessels; "B", lymph vessels. "D D D," dust particles free in air space.

We can, therefore, make the following outline of the various kinds of dusts:

I. Poisonous (chemically)
   Lead, mercury, arsenic, and others.
II. Non-poisonous
   A. Organic—cotton, jute, hemp, sugar, flour, and others.
   B. Inorganic—(mineral)
      1. Non-silica dusts, such as coal and lime.
      2. Combined silicates. The orthosilicates such as clay, the metasilicates such as talc, and the trisilicates such as feldspar.
      3. Harmful silica, existing in the forms of pure silicon dioxide or sand, granite, quartz, and the various kinds of sandstone.

There is still some difference of opinion as to whether the various combined silicates and harmful silica itself can be considered non-poisonous. When this outline was made some years ago, it was thought that silica exerted its harmful influence purely by mechanical irritation. With recent years there has been some scientific evidence pointing to the fact that silica may exert its harmful influence because of its chemical attributes.

What Happens to Inspired Dust

Here we are concerned only with particles which have a diameter of less than ten micromillimeters. Since a micromillimeter is equivalent to

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1/25000 of an inch, it will be seen that the particles which can cause trouble in the lungs are microscopic in size and cannot be counted except by microscopic methods.

It is now well for us to consider somewhat in detail the microscopic structure of the lungs showing just what happens to respired dust particles.

The sketch above shows a typical air sac which has been magnified approximately 1000 diameters. We are looking at a cross-section of the air sac showing the air space, and the walls of the air sac, within which are the lymph vessels and the blood vessels, "L.V." and "B.V." in the diagram. Note that there are some round cells which have gathered up particles of dust within them and also that there are free particles of dust in the air space. These round cells are called phagocytes, meaning that they are devouring cells. They actually surround foreign material and engulf various kinds of foreign particles which may enter the air sac. These cells then migrate through the wall of the air sac and through the wall of the blood vessels and the lymph vessels and are carried by the blood and lymph streams to various portions of the body.

Causes of Fibrous Reaction

We are particularly interested in what happens to the cells which enter the lymph stream, especially in the case of the lungs, because at various points along the course of these lymph vessels, there are interposed sets of lymph glands, which strain out the phagocytes and the foreign material. In doing this, they become enlarged, and in a great many instances these cells disintegrate leaving free silica in the glands, which then undergo fibrosis. This is what makes them visible in X-ray pictures, because of the increased density resulting from the formation of fibrous tissue.

A number of theories have been held as to the actual cause of the fibrous reaction in the lungs. An Italian named Mavrogordato first held that it was the quality of inspired dust and not its quantity which was the chief cause of a fibrous reaction. A little later an Englishmen named Collis proved by microscopic anatomical studies, that the devouring cells actually plugged up the lymphatic vessels and the glands at the roots of the lungs. So great is the stoppage of these vessels that they back clear up into the lung tissue proper, and there disintegrate leaving the free silica which because of its peculiar characteristics, is not absorbed like other dusts are. Being a constant source of irritation, a serum is poured out, which finally changes to fibrous tissue. This is a characteristic reaction which takes place in any part of the body where a foreign material is not absorbable and produces an outpouring of serum which is eventually changed to fibrous strands.

More recently, an Irishman by the name of Hefferman advanced the idea that the chief reason why silica produced a fibrous reaction was because it was a colloid in structure; you will remember from your chemistry that a colloid is a material which will not pass easily through an animal membrane or cell wall and is distinguished from the crystalloids which are soluble in body fluids and thus pass easily through membranes or cell walls and may thus be finally excreted by the fluids of the body.

Pneumonoconiosis is the scientific name applied to all types of dusted lungs, and it literally means a "dusted" lung. There are specific kinds of pneumonoconiosis, silicosis being a particular kind caused only by the breathing of free silica to the amount that fibrosis is produced. Abestosis is another form of pneumonoconiosis; anthracosis is a very common type, it being due to the inhalation of coal or carbon particles—most city dwellers have this type of pneumonoconiosis, which is not considered strictly speaking a real pathological change.

Silicosis

It is well to repeat that silicosis is caused by the breathing of harmful amounts of free silica, which eventually produces a fibrous change in the lungs, replacing the normal lung structures.

There are four important points in the production of silicosis:

1. Particle size.
2. Number of particles per cubic foot of air.
3. Mineral composition of the dust.
4. Length of exposure.

We have already commented on the particle size, stating that it is only particles of less than ten micromilimeters in diameter that can cause trouble, because only these can get into the lungs.

The number of particles per cubic foot and the mineral composition of inspired dust can be considered together. These two factors have a distinct relationship to each other and this relationship is indirect, meaning that a safe concentration of dust will reach a higher number of particles per cubic foot when the silica content of the dust is low; contrariwise when the silica content of the dust is high, a safe concentration of particles will be correspondingly lower. Let us illustrate this general statement: if the silica content of any given dust is 35 per cent, the standard of safety can be placed at approximately fifteen million particles per cubic foot of air in the breathing zone of the workman; if we reverse this situation and consider a dust which has a silica content of 70 per cent, then the margin of safety will be approximately seven and one-half million particles per cubic foot of air. When you reach 90 to 100 per cent of free silica, as in sandblasting, the standard is placed at approximately six million particles per cubic foot of air.

Length of Exposure

Our ideas concerning the length of exposure in the development of silicosis, have undergone a change within recent years. It was formerly thought that ten years was about the average exposure in the cases of silicosis which had developed. It is now known that a great number of cases of silicosis have developed in much less than ten years of exposure. For instance, in some of the Canadian silicosis laws, before a man can get compensation, it is required that he show a minimum of three years' exposure in stone-cutting occupations, and a minimum of five years' exposure in other occupations. This would tend to show that the average time necessary to the development of cases of silicosis is very much less than ten years. There are cases on record of where employees have developed silicosis within a period of two years, within a period of one and a half years, and in certain rare circumstances, even under an exposure of one year.

It must be remarked that not all persons are alike in the rapidity with which they develop silicosis under similar conditions of exposure. However, there is no immunity, natural or acquired, by which any individual may feel that he is entirely safe from

the contracting of silicosis, if exposed to conditions which ordinarily produce this disease.

Neither is there any known way by which a person may be freed of silicosis after he has developed it. One of the important points about silicosis is its progressiveness; after a victim has reached the third stage and in some instances the second stage, the disease goes on to a fatal termination, even if the employee has been removed from further exposure. This is called a "progressive" type of lung pathology.

Stages of Silicosis

Three stages are recognized: the so-called ante-primary, the primary, and the secondary. These names were given to the various stages through the British experience in South Africa, which has finally come to be a model for all other countries.

In the ante-primary stage there may be no other symptoms than those of a common cold. The physical signs may be quite indefinite and not characteristic. Usually, however, there is some diminution of chest expansion and some shortness of breath on exertion.

In the primary stage there is more diminution of chest expansion, more shortness of breath on exertion, and the chest findings are indicative of definite lung disease. The X-ray picture quite frequently shows an enlargement of the glands at the root of the lungs, with an increased amount of fibrosis, an extension of the lineal markings out beyond what is called the "periphery," and a beaded appearance of these bronchial markings caused by the laying down of fibrous tissue, and collections of devouring cells.

In the secondary stage, which is the final stage of silicosis, there is considerable disability due to the increased diminution of lung expansion and shortness of breath on exertion. There may be cough and some sputum. The X-ray findings are quite definite now and are characteristic, showing an infiltration over the entire middle portion of both sides of the lungs, with what is known as a coarse mottled or "snowstorm" appearance.

We have to keep in mind always that any case of silicosis may be complicated by tuberculosis, in fact in a certain number of instances, the terminal picture is one of silico-tuberculosis or pure tuberculosis.

Post-mortem findings have consistently shown that there are small nodules of fibrous tissue formed in advanced cases of silicosis. In such cases there are even particles of very fine sand present. This is not a new finding, it having been demonstrated by an Italian named Ramazzini, as far back as the year 1700.

Methods of Protection

In considering methods of protection it is wise to mention that the National Safety Council in conjunction with the U. S. Public Health Service has just completed a field study relating to the health hazards of sandblasting processes in industry. Briefly, surveys have been made of various types of sandblasting equipment, protective apparatus, and other relative factors. Dust counts have been made, showing the exact number of particles per cubic foot of air under various conditions. Petrographic analyses have been made showing the exact percentage of free silica under certain conditions, and medical histories, together with X-ray findings have also been developed. It is hoped that through this study, certain standards can be suggested for the safe conduct of sandblasting processes, which will form a definite step toward the protection of workmen in these occupations.

So far, the best method of protection as far as respiratory apparatus of each exposed employee is concerned, is the positive pressure hose mask. Together with this, it is often necessary to use properly equipped and maintained systems of exhaust ventilation.

In addition to these methods, special housing in the form of cabinets and barrels and sandblast rooms have also been found valuable. It is very important, however, that such enclosures should not only completely house all apparatus, but should be built in such a way that the dust concentration will be kept down to a point which is compatible with healthful working conditions.

In the experience of most companies, it is impossible to adequately protect such workmen without periodic medical supervision. This takes the form of periodic examinations, together with X-ray pictures, on employees who are already working, and also a very painstaking employment examination, and history, which will show the employee capable of working under such conditions.

Legislation

It is interesting to note that in three provinces of Canada, namely Ontario, Saskatchewan and Quebec, there are silicosis laws, which provide for the protection of workmen in industries where such processes are commonly used.

The Ontario law may be taken as an example. To carry out the various procedures necessary, a Silicosis Commission composed of three expert physicians has been appointed; it is the duty of this commission to make employment examinations on all persons entering industries where there is a silicosis hazard, to make periodic examinations on all persons so employed, and to rate persons who have contracted silicosis.

The Ontario law provides that there must be from three to five years' exposure, three years in stone-cutting trades and five years in other industries. In the ante-primary stage, showing the earliest detectible physical signs of silicosis, which have been or are present, a lump sum of $500 is awarded, whether or not the capacity for work is or has been impaired. In the primary stage where physical signs are more constant and radiographic changes are also more characteristic, there is an impairment which is usually not serious and permanent; for this stage a lump sum of $1000 is awarded. In the secondary and final stage of silicosis where the findings are rarely unmistakable, the impairment is considered serious and permanent and the employee is awarded two-thirds of his wages for the rest of his life.

Although the laws in Saskatchewan and Quebec are not exactly the same, they are very similar and in these other instances are administered in a similar way.

There are six states in the United States, which have complete occupational disease coverage, California, Connecticut, Maryland, Massachusetts, North Dakota, and Wisconsin. Hawaii is also included.

It is interesting that Illinois enumerates the dangerous processes for which compensation will be paid, if the claim is justified: these include processes involving lead, arsenic, brass, and zinc.