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Industrial Dust — The Pneumoconioses

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THE RELATION between dust and certain diseases of the lungs has been recognized for centuries, but only in recent years has it been given the intensive study it deserves. In the fifth century B.C., Hippocrates noted symptoms in a metal digger, comparable to those observed among present-day miners suffering from silicosis. Since that time, other investigators have recorded their impressions and observations on the effects of inhaled dusts and have emphasized the association of these with tuberculosis. It was not until the latter part of the nineteenth century, however, that real interest in the subject was manifested.

To South Africa must be given the credit for the earliest serious contribution to our knowledge of the industrial dust hazard. Here, in 1902, physical examinations were made on 3000 rock drillers and for the first time, x-rays of the chest were used in a comprehensive study of 300 cases. Diagnostic standards of disease, safe limits of particle concentration in the air of working places, and engineering methods for dust control were instituted. From 1915 on, Great Britain, Germany, Australia, Italy, Canada, and the United States made further investigations, but the most noteworthy progress in these countries occurred during the last few years.

Kronenberg and Morse[1] have suggested the following classification of industrial dusts:

TABLE I.

I. ORGANIC DUSTS:	II. INORGANIC DUSTS:
A. Non-Living:	A. Toxic and (or) Irritant.
1. Toxic and (or) Irritant.	B. Fibrosis Producing.
B. Living:	C. Non-Fibrosis Producing.
1. Bacteria	
2. Fungi.	

Generally speaking, organic dusts originate from plants or animals, but many thousands of these substances are made synthetically. They may produce toxic and systemic effects, but usually their main consequence is local or irritant. They do not cause pulmonary fibrosis, but such things as dermatitis, dental lesions, irritations of the nasal mucous membranes, and conjunctivitis are fairly common in workers handling these materials. Digestive disturbances have been observed. Allergic symptoms may occur in those exposed to pollens, horse hair, furs and wool, and various types of wood dust are known to affect susceptible persons. Bacteria such as the anthrax bacillus are capable of producing cutaneous lesions, or "wool sorters' disease," when inhaled. Sappington[2] has listed over 100 occupations which may produce exposure resulting in allergy, asthma, or irritation of the skin and upper air passages.

Fungi, such as mycelia and spores of molds, are apt to cause rashes and painful fissures of the skin, while cotton weavers develop a form of disease known as aspergillosis from inhaling spores of a mildew which occasionally occurs on threads.

The inorganic dusts which have significance in industry are derived mainly from minerals and metals. In the process of grinding, crushing, blasting and drilling these earthy substances, dust particles ranging in size from microscopic to visible are liberated and remain in suspension in the air for varying periods depending on their size and settling velocity. Inhalation of those particles which contain silica in the uncombined state produces the type of pulmonary fibrosis known as silicosis. Gardner[3] demonstrated the specific action of silica and made it clear that only silica in the "free" state is capable of causing this type of tissue reaction.

The toxic inorganic dusts are those of the heavy metals and their salts, such as lead, mercury, manganese, etc. They are usually considered under the heading of industrial poisons rather than of dusts.

The text of DR. HAMLIN'S Lecture at the Second Post-Graduate Course in Industrial Medicine, Long Island College of Medicine, Brooklyn, Friday, November 5, 1948.

IN DISCUSSING hazardous particles, the distinction between dust, fumes, smoke and mist is frequently overlooked. Drinker and Hatch[4] state that dust is formed by reducing earthy materials to small-sized portions, sub-microscopic to the visible, the composition of the particles being the same as that of the parent material. Common examples are the mineral dusts derived from the disintegration of rock and the organic dusts like wheat and flour.

Fumes are formed by processes like combustion, sublimation and condensation. The particle size is generally below 1 micron.

Smoke is generally of organic origin and is characterized by a particle size below 0.5 microns.

Mists or fogs are formed by the condensation of water vapor upon suitable nuclei. The particle or droplet size varies widely, depending on the condition prevailing.

The significance of these distinctions becomes apparent when evaluating the exposure in an individual manifesting signs or symptoms of pneumoconiosis. Since men working in dusty atmospheres are frequently exposed to a combination of these factors, the actual cause of fibrosis may be obscured, a fact which has considerable importance in cases involving litigation.

Like "rheumatism," the term "pneumoconiosis" covers a variety of conditions. It has been defined as a "chronic pulmonary fibrosis due to the inhalation of irritating dusts which produce a proliferative reaction" (Johnson[5]), or a condition due to "the effects upon the lungs of the inhalation of excessive quantities of dust, manifested by structural changes in the lung tissue and entirely distinct from the action of poisonous dust such as lead or mercury, in which case the lungs act merely as the point of entrance into the body without definite local influence" (Pancoast[6]). Perhaps the simplest way of stating it would be to say that the term refers to a condition of the lungs resulting from the prolonged inhalation of dust whether harmful or inert.

Pneumoconiosis includes such specific diseases as anthracosis, asbestosis, siderosis, silicosis, etc.

IN RECENT years few diseases have received more attention and publicity than those due to dust. The work of the United States Bureau of Mines in the Tri-State Lead and Zinc Mining districts (1924-1927) and the Metropolitan Life Insurance Company[7] directed attention to other industries where disabling pulmonary diseases were known to exist. Other investigations were made by the U. S. Public Health Service, and in 1933 Gardner and Cummings, of the Saranac Laboratory, began extensive studies in the iron ranges of Northern Michigan and Wisconsin. Since that time, many industries, here and abroad, have established clinics and laboratories for further research and control of the hazard.

The pathology of these diseases was definitely established by Dr. Leroy U. Gardner,[8] Director of the Saranac Laboratory, in 1934. He demonstrated the effects of various dusts on the lungs of laboratory animals and studied the part played by tuberculosis in the progress of the disease. The etiology, physical signs and symptoms, x-ray and laboratory findings were fully described and many other details of a technical nature determined. During this period also, concentration codes, representing "safe" limits of air borne dust, were recommended. These vary in different locations but, generally speaking, the following table, set up by the Committee on "Prevention of Silicosis Through Medical Control" of the National Silicosis Conference, offers a fair standard.

TABLE II.

PERMISSIBLE DUST CONCENTRATIONS IN VARIOUS INDUSTRIES

Industry	Percentage Silica in the Dust	Permissible Dust Concentration millions per Cubic Foot
South Africa*	80	4½
Ontario Gold Mines*	about 85 (in the rock)	8½
Australia Sandstone*	90 (in the rock)	6
Barre Granite*	81 to 88	00 to 20
Pennsylvania Anthracite Coal Mines**	85	5 to 10
	13	10 to 15
	5	50
Broken Hill, Australia*	10 to 17	14

*Based upon engineering practice.
**Based upon clinical studies.

Of the dusts studied up to the present time, only silica and asbestos produce definite pulmonary fibrosis. All the other types of pneumoconiosis exhibit the same general kind of tissue change with a similar pattern of shadows on the roentgenogram. Gardner states that the pattern in this instance consists of a mere accentuation of the normal branching, tree-like shadows cast chiefly by the pulmonary blood vessels. It represents a simple benign type of linear fibrosis and is difficult to distinguish from the mild accentuation of linear markings sometimes seen in x-rays of individuals with no known history of dust exposure.

Various terms are used to indicate special types of pneumoconiosis. For instance, "anthracosis" designates a condition of the lungs found among coal miners due to the inhalation of coal dust. "Siderosis" describes the tissue reaction occurring in some iron ore miners, and such terms as "byssinosis," and "tobacosis" refer to the pulmonary changes resulting from exposure to dust from cotton and tobacco. While such distinctions do not add much to our knowledge of diseases due to dust, nevertheless it is desirable to have a general conception of the important physical and roentgenological features of each for the sake of diagnosis.

In this connection it is advisable to mention the status of the silicates. The term "silicatosis" has been used from time to time to describe changes observed in the chest x-ray of persons exposed to dust from such substances as talc, soap stone, mica, feldspar, garnet, etc.; but the exact role played by these silicates in the production of pulmonary change has not yet been definitely established. They constitute a group of numerous minerals which find widespread use in industry, but with the exception of asbestos, their capacity to produce fibrosis has not been demonstrated. Petrologists warn against the potential hazard from silicates but experiments so far have produced no evidence of connective tissue proliferation as a result of their use. On the other hand, it should not be assumed that lack of such evidence indicates absolute inertness. The changes observed in the chest x-rays of persons exposed to silicate dust have been theoretically explained as a mild silicosis arising from silica left after the body fluids have leached the bases out of the silicotic molecule. A representative group of 24 silicates used in industry, listed by Gardner, appears in Lanza's book, "Silicosis and Asbestosis." The inert dusts, which include most of the silicates, are relatively unimportant because of their non-disabling character. A few of these materials are limestone, marble, talc, chalk, calcined magnesium for insulation, furnace linings, carbon dust, iron dust, tobacco dust, cement, cotton, molds, fungi, etc.

Anthraco-silicosis results from excessive exposure to coal dust which contains amounts of free silica. Since carbon is one of the inert dusts, it seems reasonable

to assume that the fibrosis is simply the result of the action of free silica which is present in sufficient concentration to induce proliferative cell reaction. Because it occurs chiefly in hard coal miners, it has been known for many years as "miner's asthma."

The cardinal symptom is shortness of breath, frequently associated with productive cough. More advanced cases complain of weakness, chest pain, gastric disturbances and hemoptysis. Sayers[9] states that in a U. S. Public Health Survey of anthracite miners in Pennsylvania in 1933, fever and night sweats were seldom mentioned. (This is true of silicosis cases in iron ore miners.) He notes such physical signs as dyspnea, prolonged expiration, change in contour of the chest, decreased chest expansion, clubbing of the fingers, change in breath sounds, altered fremitus and impaired resonance. Where infection complicated the picture, the symptoms were more marked and included cyanosis and loss of weight and strength.

The pathology of anthraco-silicosis is characterized by accumulations of coal dust in the lungs associated with varying degrees of pulmonary fibrosis. The lungs are gray and firm and may show dark colored markings on the pleural surfaces. On section, areas of black pigmentation appear scattered throughout the lung fields. Coalescence of the nodules produces larger areas, particularly in the hilar regions, but many large discrete nodules appear throughout the parenchyma.

Fibrous hyperplasia can be seen along the lymphatics. Microscopic section reveals deeply pigmented areas of fibrous connective tissue with dust laden macrophages around the outer border of the nodule.

The earliest x-ray evidence of anthraco-silicosis consists of exaggeration of the linear markings which increases with continued exposure, until the general pattern is obscured and replaced by definite nodulation. Further progress of the disease will be evidenced by more massive conglomerate shadows which may be complicated by the presence of infection.

Asbestosis as an occupational disease has been recognized only in recent years. Forty-one deaths were reported from this cause in England up to 1934. In this country it has been estimated by Lanza that approximately 10,000 persons are exposed to asbestos dust. In a study by Dressen, Edwards and Miller[10] of the U. S. Public Health Service, 541 asbestos workers were examined. No cases of asbestosis were found among workers exposed to dust concentrations below 2,500,000 particles per cubic foot of air.

THE greatest occupational hazard exists in mining, handling and crushing crude asbestos, making insulation and the carding and weaving of asbestos. In other industries such as the compounding of materials for automobile brake linings, the hazard is recognized but the disease is uncommon.

Asbestos is a hydrated magnesium silicate. It is the one silicate which does produce a fibrosis, but this differs pathologically and roentgenologically from the nodular reaction of silica. Industrial asbestos dust is comprised of very small fibres. Only the larger ones, over 2 microns in length, are thought to be capable of producing fibrosis. Gardner[11] is of the opinion that inhaled asbestos fibres are irritating not because they are silicates, but because they are stiff fibres which mechanically irritate the lungs. When the fibres used in animal experiments were finely ground (under 2 microns), the irritating property of asbestos practically disappeared. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body other than the lungs. While the action of free silica is chemical, that of asbestos is mechanical. The typical lesions, observed microscopically, show large macrophages in the distal portions of the bronchial tree, the formation of the asbestos giant cell, and the generalized fibrosis surrounding the bronchioles, alveoli, air sacs and blood vessels. This results in obliteration of the lung structure. The alveoli have literally become plugged, and the function of the lung impaired mechanically.

Merewether and Price[12] examined 363 workers exposed to practically pure asbestos dust in factories in Great Britain. The following table indicates their findings as regards exposure and fibrosis.

TABLE III.

Years at Work	Cases Examined	Showing Fibrosis	Per Cent
0 to 4	89	0	0.0
5 to 9	141	36	25.5
10 to 14	84	27	32.1
15 to 19	28	15	53.6
20 and over	21	17	90.9

As in cases of fibrosis produced by other dusts, there is no typical symptom of asbestosis. Dyspnea is the most striking feature of the disease. The onset is gradual, and the symptoms increase as the condition advances. Cough, expectoration, cyanosis, loss of weight, and emaciation are late occurrences probably associated with infection.

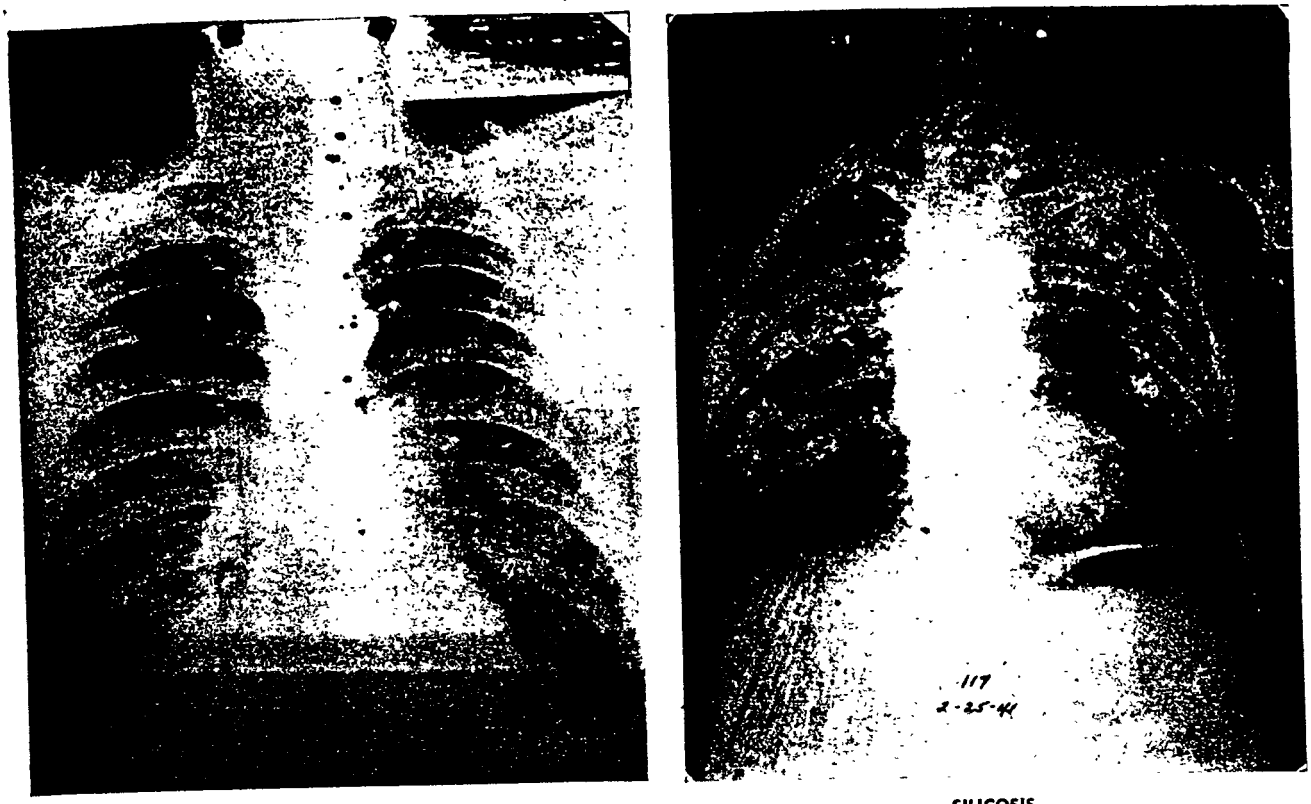
A feature of asbestosis is the occurrence of so-called asbestos bodies. Cook[13] states that "the 'curious bodies' so characteristic of pulmonary asbestosis are found in the alveoli and bronchioles and in the fibrous and necrotic areas. They measure 20 to 100 microns in length. One or both ends are bulbous, giving a clubbed or dumb-bell appearance. The shafts are either homogeneous or segmented crosswise. They are golden yellow to brownish in color. They do not stain but give a Prussian blue reaction to iron. From a diagnostic standpoint, it is generally agreed that the 'curious bodies' signify exposure to asbestos dust but cannot be depended upon for a diagnosis of asbestosis." Apparently the asbestos body is formed from the original fibre by a tissue reaction, the nature of which is still obscure. The fact that segmented figures are not found in crude asbestos or asbestos dust indicates that they are formed only after the fibres have come in contact with living tissue.

Another interesting skin lesion found in this disease is the "asbestos corn" due to the penetration of fibres into the superficial layers of the skin.

To establish a diagnosis, a history of exposure to asbestos dust is essential. The length of this exposure is important and should be correlated with the chest roentgenogram in which the "ground glass appearance" is characteristic. The lesions are limited to the lower halves of the lungs and there is hyperventilation in the upper portions. The cardiac outline becomes blurred and the domes of the diaphragm obliterated. The appearance of the heart and lower chest may suggest cardiac disease which should be definitely ruled out in a worker in an asbestos plant before diagnosing his condition as asbestosis. The diagnosis of asbestosis from the x-ray is not an easy matter, but the following features are fairly characteristic:

1. Lesions basal in character.
2. Hyperventilation in upper lobes.
3. Blurred cardiac outline.
4. Obliteration of the diaphragm.
5. No nodulation.
6. "Ground glass" appearance.
7. May be unilateral.

The occupational hazard of asbestos is not particularly significant. Its recognition as a clinical entity



ASBESTOSIS SILICOSIS

X-rays demonstrating shadows in asbestosis and advanced silicosis

TABLE V.
X-RAY APPEARANCE

ASBESTOSIS	SILICOSIS
Diffuse lesions limited to lower halves of lungs—Hyperventilation in upper portions.	Nodular lesions distributed more in upper and mid-lung fields or generalized-emphysema in lower halves.
Obliteration of the diaphragm.	Shortening of long diameter of chest with adhesions and tenting of diaphragm.
No nodulation.	Marked nodulation.
"Ground glass" appearance.	Discrete nodulation to massive conglomerate shadows.
May be unilateral.	Bilateral.

Certain other conditions such as fungus infections, miliary tuberculosis, miliary calcification and miliary carcinoma produce shadows in the roentgenogram which may be confused with those of silicosis. In these instances careful study of the film and previous occupational history will usually be sufficient to make an accurate diagnosis.

As in all diseases due to dust, treatment is an engineering problem rather than a medical one. Once fibrosis is established in the lungs, it is permanent. Progression of the disease is very slow and chronic, except in some instances where infection occurs. Continued exposure to hazardous dust appears to be the biggest factor in the advancement of fibrosis. When dust is kept at a safe concentration in the air of working places, there is no good reason why a person with uncomplicated silicosis cannot continue his occupation.

By the same token, extreme care should be taken to eliminate contact with tuberculosis from these workers. This can only be accomplished by repeated chest x-rays of all those exposed to hazardous dust and removal of the ones showing evidence of infection.

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Chest Conditions Simulating Silicosis

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