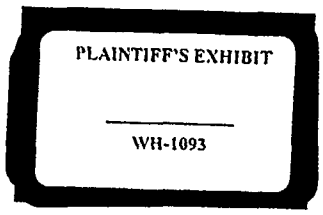

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THE RELATION between dust and certain diseases of the lungs has been recognized for centuries, but only in recent years has it been given the intensive study it deserves. In the fifth century B.C., Hippocrates noted symptoms in a metal digger, comparable 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.

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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	16	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

in industrial medicine is important, but judging from the comparatively small number of persons exposed in this country, the actual amount of disability resulting from the disease is not great.

In one of our plants where considerable asbestos is used in the manufacture of automobile brake linings, a recent survey of 189 employees exposed to variable amounts of dust, revealed no actual cases of fibrosis. A few men's films showed haziness which suggested evidence of the disease, but they were not sufficiently typical to warrant a diagnosis of asbestosis. However, it should be stated that the hazard in this particular plant is well controlled by adequate exhaust ventilation.

FROM the mass of evidence accumulated during the past 25 years, it now seems definitely established that silica is the public enemy No. 1 of those engaged in dusty occupations. By itself and in combination with other dusts, it offers a harmful exposure to a conservatively estimated 500,000 workers in the United States alone. In spite of this fact, most observers are now of the opinion that silicosis, uncomplicated by infection, is not a disabling disease. However, the fertile field it affords for the development of tuberculosis makes it imperative to disregard its benign characteristics and view it with suspicion until otherwise proven innocent.

The Committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association defines silicosis as "a disease due to breathing air containing silica ($Si\ O_2$), characterized anatomically by generalized fibrotic changes and the development of military 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." Gardner[14] points out that an objection to this definition is that it recites symptoms which are not always present except in advanced cases. He suggests the following as being more simple and adequate:

"Silicosis means a disease of the lungs due to breathing air containing uncombined silicon dioxide ($Si\ O_2$) dust, characterized anatomically by generalized nodular fibrotic changes throughout both lungs which are demonstrable by x-ray examination and by autopsy and resulting from any process of occupation involving inhalation of silicon dioxide dust."

The disease has been known as a clinical entity since the year 1871, but not until recent years was it recognized in industries other than hard rock mining. Silica is a most abundant constituent of rocks and minerals. With its compounds, it makes up 65% of the earth's crust. It occurs in two forms, free and combined. The combined forms are known as silicates (previously referred to).

Probably no other mineral is more widely used than silica and its compounds. Some of the more common occupations providing exposure are mining, tunneling, processing ores, quarrying, stone cutting and polishing, manufacture of abrasives, sand blasting, and grinding. For a complete list of the uses to which silica may be put see the table "Occupational Environment" by LaDoo.

To appreciate the pathological changes occurring in silicosis, it is well to recall the natural mechanism of defense in the respiratory tract itself. Nature has provided a barrier to dust and foreign particles at the point of entry into the body. The fine hairs of the nostrils and the mechanical arrangement of the upper

Diagrammatic representation of pathway of dust particle illustrating factors involved in natural defense mechanism of the body

respiratory tract offer considerable resistance to large particles and provide a very effective means of protection against all but the very fine dusts. Probably the greater part of the inhaled particles are eliminated by the upward current created by the ciliated epithelium of the trachea and bronchi. Beyond this point, the alveolar phagocyte or "dust cell" provides further elimination by transporting particles to the point where the cilia become effective.

It is believed that the particles from 0.5 to 3 microns in size are the only ones capable of producing pulmonary fibrosis. Those above 3 microns are eliminated by the upper respiratory tract while few of those below 0.5 micron actually remain in the alveoli.

In the actual production of fibrosis, the phagocytes, which probably originate from the inner surface of the air sac or the lining of the capillary blood vessels, pick up the fine dust and pass to the lymph spaces to be transported to more distant points by the lymph vessels along the normal course of drainage towards the hilum of the lung. In this process, particles become deposited in the interlobular tissue and nodes along the vessels. Here, it is thought, the body fluids produce a slow chemical reaction which results in the death of the dust cells and necrosis of the surrounding tissue. This stimulates the proliferation of fibroblasts and the ensuing scar inhibits the further removal of dust. The interference with the flow of lymph causes a spread of the phagocytes toward the pleura, and fibrosis appears in the interlobular septa and along the lymphatics which accompany the blood vessels. As the process goes on, small nodules of fibrous tissue become scattered throughout the lung giving rise to the so-called noduar condition typical of silicosis. These nodules may increase in size and eventually coalesce, forming massive fibrotic areas which destroy the air sacs and result in compensatory enlargement of neighboring alveoli, or in other words, emphysema.

Microscopically, the typical nodule of mature form consists of concentric whorls of dense hyaline collagen

fibres. The border is sharply defined with no exudate in the adjacent air spaces. The nodule may contain black pigment distributed either about the periphery or in focal collections in the interior.

Symptoms in silicosis depend largely upon whether the disease is complicated by infection (tuberculosis) or not. In simple or uncomplicated silicosis, they are absent or very few. In a great many instances people with well developed nodulation are entirely unaware that anything is wrong with their lungs. The commonest symptom is "shortness of breath," but frequently this complaint has to be elicited from the individual, and then he will often qualify it by stating that "he is not as young as he used to be." In some cases the actual dyspnea as observed by exercise tests, is less than the amount complained of. These discrepancies demonstrate the need of looking for causes of shortness of breath other than silicosis. Fever, cough, expectoration and rales are rarely encountered. If they are met with in moderate pulmonary fibrosis, they are probably due to an acute respiratory infection. Some silicotics tolerate colds and even pneumonia with surprising resistance. Physical signs are usually lacking.

When the disease progresses, however, and if the individual *does* show evidence of his condition, the symptoms and signs can be more definite. The shortness of breath is practically constant and is often associated with palpitation of the heart. The appearance of dry cough, sputum, loss of appetite, and increased fatigue should arouse suspicion of infection.

"Complicated silicosis" in the vast majority of cases means silico-tuberculosis. It is what the older men knew as "miners' complaint" or miners' consumption. The susceptibility of silicotics to tuberculosis is well known, but the reason for it is still obscure. The fatal outcome in practically all cases of death from silicosis is usually due to tuberculosis. Not so many years ago it was believed that persons with silicosis and tuberculosis would inevitably die. There is now reason to believe that this is not necessarily true. In my own experience, I have seen cases with well marked nodulation develop infection and show a positive sputum which later became negative after a period of hospitalization. A survey of practically any group of hard rock miners will show a considerable number with evidence of healed tuberculosis. However, the fact remains that once tuberculosis becomes superimposed on silicosis, the prognosis is extremely serious.

In chronic silico-tuberculosis the lungs, on gross appearance, are leathery or rubber-like in consistency, pigmented, and show areas of fibrous pleurisy where the lesions extend to the surface. The nodular fibrosis which is the characteristic feature can be felt immediately beneath the pleura. On section, the cut surface is rough and gritty, and the normal lung tissue has been practically replaced by extensive fibrosis or large masses of very dense, heavily pigmented scar tissue. The pleura is thickened and adherent. Emphysema is present. In active silico-tuberculosis, caseation and pneumonia of tuberculous origin may be present. On microscopic section this can be seen about the nodule.

Infection may be active or "fresh," healed or "old," and indeterminate. Symptoms will vary according to the stage of the disease. As the condition advances, the patient may exhibit the characteristic phthisic symptoms and signs, such as cough, loss of weight, dyspnea, chest pain, night sweats, tubercle bacilli in the sputum, and haemorrhage. He dies a characteristic tuberculous death. This is not always the case, however. Many individuals with far advanced silico-tuberculosis show surprising resistance and may be comparatively active up till a few hours before death,

Discrete nodulation of second degree silicosis (uncomplicated) in an iron miner

which may come rather suddenly and easily. I have seen such individuals and talked with them shortly before their demise and have anticipated no sudden termination of existence.

Progression of silicosis is extremely slow. Sometimes even in the presence of infection, it does not appear to advance very rapidly, but in certain instances the disease proceeds with astounding speed. Silicosis takes time to develop. Experience in South Africa indicates that approximately eight years are required for the condition to progress from a pre-silicotic stage to a silicotic one.

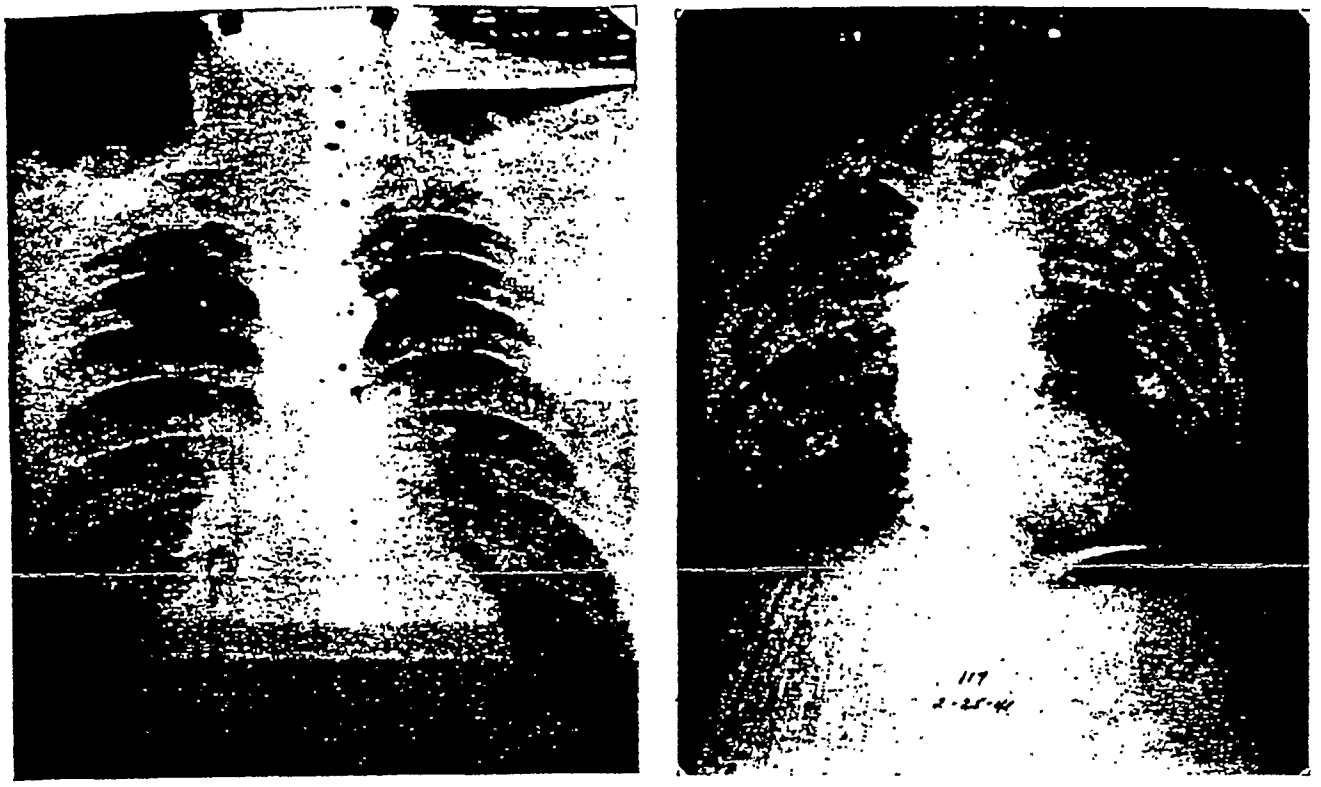
Because of the lack of physical signs and symptoms, a classification of the stages of silicosis is only practical when based on x-ray findings. Various observers have made their own tabulations, but the one worked out by Sampson[15] affords a practical and satisfactory grouping:

TABLE IV.

A. UNCOMPLICATED SILICOSIS:
   - N —Normal chest.
   - P_1—Stage of peritruncal exaggeration.
   - P_2—Stage of marked peritruncal exaggeration. (Pre-Silicosis)
   - S_1—First degree nodulation — (Linear markings obliterated and nodules up to 2 mm. in diameter present.)
   - S_2—Second degree nodulation (Nodules 2 to 4 mm. in diameter.)
   - S_3—Third degree nodulation (Nodules over 4 mm.)

B. COMPLICATED SILICOSIS:
   1. Silicosis with fresh infection.
   2. Silicosis with old infection.
   3. Silicosis with indeterminate infection.

In silicosis the x-ray appearance of the chest differs considerably from that of abestosis. In the accompanying table the more characteristic features of each are tabulated as an aid in differential diagnosis:



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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