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THE PNEUMONOCONIOSES

Which Industrial Dusts Are Inert? Which Are Harmful? Why?

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The term pneumonoconioses has been so loosely applied to industrial pulmonary conditions that some clarification appears to be desirable. It is an all-inclusive caption for a variety of pulmonary affections resulting from the inhalation of inert or harmful dust. Generically it does not imply fibrosis but simply "dust in the lungs." If one is to accept this meaning, therefore, pneumonoconiosis must include any retention of dust in the lungs, whether of industrial origin or not and whether of toxic, irritant, proliferative or inert dust. It must also include the anthracotic pigmentation from dust, carbon and smoke which all normal lungs exhibit; in the interpretation of pathologic change, the matter of degree as well as specificity must be considered.

From a practical standpoint, however, pneumonoconiosis refers mainly to pulmonary alterations attributable to those dusts encountered in industry which can cause proliferative reactions, roentgenologic patterns due to radiopaque properties or ample exaggeration of normal linear markings. These lungs are entirely distinct from the action of toxic dusts, such as lead or mercury, for which the lungs are merely as the point of entry into the body. They differ also from the reactions produced by mycotic and other organic dusts, as will be explained.

Read in a joint meeting of the Section on Preventive Medicine and Public Health and the Section on General Medicine at the Ninety-Seventh Annual Session of the American Medical Association, Chicago, June 23, 1948.

Industrial dusts are listed by Kronenberg and Morse[1] as follows:

I. Organic Dusts:
  A. Nonliving:
    1. Toxic and/or irritant
  B. Living:
    1. Bacterial
    2. Fungous

II. Inorganic Dusts:
  A. Toxic and/or irritant
  B. Fibrosis producing
  C. Nonfibrosis-producing

Classification of industrial dusts into "harmful" and "inert" categories is not easy because some which are usually considered innocuous may produce definite pathologic changes under certain conditions, such as prolonged excessive exposure. In the following modification of Sander's[2] grouping, certain dusts are designated as harmful even though they do not produce bluish pulmonary alterations.

Organic dusts arise mainly from animal or vegetable materials, but many are synthetic. They do not cause pulmonary fibroses but are capable of producing local irritation which results in such things as dermatitis, dental lesions, irritation of the mucous membranes and conjunctivitis. Sappington[3] has listed more than one hundred occupations in which these symptoms and/or allergic manifestations may be produced from substances like pollens, horse hair, furs, wool and wood dust.

Byssinosis, an occupational condition resulting from dust encountered in the cotton industry, has been reported in Europe as a definite occupational disease, but its existence as a clinical entity in this country is questionable. Acute symptoms and roentgenologic findings suggestive of pneumonia of the influenzal type have followed exposure to an organic dust produced by crushing or shredding bagasse, a waste product of

1. Kronenberg, M., and Morse, K. M.: Health Hazards of Occupational Environment, Circular 154, Springfield, Ill., Illinois State Department of Health.
2. Sander, O. A.: The Pneumoconioses, personal communication to the author, Nov. 14, 1944
3. Sappington, C. O.: Essentials of Industrial Health, Philadelphia, J. B. Lippincott Company, 1943, pp. 186-187.

TABLE I.—*Industrial Dusts*

Harmful or Potentially Harmful		Inert	
Substance	Type of Effect	Substance	Type of Effect
I. Organic			
A. Animal	Allergic		
Dander			
Fur			
Hair,			
Etc.			
B. Vegetable	Allergic or infectious pneumonitis		
Pollens			
Grains			
Cotton (byssinosis)			
Sugar cane (bagassosis)			
Tobacco (tabacosis)			
C. Bacteriae			
D. Fungus			
Aspergillosis			
Coccidioidomycosis	Irritant		
Blastomycosis	Fibrogenic		
Sporotrichosis	Allergic		
Geotrichosis	(Occasionally occupational)		
Monilinsis			
Histoplasmosis			
II. Inorganic			
A. Metallic			
Chrome and its compounds	Irritant, causing local irritation of upper respiratory tract	Carbon (Anthracosis)	Roentgenologic changes only
		Calcium (Calcicosis)	
		Iron (Siderosis)	
Cadmium		Barium (Baritosis)	
Arsenic	May be toxic	Tin (Stannosis)	
Lead	Absorption and intoxication	Aluminum	
Manganese*		"Carborundum" (silicon carbide)*	
Mercury		Emery †	
Zinc		Aluminum oxide	
Beryllium	Pneumonitis		
Bauxite	Chronic granulomatosis (exact etiology undetermined)		
B. Free Silica (silicon dioxide)			
C. Silicates		Talc, mica, soapstone, feldspar, etc.	(Few cases reported: e. g., talc "pneumonoconiosis," probably due to quartz impurities)
Asbestos (Only silicate thought capable of producing fibrosis)	Asbestosis	Talc	
		Mica	
		Soapstone	
		Feldspar	
		Etc.	

* Carborundum does not exist in nature but is a manufactured product fused from sand, coke and sawdust.
† Emery is an aluminum oxide manufactured from bauxite. Corundum is natural aluminum oxide.

sugar cane. The condition is described as bagassosis and, like byssinosis, may be due to an allergic reaction rather than to actual pathogenic properties of the dust itself. There is also some recent evidence to show that bagassosis may be caused by a fungus or possibly by bacteria attached to the dust and that the dust itself is relatively innocuous.[4] C. F. Long[5] investigated more than 1,247 workers in the tobacco industry and concluded that tabacosis does not develop in the lungs of those who inhale tobacco dust. Recent studies, however, by McCormick, Smith and Marsh[6] indicated that there may be a significant free silica hazard in the stemming and redrying operations in that industry.

Pulmonary diseases caused by fungous infections have been receiving more attention in recent years. Increasing recognition of these cases has led many observers to believe that they may be more prevalent than is generally supposed. Mycotic diseases are usually nonindustrial and are found only in certain geographic locations. Nevertheless some cases have been attributed to occupational exposure. In 1945 Coe[7] reported a case of bronchopulmonary aspergillosis, occurring in an employee of the Chicago Stock Yards, for which an award of compensation was granted and upheld by the Industrial Commission of Illinois.

Fungi are filamentous plants of simple structure, among them being various yeasts and molds. They may attack the lungs or other parts of the body and give rise to such conditions as moniliasis, aspergillosis, sporotrichosis and coccidioidomycosis. It is important for one to bear this fact in mind, particularly when reviewing the industrial roentgenogram of the chest. The pulmonary shadows produced by yeasts and molds may easily be confused with those caused by free silica unless careful consideration is given to the patient's occupational history and exposure to dust.

In recent years considerable research has been done on multiple pulmonary calcification. This condition

4. Gerstl, B.; Tager, M., and Marmaro, N. A.: Pathogenicity of Bagassar, Arch. Path. 44: 141-149 (Oct.) 1947.

5. Long, C. F.: Tobacco Dust and the Human Lung, Indust. Med. 8: 365-369 (Sept.) 1939.

6. McCormick, W. E.; Smith, M., and Marsh, S. P.: Health Hazards of the Tobacco Stemming and Redrying Industry, J. Indust. Hyg. & Toxicol. 30: 43-51 (Jan.) 1948.

7. Coe, G. C.: Primary Bronchopulmonary Aspergillosis, an Occupational Disease, Ann. Int. Med. 23: 423-425 (Sept.) 1945.

CORRECTION: Paragraph at top of Page 5, Lines 7 and 8.

"It now seems reasonably certain that they are caused by the fungus Histoplasma capsulatum." should be more accurately stated as follows: "These calcifications are often associated with skin sensitivity to antigen derived from this fungus".

It has not been shown conclusively that Histoplasma capsulatum is the actual cause of these pulmonary calcifications.

poisoning. These include such substances as chromium and its compounds, cadmium and arsenic. While it is recognized that the great majority of industrial dusts are inert, as far as causing disabling fibrosis is concerned, one should not forget that certain other substances can produce dust or fumes which may cause serious illness. These are the heavy metals, such as lead, mercury and manganese. As pointed out earlier, dust or fumes from them do not cause structural lung changes but merely use the lungs as a portal of entry into the body. In later years serious toxic effects have been attributed to beryllium and its compounds and to fumes arising from the fusing of bauxite in the manufacture of artificial abrasives. These two conditions have resulted in severe pneumonitis with definite roentgenologic findings and some fatalities. The exact etiology has not yet been determined in either, and it is questionable whether they should be classified as pneumoconioses until their status has been more accurately demonstrated.

When inhaled over long periods of time certain dusts may produce shadows in the roentgenogram which closely resemble those of silicosis, so that one should resist the temptation to make a diagnosis of occupational disease on the roentgenologic factor alone.

Some physicians have had the embarrassing experience of making a "long distance" diagnosis solely from the roentgenogram, only to find out later that the person concerned had worked in an office all his life without ever having been subjected to dangerous dust. Even when the roentgenologic evidence is associated with a history of employment in a dusty industry, care should be exercised by the examiner to make sure that the *occupation offers adequate exposure to dangerous-sized* particles of material capable of causing actual damage to pulmonary tissue. It is still a common practice among physicians and roentgenologists who have insufficient knowledge of industrial environments or modern hygienic practices to arrive at premature and inaccurate conclusions simply because they have based their interpretations on too little information. Unfortunately they sometimes express very dogmatic opinions without stopping to consider the effect on the employer or the worker himself.

The distressing features of this conduct are that employers become needlessly alarmed, forsake their lifetime vocations, suffer financial loss and forfeit seniority. Industry is deprived of the services of skilled workmen, confronts lowered production and faces unjust and costly litigation--all because of unwarranted roentgenologic interpretation and unfamiliarity of the examiner with progressive industrial medicine and hygiene.

The number of unrelated conditions capable of producing roentgenographic shadows in the lungs simulating silicosis is constantly increasing. In 1946 I listed some 23 of these, which ranged all the way from certain cardiac afflictions to exaggerated linear markings representing pulmonary vascular changes due to advancing age.

Gardner[8] and others have demonstrated that silicosis is likely to develop only when significant concentrations of respirable-sized particles (those smaller than 10 microns, and especially those smaller than 3 microns) of free or uncombined silica have been inhaled over long periods of time (2 to 25 years, with an average of 10 years). This fact emphasizes the need for careful appraisal of injurious contamination by dust in the

8. Gardner, L. U.: Pathology of Silicosis, in Kueckle, B. E.: Second Symposium on Silicosis, Wausau, Wis., Employers Mutuals, 1935.

actual working places and correlation of this appraisal with the occupational history, clinical and roentgenologic findings when one is evaluating any case of suspected occupational fibrosis. The most important consideration in any such investigation is, "How is the man himself affected?" Although the amount of visible dust in the atmosphere around a specific job appears to be great, that fact does not necessarily mean that pathologic quantities of the particles are reaching the alveoli of the lungs. Accumulations at the breathing level and the defensive mechanism of the upper respiratory tract must be taken into account along with the length and constancy of exposure.

The absence of pathologic change in the presence of the inert dusts may be explained in part by several factors. The natural barrier to dust provided by the anatomic arrangement of the upper respiratory tract, the ciliated epithelium of the mucous membrane lining the bronchi down to the terminal bronchioles, and the phagocytic cells in the alveoli all serve to eliminate inhaled dust. When one considers a pulmonary ventilation rate of some 18 cubic feet per hour, which may increase from 50 to 80 cubic feet per hour on exertion, it is not difficult to see how well this defensive mechanism of the body works. The long period required for the development of recognizable silicosis, even under heavy exposure, attests the efficiency of the arrangement.

The size of the particles of the offending dust is also an important consideration. To be inhaled, dust must be air borne and also must be small enough to reach the alveoli. The diameter of an alveolus is approximately 250 microns, but, as pointed out previously, experience has demonstrated that very few particles of more than 10 microns in size ever reach them, certainly not enough to produce damage. And, as was also stated before, the inert dusts do not appear to have a chemical reaction on lung tissue, such as silica does. This factor is perhaps one of the most important reasons why these dusts do not cause fibrosis

It may be said that up to the present time only free silica and asbestos have been believed capable of causing definite pulmonary fibrosis, and the reaction in each instance is quite different. Most of the other pneumono-

conioses exhibit a characteristic tissue response. According to Gardner[9] the roentgenographic appearance in these cases consists of a mere accentuation of the normal, branching, treelike shadows, cast chiefly by the blood vessels. It represents a simple linear fibrosis and is difficult to distinguish from the mild accentuation of

TABLE 2.—*Classification of Pulmonary Conditions, Roentgenograms of Which Simulate Those of Silicosis*

A. From Industrial Dusts	
I. Organic	
(a) Byssinosis	
(b) Bagassosis	
(c) Tabacosis	
II. Inorganic	
(Inert)	
(a) Anthracosis	
(b) Radiopaque Deposits	
Barium (Baritosis)	
Iron (Siderosis)	
Tin	
(Toxic)	
(c) Reaction to Beryllium	
(Proliferative)	
(d) Asbestosis	
B. From Nonindustrial Dusts	
I. Mycotic	
(Yeasts and Molds)	
(a) Aspergillosis	
(b) Monilliasis	
(c) Wood dust spore infection	
(d) Coccidioidomycosis	
(e) Blastomycosis	
(f) Actinomycosis	
II. Miscellaneous	
(a) Miliary tuberculosis	
(b) Sarcoidosis	
(c) Miliary Stenosis	
(d) Cancers (metastases)	
(e) Vascular changes	
(f) Miliary calcicosis ("wheatens")	
(g) Erythremia	
(h) Bronchiolitis	

linear markings sometimes seen in roentgenograms of persons with no known history of exposure to dust. He designated this effect as "benign nonspecific pneumonoconiosis," and it includes every known reaction to mineral dust except that to soluble poisons, such as lead; the specific diseases silicosis and asbestosis, and certain "roentgenologic conditions," such as baritosis, stannosis and the pseudonodulation of arc welders.

9 Gardner, L. U.: The Pathology and Roentgenographic Manifestations of Pneumoconiosis, J. A. M. A. 114:535-545 (Feb. 17) 1940.

Numerous observers have pointed out that deposition of inert iron oxide into the lungs from occupations like welding, burning, polishing and grinding has resulted in pigmentation of the pulmonary tissue, which gives rise to discrete nodular shadows closely resembling those produced by free silica. Postmortem examinations of several patients with these signs have shown no evidence of the typical nodular fibrosis of silicosis.

For the past two and a half years, because of roentgenologic findings suggestive of silicosis in the roentgenograms of the chest of some burners and grinders, my associates and I have been conducting experimental work at the Saranac Laboratory, in which studies animals have been exposed to representative concentrations of air-borne dust from grinding operations. Analysis of this dust shows relatively low amounts of free silica but very high quantities of iron oxide particles. Animals killed thus far demonstrate no pulmonary reaction except that comparable to reactions from other inert dusts. The work is continuing, and results will be published when sufficient time has elapsed to produce valid conclusions. Other inert dusts, like those derived from tin and barium, are capable of causing changes in the roentgenologic pattern which, although very evident in the film, are nondisabling and do not result in fibrosis. They are very important, however, from the standpoint of diagnosis.

It has long been recognized that silica, or silicon dioxide (SiO_2), which makes up 60 per cent of the rocks and minerals of the earth's crust, is the specific cause of silicosis. It exists either in the free state or in combination with certain mineral oxides to form silicates, such as mica, clay and feldspar. Thus far only free silica is known to produce silicotic fibrosis of the lungs. The term free silica usually means quartz, of which there are many varieties. There are other forms of free silica, as shown in table 3.

It has not been demonstrated authentically that silicates other than asbestos produce real pulmonary fibrosis. Pulmonary changes due to dusts containing them have been reported, but they probably resulted from small amounts of free silica, which existed as

impurities. However, more investigation is necessary before one can make definite assertions about the innocuous character of these silicates.

Industrial pulmonary disease offers a real challenge to the general practitioner as well as the industrial physician. As the family doctor he may be called on to express his opinion as to whether changes observed in the roentgenogram of the chest are caused by occupation or not. Even though he is not directly associ-

TABLE 3.—*Forms of Free Silica, Causing Silicosis, Classic and Modified*

Crystalline (Crystalline structure)
Crystobalite
Tridymite
* Quartz
Cryptocrystalline (Crystal structure too fine to be easily distinguished)
Chalcedony
* Flint
* Chert
Jasper
* Tripoli
Amorphous (Noncrystalline)
* Diatomite
* Silica Gel
* Vitreous Silica
Opal

* The compounds marked with an asterisk are those of the greatest industrial importance.

ated with industry, most manufacturing concerns will welcome his interest and be more than willing to afford an opportunity for visits to the plant so that he can obtain first hand knowledge of actual working conditions. Through such contacts he will find that his appraisal of industrial diseases will be greatly enhanced, and the experience will prove to be fascinating and surprisingly valuable.

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