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INDUSTRIAL DUST

Hygienic Significance, Measurement and Control

BY

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fully that, if he has silicosis, he got it in Canada and Canada will take care of him.

In the case of a first-stage silicotic without tubercular infection, there is no method, as yet, for determining his lessened efficiency. The lung change, if any, caused by his dust inhalation is too small to measure.

McCann and Hurtado (177) have estimated disability from lung fibroses of various origins but have not yet succeeded in developing a method that is simple enough for routine use by compensation boards, insurance examiners, and the like. Their procedures involve various simple measurements of respiratory function but they have not established any methods of detecting fibrosis which are more sensitive than the present X-ray examination.

Of course, industry would welcome a diagnostic routine for detecting early harm from dust or, better, for forecasting harm. No such method is available and it seems fruitless to expect such help. The pneumoconioses are not diagnosed until harm is demonstrable by X-ray.

Collis (42) has stressed the difference between the age groupings in pulmonary tuberculosis and in silicosis, with or without tubercular infection superimposed. Tuberculosis is most prevalent between the ages sixteen to twenty-four, while silicosis rarely becomes disabling until later in life. Inasmuch as a good many years of dust exposure are required before the average case of silicosis (or asbestosis) becomes evident, it is obvious that it is not likely to be acquired early in life. However, there is an epidemiological side to the question which is of great importance and it is that which Collis especially emphasized. The average healthy individual has acquired a fairly effective immunity or resistance to tuberculosis. His chest X-ray is likely to show one or more healed scars or fibrosed areas from tuberculosis. Such an individual, Cummings emphasizes (211), is a better silicosis risk than the man who shows no evidence of past exposure to pulmonary tuberculosis. Consequently it is wise to select for dusty jobs men who are past the age of forty and not young men just taking up a trade.

Asbestosis.—The X-ray picture of the typical asbestotic chest is confusing to the layman. The effect is described as a diffuse fibrosis. Characteristic silicotic nodules are absent and even

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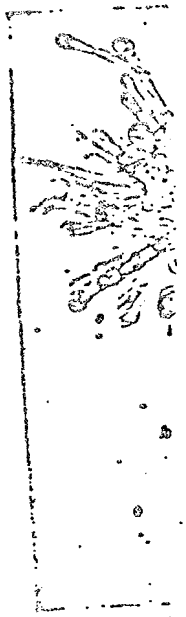

FIG. 13.—Asbestosis b

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The pathology produced by asbestos is not like that of silicosis. The asbestos fibers group about the neck of an alveolus and shut it off, causing what is known as *atelectasis*. There is no definite migration or transportation of the dust particles to the lymph nodes and no formation of the fibrous nodules as shown in Fig. 13. As the atelectatic areas increase, the reduction in lung

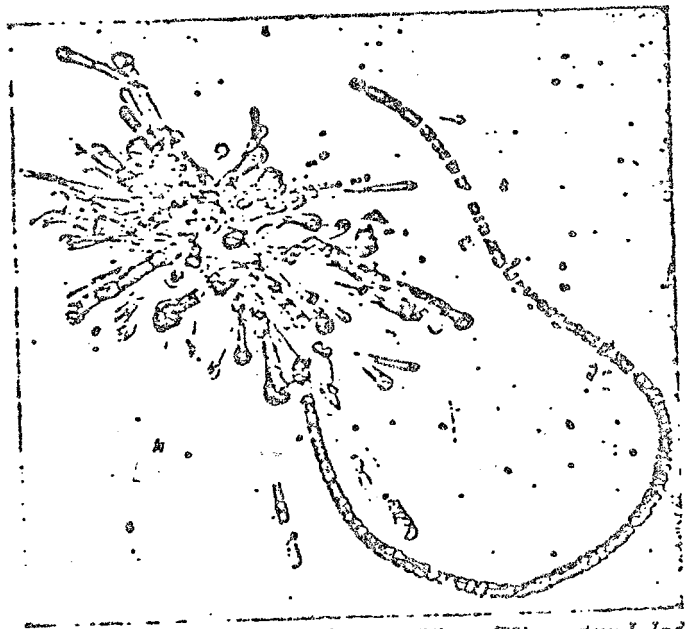

FIG. 15.—Asbestosis bodies in sputum. (*After Ellman* (75); *courtesy J. Ind. Hyg.*)

area causes serious dyspnea or labored breathing. Lanza (158) suggests that the enlarged hearts noted frequently in his cases of second-stage asbestosis may be the result of the increased work of the heart resulting from this condition; it takes more work to pump blood through the atelectatic than through the normal lung.

In silicosis it seems to be a general rule that, after a certain point, the victim's condition grows worse even if his dust exposure has ceased. But Wood and Gloyne (258) state that they have seen patients with asbestosis "whose condition appears to have

remained stationary since stopping work in the factory," but they advise definitely that the asbestotic individual be removed from his dusty job. Merewether (183) and Lanza are less certain on this point.

Asbestosis Bodies. In the lungs of patients who died after prolonged exposure to asbestos dust and in the sputum of men with considerable asbestos-dust exposure are found what first were called *curious bodies* and later *asbestosis bodies* (Fig. 15) (75). While somewhat similar bodies occur in the lungs of coal workers and even of normal persons, it is admitted that asbestosis bodies in sputum are characteristic of asbestosis. Stewart (223) gives considerable diagnostic weight to their presence.

Pneumoconiosis in Animals. Silicosis has been produced experimentally by quartz dusting guinea pigs, rabbits, mice, cats, and domestic fowl, thus emphasizing the specificity of quartz dust to biological tissue. In like manner asbestosis has been produced in experimental animals.

The Equidae, such as horses and mules, apparently are not susceptible to ordinary pulmonary tuberculosis but there is no reason to believe they have any special immunity or resistance to silicosis. The normal silica content of horses' or mules' lungs is not known but it would seem wholly reasonable to use the lungs of animals which have worked 5 to 15 years underground in mines as physiological dust samples. A brief report on this subject was published by Haynes (121) but data such as one needs are singularly lacking.

TOXIC DUSTS

Poisoning from inhaling toxic dusts is much more likely than poisoning from swallowing them. Dusts that reach the lungs may pass directly into the blood stream, thence to the heart, and immediately be pumped all over the body. Distribution to all the body tissue is thus brought about rapidly and effectively. But dust taken in with the food goes to the stomach and the major part passes out in the feces. Some is picked up by the portal blood circulation and moves on to the liver. That portion which causes poisoning must first pass through the liver, which is an effective filter and detoxifier; only then can it enter the general circulation.

EXHIBIT K: 8e

EXHIBIT "M" . 8c.

Handbook of Dangerous Materials

By

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BOOK DIVISION

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1951

Arsenical Mixtures or Compounds, N. O. S.* Solid

Hazardous Properties: Poisonous materials. See Arsenic.

Shipping Regulations:†

Poison B 352, 354* Poison label 200 lbs. (max. lot)

Arsenical Dip, Liquid (Sheep Dip)

Hazardous Properties: Poisonous liquid. See Arsenic.

Shipping Regulations:†

Poison B 338, 349* Poison label 55 gals. (max. lot)

Arsenical Dust

Hazardous Properties: A poisonous material. See Arsenic.

Shipping Regulations:†

Poison B 352, 355* Poison label 200 lbs. (max. lot)

Arsenical Flue Dust

Hazardous Properties: Poisonous material. See Arsenic.

Shipping Regulations:†

Poison B 352, 355* Poison label 200 lbs. (max. lot)

Arsenous Acid, Solid

Hazardous Properties: A poison. See Arsenic.

Shipping Regulations:†

Poison B 352, 361* Poison label 200 lbs. (max. lot)

Arsenous and Mercuric Iodide Solution, Liquid

Hazardous Properties: A poisonous liquid. See Arsenic and Mercury.

Shipping Regulations:†

Poison B 338, 349* Poison label 55 gals. (max. lot)

Arsine

*Maximum Allowable Concentration in Air:** 0.05 parts per million.

Hazardous Properties: See Arsenic. Caution! Arsine can be liberated from unusual places. For instance, in cleaning out steel tanks which contained concentrated sulfuric acid it is possible to liberate arsine; although concentrated sulfuric acid does not react with steel, dilute acid does react with it momentarily, liberating hydrogen, which reacts with the arsenic, forming arsine. This is but one of the very numerous possibilities for industrial exposure to this toxic material.

Synonyms: Arsenic hydride; arseniuretted hydrogen

Formula: AsH_3

M. Wt.: 77.9 *M. P.:* —113.5°C

B. P.: —55°C *Density:* 3.484 g/liter

Description: Colorless gas

Shipping Regulations:† Poison label (1).

Arsphenamine

Hazardous Properties: A toxic material. See Arsenic.

Synonyms: 3-Diamino-4-dihydroxy-1-arsenobenzene hydrochloride; 606

Formula: $C_{12}H_{12}As_2N_2O_2.2HCl.2H_2O$ *M. Wt.:* 475.0

Description: Light yellow hygroscopic powder

Shipping Regulations:† None.

"Artic"

Trade mark of a refrigeration grade of methyl chloride. A proprietary material. See under component as listed.

Asbestos Particles or Asbestos Dust

*Maximum Allowable Concentration in Air:** 5 million particles per cubic meter.

Hazardous Properties: Asbestos is a class name for various minerals of a fibrous nature. The commercial material is mainly fibrous serpentine, known as chrysotile. The implication is that this material merely constitutes a nuisance dust. The figure given for the M. A. C. is probably the safe limit. There is a specific lung disease known as asbestosis, which may be caused by this material; it can also cause chronic conjunctivitis with hypertrophy (6). Asbestosis is a comparatively new disease which was first described by English writers (1920 to 1925). Finely ground asbestos is said not to be dangerous, and this theory is supported clinically. The time necessary to develop asbestosis is from 7 to 9 years, if the exposure concentration is fairly high. Lower concentrations are likely to require 15 to 20 years of exposure. Once established, the disease is progressive, even though exposure has stopped. It would seem that the irritation due to this material is related to its physical nature rather than its chemical composition. The first sign is dyspnea; the cough is usually dry and auscultation reveals few if any physical signs; loss of weight, pallor and cyanosis are also early symptoms. Later, emaciation and chest symptoms appear. There is a distinction between this disease and silicosis, *i.e.*, the chest appears emaciated and lacks the robust character noticed in silicosis. Occupational exposure occurs in the packing, insulating and fire-proofing industry or where this material is combined with textiles.

Treatment and Antidotes: Rest and removal from exposure are indicated. Consult a physician.

Storage and Handling: Personnel exposed to high concentrations or to unknown concentrations of asbestos particles should wear respiratory protection of an approved design.

Asphalt

Hazardous Properties: Asphalt causes skin eruptions which vary roughly in relation to the specific gravity and boiling point of the asphalt or bitumen. It can cause dermatitis. The fumes evolved are noxious and unpleasant with a strong odor, so that kettles of this material

EXHIBIT "L" 8b

Please xerox an extra copy of all this for BC. These are primary file mat'ls

OCCUPATIONAL MEDICINE AND INDUSTRIAL HYGIENE

By

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CHAPTER XXX

ASBESTOSIS

Asbestosis is a comparatively new disease. The first comprehensive reports regarding asbestosis came from the English writers in the years between 1920 and 1925. In America in the early thirties studies were made by Lanza, Sayers, Bloomfield, and others.

Asbestos is a hydrated magnesium silicate. Occupational exposure occurs in those trades where it is used for packing, insulating, or fireproofing, or where it is combined with cotton or other material in textile processes.

Signs and Symptoms.—As in silicosis,[1] the first sign is dyspnea, the cough is usually dry, and auscultation reveals few, if any, physical signs. Loss of weight is usually noticeable, the color is pale, and cyanosis is a fairly early sign. After some time, fine, crackling râles may be heard, and the chest appears emaciated and lacks the robust character noticed in silicosis. Early asbestosis must be differentiated from acute emphysema. If tuberculosis is present, the symptoms may take on the character of this disease, but this complication is far less frequent than in silicosis.

Pathology.—

Pulmonary Changes.—The pulmonary fibrosis of asbestosis is diffuse in character, peribronchial, and basal in location in contrast to silicosis in which the fibrosis is nodular in character and present in the upper part of the lungs. As the process continues, the fibrosis extends into almost all portions of the lung tissue. Bronchiolectasis and bronchiectasis are present within the substance of the fibroid areas, and bronchopneumonia and acute tracheobronchitis often cause the death of these patients. The pleurae, especially in the basal regions, are thickened and adherent. In some cases the pleural sac is completely obliterated; in others a fibrous or serous exudate may be present within it.

Action of Asbestos and Silica.—The difference between chrysotile asbestos and quartz silica in their mode of action has caused considerable speculation as well as experimentation. Gardner[2] particularly, at the Saranac Laboratory, carried on intensive study of these two materials. He felt that while the action of free silica is chemical in nature, the action of asbestos is mechanical. He stated:

"We have come to the conclusion that inhaled asbestos fibres are irritating not because they are silicates but because they are stiff fibres which mechanically irritate the lungs. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body; only those in the lungs are affected. It was inferred that these organs were

affected because the movements of respiration are so much more rapid and continuous than those of other viscera. Then it was discovered that if asbestos was ground very finely so that few of the fibres were longer that 2 microns in length the irritating property of the asbestos was practically destroyed. Inhalation experiments with such fine chrysotile asbestos were carried out over a period of several years. No fibrosis has developed in spite of the fact that an average atmospheric concentration of 125 million particles per cubic feet of air has been maintained. In contrast in a previously reported experiment one-third this concentration of long fibre asbestos dust produced well marked fibrosis after about two years."

If the effect of asbestos were chemical, one would expect that a decrease in size would accelerate tissue response. With free silica large particles have little effect, but as their size decreases cellular reaction becomes more vigorous and even constitutional symptoms may ensue. With fibrous asbestos the reverse is true.

The histology of early asbestosis does not suggest a chemical injury. Even under the most extreme conditions that can be created by artificial injection there is no preliminary phase of tissue necrosis with infiltration of leucocytes as occurs with high concentrations of very fine quartz. The connective tissue cells merely multiply very slowly in areas where the asbestos fibres are caught in the bronchioles. As collagen forms and contracts, the air spaces are obliterated by scar tissue. In experimental animals, at least, this change is not a progressive one after cessation of exposure to the dust as is the case in the response to quartz. Perhaps the reason is the deposition of the peculiar iron-containing coating on the surface of the inhaled fibres giving rise to the characteristic "asbestosis bodies" (Figs. 50 and 51).

Finally, it is most suggestive that among dozens of different silicate minerals only the five known as asbestos, which are unique because they are fibrous in structure, should be commonly recognized as pulmonary irritants. The variation in chemical composition within this group is greater than that between them and many other silicates. In fact, chrysotile asbestos has the same chemical formula as a nonfibrous silicate, serpentine, which is physiologically inert. Obviously irritation would seem to be associated with the physical rather than the chemical composition of these minerals.

One point of practical significance may be indicated by these observations: namely, that very finely ground asbestos is not dangerous. This conclusion has support in clinical observation, for it has long been known that at the Thetford Mills there was no clinical asbestosis even though in former years the atmosphere was very dusty and the dust was extremely fine. The fact that fabrication of fibres of the same mineral in American plants could produce disease was one of the puzzling fea-

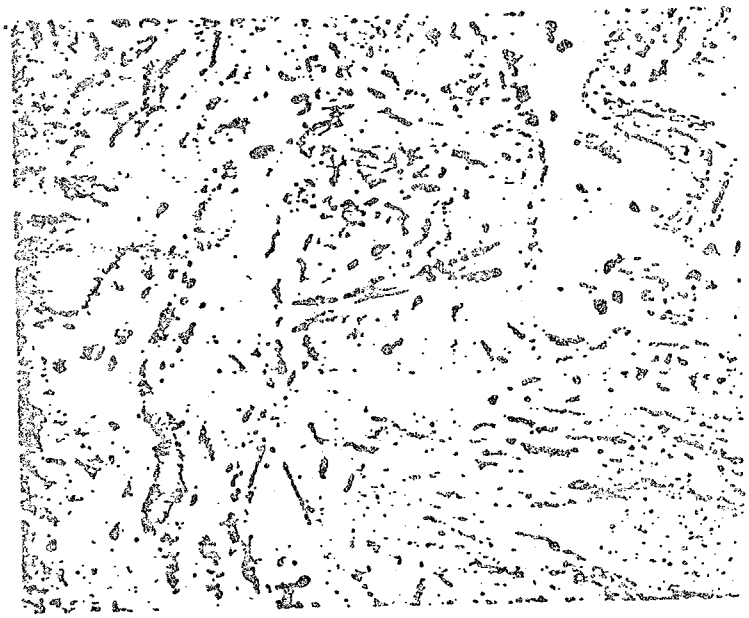

Fig. 50.—Right lung, showing markedly thickened pleura with fusion of interlobar pleura. Dense fibrous tags over entire surface. (U. S. Public Health Service, Bulletin 241.)

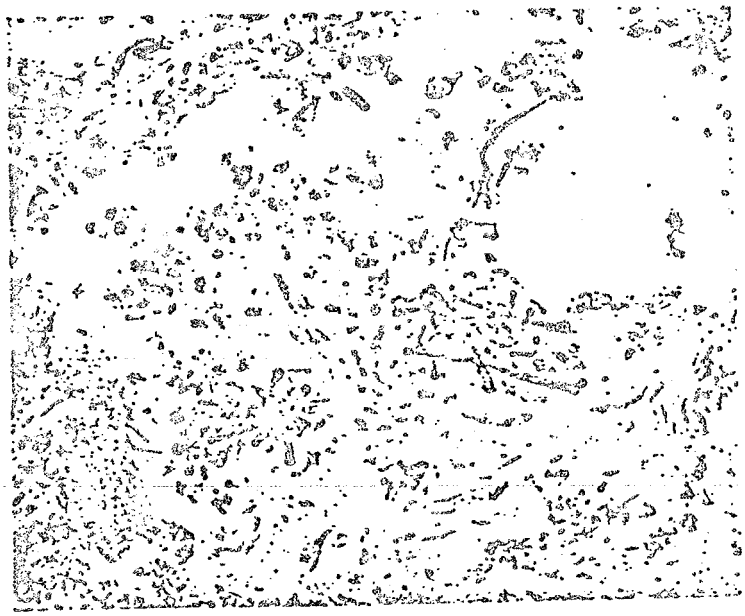

Fig. 51.—Asbestosis bodies accompanied by giant cells. (U. S. Army Medical Museum, Specimen 52255.)

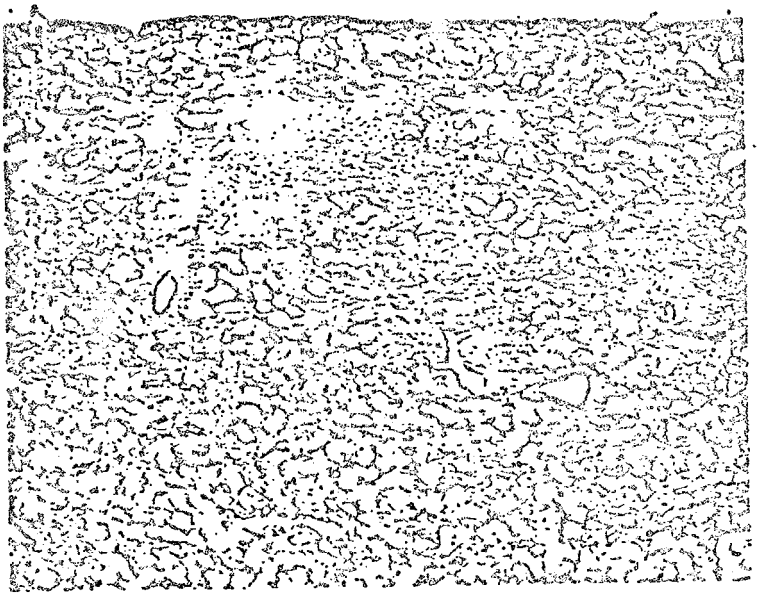

Fig. 52.—Section of a lung in asbestosis. (Gardner.)

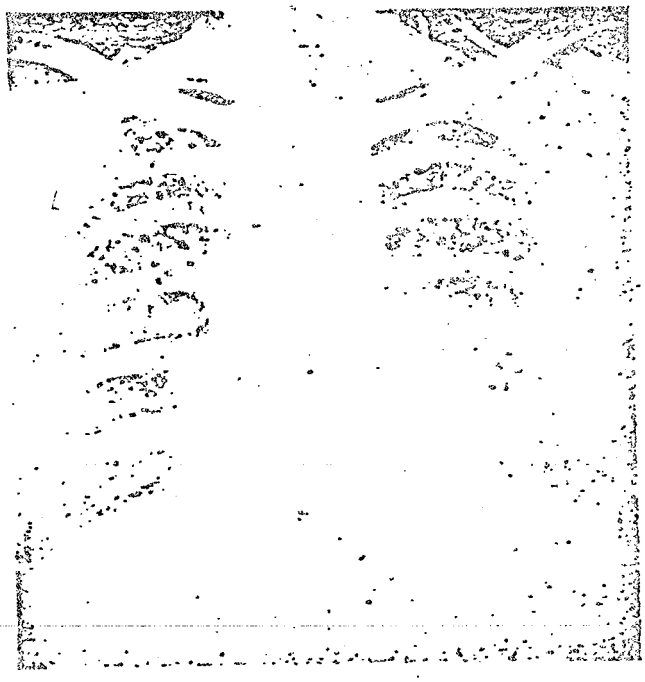

Fig. 53.—X-ray film of a patient with asbestosis. (Pendergrass.)

tures of this disease. But these experiments offer a plausible explanation. The fine dust in the mills is composed of serpentine and extremely short chrysotile fibres; that in the spinning and weaving mills contains many more long fibres.

Microscopic Appearance.—In the early phases of the disease there is a thickening of the alveolar septa as a result of fibroblastic proliferation. The alveolar spaces contain numerous alveolar phagocytes. With progression of the disease, fibrosis becomes more marked and the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few remaining alveoli which lie in the area of fibrous tissue are lined by a low cuboidal epithelium and have a glandular appearance (Fig. 52).

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures (first described by McDonald[4]) known as asbestosis bodies. These bodies are slender in their center and bulbous at each extremity. Many are arranged like strings of graduated beads with the largest at the end of the chain.

X-Ray Findings.—Certain peculiarities exist here as contrasted to silicosis. In asbestosis, the lesions may be bilateral or largely unilateral. According to the experience of Pendergrass,[3] the roentgenologic findings in asbestosis are largely limited to the lower half or two-thirds of the lung fields, while in silicosis the upper portions of the lungs are also involved. In moderately advanced asbestosis, x-rays will reveal lessened ventilation of the lung fields, the parietal pleura is thickened, and there is a "ground-glass" appearance to the picture. The vascular shadows lose their identity. Nodulation is absent. Pendergrass[3] feels that a roentgenologic diagnosis of early asbestosis is unreliable and that the condition must be moderately or markedly advanced to make a differential diagnosis from the roentgenograms (Fig. 53).

Medicolegal Aspects.—It is felt that the time necessary to develop asbestosis is, on the average, from seven to nine years in a fairly high concentration; with a less severe concentration, from fifteen to twenty years. The allowable concentration is 10,000,000 particles per cubic foot of air, of a size between 0.5 and 5.

Once established, the disease is progressive, even after the cessation of exposure. The basis for diagnosis is similar to that given under Silicosis.

References

1. Lanza, A. J.: Asbestosis, J.A.M.A. 106: 368 (Feb.), 1936.
2. Gardner, L. U.: Recent Developments in Relation to Silicosis, Indust. Med. 9: 45 (Feb.), 1940.
3. Pendergrass, E. P.: Silicosis and Asbestosis, New York, 1938, Oxford University Press.
4. McDonald, S.: Histology of Pulmonary Asbestosis, Brit. M. J., 2: 1025, 1927.