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INDUSTRIAL HYGIENE AND TOXICOLOGY

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of aluminum by physicians for therapeutic or prophylactic purposes, makes only metallic aluminum available. Some investigators, notably Hannon,[73] have reported marked success in the treatment of patients with severe disability. However, when purely objective criteria have been used to measure disability the results have been somewhat less striking. Gardner, Dworski, and Delahant[74] have suggested that variations in response to aluminum therapy may be due in part to the nature of the dust that has caused the silicosis. They suggest that dust which is primarily free silica may react with aluminum more readily than dust which contains high percentages of iron. The use of aluminum generally in industry for the therapy or prophylaxis of silicosis should be contemplated only with the utmost caution.

I. ASBESTOSIS

Asbestos is a hydrated magnesium silicate. More than 90 per cent of the raw mineral used in this country and Great Britian is produced in the Canadian chrysotile mines. Asbestos is used in two general types of manufacturing processes. It may be used either by itself or mixed with other insulating materials such as diatomaceous earth for fireproofing, packing, or insulating; or it may be combined with cotton and woven as a textile for fireproof and heat-resistant clothing and other substances. Lanza[75] estimates that there are about 10,000 persons exposed to asbestos in the United States. Most observers feel that the incidence of asbestosis in American asbestos workers is quite low. Asbestosis has, however, been reported more frequently in England and Canada.

Lanza, McConnell, and Fehnel[76] concluded from their study of asbestosis that: Prolonged exposure to asbestos dust causes pulmonary fibrosis different from that produced in silicosis and demonstrable by roentgenogram. Clinically it appears to be milder than silicosis.

Definite cardiac enlargement frequently was found to be associated with asbestosis.

A predisposition to tuberculosis, due to asbestos dust, was not indicated although it was not known to what extent asbestosis may add to the mortality from pneumonia and acute nontuberculous pulmonary infections.

Symptoms. The onset of asbestosis, as of silicosis, is slow although symptoms are apt to be somewhat more marked than in silicosis. In silicosis there may be marked x-ray findings with little complaint of symptoms, whereas the opposite is likely to be the case in asbestosis. As in silicosis, dyspnea is the cardinal symptom. Anorexia occurs frequently in advanced stages, and cyanosis and clubbing of the fingers are apt to appear with greater constancy in asbestosis.

Pathology. The fibrosis in asbestosis is diffuse and tends to predominate in the basal portions of the lungs in contrast to the generalized nodular fibrosis of silicosis

[73] J. W. G. Hannon, *Trans. Can. Inst. Mining Met.*, **48**, 180 (1944).
[74] L. U. Gardner, M. Dworski, and A. B. Delahant, *J. Ind. Hyg. Toxicol.*, **26**, 211 (1944).
[75] A. J. Lanza, *J. Am. Med. Assoc.*, **106**, 368 (1936).
[76] A. J. Lanza, W. J. McConnell, and J. W. Fehnel, *U.S. Pub. Health Repts.*, **50**, 1 (1935).

with a predominance in the upper portions of the lung. Bronchiectasis and bronchiolectasis are frequent, especially in the more fibrous portions. Whereas the proliferation of fibrous tissue is caused by chemical action in silica exposure, it is induced by mechanical action in asbestos exposures. Gardner[77] found that the fibrosis-producing character of asbestos could be almost eliminated by grinding the fibers so that no particles more than 2 μ in length were present. As previously mentioned, asbestos is classified among the inert dusts.

Microscopic Anatomy. Johnstone[78] described the microscopic appearance of the lungs somewhat as follows: in the early phases of the disease there is thickening of the alveolar septa which results from fibroblastic proliferation. The alveolar spaces contain numerous phagocytes. With progression of the disease fibrosis becomes more marked; the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few alveoli that remain in the area of fibrosis are lined with low cuboidal epithelium giving them an almost glandular appearance.

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures described first by McDonald.[79] These bodies are 20 to 100 μ in length and are bulbous on one or both ends so that they appear club- or dumbbell-shaped. They are brownish in color, do not stain, and give a Prussian-blue reaction for iron. Simson[80] has produced these bodies in guinea pigs by experimental exposure to atmosphere containing asbestos. Lynch[81] concludes that these "curious bodies" signify exposure to asbestos dust but do not necessarily indicate asbestosis.

X-Ray Examination. For an excellent review of roentgenographic findings in both silicosis and asbestosis the reader might well refer to Pendergrass.[82] Characteristic differences in the roentgenographic findings in silicosis and asbestosis are tabulated below:

Asbestosis	Silicosis
Fibrosis diffuse (film may have ground glass appearance from pleural involvement).	Fibrosis nodular.
Findings may be either bilateral or unilateral.	Findings characteristically bilateral.
Lesions largely in the lower one half or two thirds of the lung fields.	Lesions predominately in the upper two-thirds of the lung fields.
Emphysema in upper portion of lung fields.	Emphysema in lower portion of lung fields.

Control. Prevention of asbestosis depends entirely upon preventing exposure to concentrations of dust sufficiently high to produce the characteristic reaction.

[77] L. U. Gardner, *Ind. Med.*, **9**, 45 (1940).
[78] R. T. Johnstone, *Occupational Diseases*. Saunders, Philadelphia, 1942.
[79] S. McDonald, *Brit. Med. J.*, **2**, 1025 (1927).
[80] F. W. Simson, *Brit. Med. J.*, **1**, 885 (1928).
[81] K. M. Lynch, *J. Am. Med. Assoc.*, **109**, 1947 (1936).
[82] E. P. Pendergrass, "Roentgen-Ray Diagnosis in Silicosis and Asbestosis," in A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

Dreessen, DallaValle, Edwards, Miller, and Sayers[83] have found evidence to indicate that 5,000,000 particles per cubic foot of air is a satisfactory figure for the maximum permissible atmospheric concentration of asbestos to which workers may be exposed. Definite permissible standards, however, have not been established.

VI. Dust Causing Minimal Fibrosis or No Fibrosis

Gardner[77] has pointed out that any known inorganic dust other than free silica and the asbestos silicates may produce nonspecific dust reactions, as described on page 486. X-ray markings may be found in other conditions such as chronic infection and heart disease. No interference with pulmonary function or disability is produced by dust of this type and there is no influence on susceptibility to tuberculosis.

A. SILICATES

McCord,[84] from a number of sources, has compiled a list of the commonly used silicates, which is presented here:

Olivine—a magnesium silicate widely present in all basic rocks. Many varieties of this stone are known.

Calcium silicate—nonexistent in nature, but a common product of industry; may be found at blast furnaces and in production of cement and hydrated limes. Other silicates of calcium are known.

Willemite—a zinc silicate (rare).

Sodium silicate (*meta*)—the well-known water-soluble silicate, commonly called water glass. This is widely used in industry, notably as a filler in soaps. This is the only crystalline silicate of sodium, the others being amorphous glass.

Tremolite—a magnesium-calcium silicate.

Asbestos—tremolite, actinolite, chrysotile, or amianthus, all of which are essentially magnesium silicates.

Jade—another form of magnesium-calcium silicate.

Crocidolite—"blue asbestos." It is a sodium iron silicate.

Talc—a hydrated magnesium silicate with extensive industrial application.

Soapstone—a form of talc.

Agalite—a variety of talc resembling asbestos. It is used in the coating of paper, and as a paper filler.

Meerschaum—also called sepiolite. It is closely akin to talc.

Serpentine—hydrated silicate of magnesium. It is a common building stone.

Sillimanite—aluminum-containing silicate. The source of many stone tools of the stone age

Topaz—a semiprecious stone of silicate origin. Chemically it is an aluminum fluosilicate.

Fuller's earth—a hydrated silica-aluminum compound, associated with ferric oxide.

Kaolin (*kaolinite*)—many related silicates of mixed constituency.

Clays—hydrated aluminum silicates containing iron. Titanium, quartz and mica, are likely to be present in clays.

[83] W. C. Dreessen, J. M. DallaValle, T. I. Edwards, J. W. Miller, and R. R. Sayers, *U.S. Pub. Health Bull.* No. *241* (1938).

[84] C. P. McCord, *Ind. Med.*, 2, 4 (1933).

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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."

Classification.—On the basis of the structural changes induced in the lung and the type of dust, the pneumoconioses or pulmonary fibroses may be classified as follows (compare reference 43):

1. *Simple benign pneumoconiosis,* which is virtually a deposition of dust in the pulmonary tissues usually accompanied by pigmentation, and includes, for example, anthracosis induced by coal dust and siderosis induced by iron. It does not incapacitate and roentgenologically shows as a maximum change only an exaggeration of linear pulmonic markings and is of clinical interest only by way of differential diagnosis. Such cases should never be diagnosed as silicosis.
2. *Silicosis,* which is a pathologic pulmonary reaction due to free silica; for example, sandblasters' silicosis. This type of pneumoconiosis results in a classical nodular fibrosis of both lungs, demonstrable both in the X-ray and at post mortem. Massive fibrosis, varying degrees of pigmentation, atelectasis, emphysema, fibrous pleurisy, bronchitis, cavitation, and pneumothorax are among the concomitant pathologic changes.[44]
3. *Mixed forms,* that is, silicosis resulting from the inhalation of mixtures of varying amounts of free silica and more inert pneumoconiogenic dust constituents, for example, anthracosilicosis and siderosilicosis. Most cases of silicosis undoubtedly come under this classification.
4. *Asbestosis,* a characteristic diffuse, interstitial fibrosis of the lungs induced by fibrous minerals which, according to animal experimentation, is due to the mechanical action of the asbestos, producing a ground-glass appearance on the X-ray. It may cause disability and a few fatal cases have been recorded.

Etiology.—Silicosis is incurable and large numbers of workmen are potentially exposed to conditions favoring the development of the disease. The majority of cases of this chronic pulmonary disease occur among workers engaged in mining,[45] quarrying, ceramics industry, tunnel construction, sandblasting, and foundry work.

Inhalation of siliceous dust almost invariably results in sili-

Symptoms and signs do, however, assist in the determination of disability. Among the symptoms and signs of importance are shortness of breath, particularly upon exercise; cough, usually dry; chest pain, varying from a feeling of tightness in the chest to the sharp, excruciating pain typical of pleurisy; hemoptysis; general complaints such as digestive disturbances, insomnia, and dizziness; decrease in chest expansion; prolonged expiration, especially in association with emphysema; altered breath sounds; rales; and the presence of areas of increased density in the lungs. Infection may complicate the picture at any time and is usually manifested by pleural pain, fever, night sweats, weight loss, aggravation of dyspnea, anorexia, weakness, and a cough producing large amounts of blood-tinged sputum.

Diagnosis.—In establishing a diagnosis of one of the pneumoconioses, all of the following three factors should be considered.*

1. An occupational history which reveals definite prolonged exposure to siliceous dust or asbestos dust.
2. Symptoms and physical signs which furnish valuable information in (*a*) gauging the extent to which pneumoconiosis has progressed, (*b*) showing the degree of disability, and (*c*) excluding other diseases.
3. X-ray findings, which, if classified on the basis of the system recommended by the U. S. Public Health Service, show bilateral ground-glass, nodular, or a more advanced type of lung-field marking.

So long as the chest roentgenograms show predominantly linear pulmonic markings, silicosis or modified silicosis need not be given serious consideration. It is when the shadows in the lung field assume a ground-glass, granular or nodular appearance that they become specific and assume more diagnostic characteristics. Using Irvine and Steuart's analogy,[48] the usual linear pulmonic markings are likened to the branches of a tree and the granular, stippled, or nodular lung-field markings simulate the leaves. Complete foliation then means silicosis. Viewed stereoscopically, the films at this stage will show fine nodulation. Some writers describe a related change in the lung-field appearance as reticulation.[49] Although not definitely determined, this type of lung-field marking may result from the inhalation of mixed dusts. Later stages will show massive areas of fibrosis, and if infection is present the X-ray shadows tend to be asymmetrical.

Asbestosis is exceptional from the standpoint that the pul-

* In the field of forensic medicine it has been frequently demonstrated that autopsy studies will yield essential information.

monary fibrosis is caused by dust containing little if any free silica. The asbestotic patient will usually show clinical symptoms and signs which are out of proportion to the apparently small amount of pulmonary involvement shown by the chest roentgenogram. Large massive shadows are very rarely seen in the chest roentgenogram except in the presence of a complicating infection. On the contrary, the diffuse interstitial fibrosis is manifested by a ground-glass appearance, frequently with fine pinpoint stippling of the middle and lower lung fields. It progresses to terminal diffuse fibrosis usually without nodular predominance in about twenty years.[50] A shaggy appearing cardiac silhouette due to involvement of superimposed lung structures and pleuropericarditis is not infrequent. Asbestosis bodies may be demonstrated in the sputum or the lung tissue.

Medical Control Measures.—All applicants for employment in dusty trades should be examined by X-ray. Periodic medical examinations at intervals of one year to possibly three years, including X-ray study of the chest, should be made of all workers in dusty trades in order to detect evidence of active pulmonary tuberculosis and early silicotic changes. The length of the interval between examinations depends mainly on the degree of hazard and the prevalence of endemic tuberculosis.

Clinical study of patients suspected of having pulmonary tuberculosis should be made to determine the dynamic status of such complication. No worker should be rejected on preemployment examination or removed from work, which he is accustomed to perform, merely because of a diagnosis of simple silicosis, but rather the atmospheric dust in which he works should be brought within safe limits. The worker whose first roentgenogram shows healed primary tuberculosis should not be denied employment in a dusty trade on this account alone. If the worker has minimal, arrested, or healed reinfection tuberculosis, he should be allowed to continue his work but should be observed with the same precautions as a man with simple silicosis. Close medical supervision is recommended for all silicotic workers in order to control or prevent serious complications of the common respiratory infections.

Benzene (Benzol) Poisoning

Benzene (benzol) is an excellent solvent for gums, resins, fats, and oils, and as such has found many industrial applications. It is used in the manufacture of rubber, rubber goods,