

In 1556, in *De re metallica*, Agricola¹⁷ stated:

On the other hand, some mines are so dry that they are entirely devoid of water, and this dryness causes the workman even greater harm for the dust which is stirred and beaten up by digging penetrates into the windpipe and lungs and produces difficulty in breathing.

The best of the early writings on silicosis is that of Ramazzini,¹⁸ translated in 1705 in *A Treatise of the Diseases of Tradesmen*, in which he stated:

For in hewing marble or stones out of the rock, in polishing and cutting them, they oftentimes suck in by inspiration the sharp, rough and cornered small splinters or particles that fly off; so that they are usually troubled with a cough, and some of them turn asthmatic and consumptive. . . . And in dissecting the corps of such artificers, the lungs have been found stuffed with little stones. Diemerbroeck gives a curious relation of several stone cutters that dyed asthmatic, and were opened by him; in whose lungs he found such heaps of sand that in running the knife through the pulmonary vesicles he thought he was cutting some sand body. He adds that he was informed by a master stone cutter that in cutting stones there rises such a subtle dust, as is able to penetrate through ox bladders hung in the shop. . . . And this very dust he took to be the cause of the death of many unwary workmen.

In recent years the contributions to knowledge of silicosis have been sufficiently substantial, numerous, and varied to make this perhaps the most widely discussed occupational disease. Silicosis is of particular interest to students of preventive medicine not only because of its general distribution in industry but more particularly because of its tendency to increase susceptibility to tuberculosis—a characteristic inherent in the disease. The limited space of this chapter does not permit reference to many sound contributions which have led to our present understanding of dust disease. An exceedingly interesting and somewhat detailed review of the history of diseases caused by dust may be found in the work of Lanza.¹⁹

2. Exposure to Silica in Industry

Since siliceous material makes up the bulk of the earth's crust, it is not surprising to find silica exposures in industry, where raw materials from the earth are used to make many of the commodities necessary to our daily lives. Rocks and minerals are often intimately associated with free silica and it is obvious that those occupations concerned with mining, rock grinding, or drilling may constitute silicosis hazards, as well as occupations concerned with the processing and industrial use of siliceous products.

According to Knopf²⁰ the most common forms of free silica used industrially are massive crystalline quartz, quartzite, sandstone, flint, tripoli, diatomaceous earths and silica sand. Table 1, from Ladoo,²¹ illustrates the great variety of uses to which silica is put in industry and indicates the kind of silica adapted to each purpose.

¹⁷ B. Ramazzini, *A Treatise of the Diseases of Tradesmen*, English trans., 1705.

¹⁸ A. J. Lanza, ed., *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

¹⁹ A. Knopf, *U. S. Pub. Health Bull.* No. 187, 1929.

²⁰ R. B. Ladoo, *Silica, Nonmetallic Minerals*. McGraw-Hill, New York, 1925, p. 525.

3. Etiology of Silicosis

Silicosis has been defined by the Committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association²² as:

A disease due to breathing air containing silica (SiO_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.

The definition adopted at the International Silicosis Conference in 1930 and reaffirmed at the 1939 conference is generally accepted and is somewhat simpler than the above. It is as follows:

Silicosis is a pathological condition of the lungs due to inhalation of silicon dioxide. It can be produced experimentally in animals.

Lanza²³ in "Etiology of Silicosis," as printed in the National Silicosis Conference Report on Medical Control, states:

Simple or uncomplicated silicosis is a chronic, fibrotic disease of the lungs, due to the massive invasion of the pulmonary tissue by silica (SiO_2), inhaled in the form of dust. In its early stages silicosis may be symptom free; in its later stages shortness of breath, decreased chest expansion, lessened capacity for work, may—one or more—be present, together with an increased susceptibility to tuberculosis. The disease presents the characteristic x-ray appearance of nodulation, without which a clinical diagnosis may not be made.

The production of silicosis depends upon the following factors: the composition of inhaled dust, the number of particles of inhaled dust, the size of the particles of inhaled dust, the length of time during which particles are inhaled, and individual susceptibility.

a. *Composition of Dust in Relation to Production of Fibrosis.* The fact that silicosis is caused by free silica (SiO_2) is attested by the great mass of clinical experience as well as by experimental observations. Gye and Kettle²⁴ have shown that silica in solution, or in noncrystalline form, stimulates the proliferation of fibroblasts in tissues. Gardner²⁵ produced a reaction in the lungs of animals in a period as short as two years by exposure to extremely high concentrations of dust. Miller and Sayers²⁶ developed an intraperitoneal injection technique for testing the tissue response to dust, and their method has been used extensively by the United States Public Health Service. On the basis of this procedure it has been found that all dusts behave in one of three ways in the body tissues; they may disappear (be absorbed), cause cellular proliferation, or remain inert in the tissues.

²² R. R. Sayers, *Am. Pub. Health Assoc. Yearbook*, 1932-33.

²³ A. J. Lanza, Report on medical control, National Silicosis Conference, *U. S. Dept. Labor, Div. Labor Standards, Bull.* No. 21, Pt. 1 (1938).

²⁴ W. E. Gye and E. H. Kettle, *Brit. J. Exptl. Pathol.*, 3, 241 (1922).

²⁵ L. U. Gardner, in A. J. Lanza, ed., *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

²⁶ J. W. Miller and R. R. Sayers, *U. S. Pub. Health Repts.*, 56, 264 (1941), Reprint 2234.