Section I
Chapter 2

HISTORY OF SILICOSIS

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I. INTRODUCTION

Silicosis is a debilitating lung disease caused by the inhalation of crystalline silica. Despite several decades of intensive research and effective (frequently unenforced) environmental dust control measures, silicosis, a disease of historical importance, continues to be a problem in our workplaces. It is important to emphasize and recapitulate the history of silicosis because in the past it was believed that the disease was a reflection of an individual's peculiar, social, personal, hereditary, and economic circumstances. The disease was considered to occur as a result of the special relationship between an individual and a complex, highly peculiarized environment. In the past, silicosis and other pneumoconioses were diseases of the poor, of slaves, or of prisoners forced to work. The rampant tuberculosis prevalent in the 18th and 19th centuries made a significant impact on the concomitant increased mortality of people with silicosis. Roentgenographic examination of the chest was not available as a diagnostic tool until 1885, i.e., long after the recognition of silicosis.

II. PREHISTORIC EVIDENCE

Silica dust as a cause of pulmonary disease (silicosis) is one of civilization's first examples of understanding the etiology of an occupational lung disease. It was certainly the oldest pneumoconiosis recognized to be associated with specific occupations such as stone cutting, quarrying, and mining. Silicosis was described to have existed in neolithic men, who were engaged in shaping flints for arrow heads, spearheads, and other weapons of defense.[1] Anthracosis has been seen in the lungs of Egyptian mummies.[2]

Mining activities which may have caused silicosis in workers were started in China, India, and along the Nile in Egypt between 4000 and 3000 B.C.[3] Hippocrates described clinical lung disease in miners (pneumoconiosis) in approximately 400 B.C.[3] He described the metal digger as a man who breathes with difficulty, which was suggestive of the presence of pneumoconiosis among the miners.

Pliny (A.D. 23–79) advocated the use of protective masks for miners to prevent the inhalation of "fatal dust" produced in the mines. Pliny's description of protective devices to avoid dust inhalation clearly points to the dangers of dust-induced lung disease known to the Romans during these early times.[4] Galen (A.D. 131–201) was the first to describe the pulmonary disease symptoms of silicosis which developed in gypsum miners.[3]

III. EUROPEAN EXPERIENCE

In 1556, Georgius Agricola[5] published a book entitled *De Re Metallica.* In it, he described mining, metallurgy, and the lung diseases of miners in Bohemia. Agricola stated that the "mines produced asthma, and some mines are so dry that they are entirely devoid of water, and this dryness causes workmen 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 and disease. If the dust was corrosive, it ulcerated the lungs and caused consumption." Agricola's statements concerning lung disease in miners are considered as one of the earliest and best, if not the first, descriptions of this dust-induced pulmonary clinical condition and its pathologic effect on lungs.

In 1567, Paracelsus, a Swiss physician and alchemist, wrote the first monograph on the diseases of miners entitled "Von der Bergsucht und anderen Bergkrankheiten." In this monograph, the mineralogy of the mines as well as the pathology and etiology of the miners' diseases are discussed in detail. He attributed the origin of miners' lung diseases to the mine air. This monograph greatly influenced the study of occupational lung diseases.[3]

In the Carpathian mines of Poland, Czechoslovakia, and Rumania, the mining tools were called "widow-makers" by the miners.[3,5] In the mining communities, records exist of women who married seven or more husbands, "all of whom this terrible consumption has carried away."[3] Van Diemerbrock, in 1649, described the lungs of stone cutters in Netherlands (who had died of "asthma") as having "piles of sand" in the lungs at autopsy.[6]

Bernardino Ramazzini's book *De Morbis Artficum Diatriba* (Padua, 1700) described the diseases of stone cutters and many other occupations.[7] He described in detail the special hazards involved in each occupation, including dust diseases and their related pulmonary lesions. He described lungs of stone cutters that were "found to be stuffed with small stones."[7] The stone cutters, sculptors, quarrymen, and marble cutters were described as "breathing the rough, sharp, jagged splinters that glance off while making statues and other objects and hence they are usually troubled with cough, and some of them contract asthmatic affections and become consumptive."[7]

In Sheffield, England, young needlepointers and cutlery workers suffered high mortality due to silicosis in the 1790s.[8] During this time flint was powdered dry and used as grinding powder in the manufacture of grindstones and in cutlery polishing. Thomas Benson was first granted patents for wet grinding of flint in 1726 and again in 1732, which reduced the dust levels in grinding operations in England. This wet grinding of flint greatly reduced the incidence of silicosis and the high rate of grindstone workers dying at the early ages of 28–32, as evident from the writings of Arnold Knight.[3]

Charles Thackrah was credited as the first to compare, on an epidemiologic basis, the life expectancy of British workers in different trades and occupations.[9] He correlated the mortality of workers with dust exposure. His conclusion was that workers in sandstone quarries died at an early age (less than 40 years) because of the large dust clouds produced at work sites. He found no evidence of lung disease among brick and limestone workers because these mines had vertical fissures which allowed water to percolate through their roofs, thus decreasing the dust exposure. He described the benefits of this process: "particles are laid as they are formed, by the continuous oozing, dropping and splashing of the insinuated water." This concept of continuously wetting mining areas is considered the "basis of the modern practice of infusing the working face in a mine with water."[9]

T.B. Peacock was the first to distinguish the differences between miners' phthisis and pulmonary tuberculosis.[10] Phthisis, a descriptive term coined by the Greeks, meaning "to waste away," was also called consumption. The clinical and pathological descriptions of autopsied stoneworkers by E.H. Greenhow[11] include deposits and drifts of sand in the lung tissue and its isolation for quantitative measurements. He was able to isolate about 30 gm of so-called sand from the lungs of one stone cutter. Greenhow is credited as the first to use polarized light microscopy to demonstrate silica particles in the lungs.[11]

In 1866, Friedrich von Zenker studied the pathology of lung disease caused by the inhalation of iron dust.[12] He described the disease as "iron lung: sclerosis pulmonum," and stated that iron dust was the "injurious agent which is the essential cause of the entire disease."[12] To group all the diseases of identical cause, he coined the term "pneumonokoniosis." This was derived from the term "konis," meaning dust. The term "silicosis," (Latin, silex, flint) was originally used by Visconti in Italy in 1870.[13]

Allison and Hugh Miller in 1869 observed that stone drillers appear to suffer more from the rapid onset of the disease silicosis than other miners.[14] From their studies it was evident that hard-stone work seriously affected the health of workers, while the soft-stone workers seem to have a less severe lung disease.

Silicosis was recognized as a distinct disease entity by the late 19th century in Europe. The introduction of the pneumatic hammer drill in 1897 by John Leyner generated large amounts of fine dust and contributed to an abnormal increase in the incidence and mortality of silicosis.[3,8] In the operation of the pneumatic hammer drill, air is blown through the drill to remove rock particles. This produced excessive amounts of dust, and once again the term "widow maker" was ascribed to a mining tool due to the short life span of the drillers. By 1898, water was commingled with air to suppress the dust in rock cutting using pneumatic drills.[8] Sandblasting was introduced in 1904 as a specialized method for the cleaning, polishing, and abrasion of surfaces.[8] This has resulted in countless numbers of silicosis cases.

In 1902, John Haldane reported that "primary injury of the lungs is solely caused by the inhalation of stone dust and that this injury also predisposes enormously to tuberculosis."[15] Silicosis, as an industrial hazard in sandstone workers of South Africa, was established by Haldane et al. in 1904.[15] In Witwatersrand, South Africa about one sixth of the rock drillers were found to have died from silicosis prior to the Boer War. In 1914, Haldane presented significant evidence on the dangerous effect of silica in the lungs before a Royal Commission, which greatly influenced the passing of the Workman's Compensation Act for silicosis in Great Britain.[16,17] After chest x-rays became increasingly popular in the medical diagnosis of lung diseases, Sutherland and Bryson found radiographic evidence of silicosis in 25% of the sandstone workers examined in 1929.[18] Sutherland et al., in a later study of granite workers, found x-ray changes consistent with silicosis in approximately 54% of the cases.[19]

IV. NORTH AMERICAN EXPERIENCE

Although several cases of silicosis were reported in North American literature during the 1800s,[20–22] awareness of the health importance of silica dust was apparently very minimal until the early 19th century. Case reports of silicosis were found occasionally in physicians' reports of lung diseases with suspected occupational involvement. The first case report on a stove foundry worker in Poughkeepsie, NY, was considered a true documentation of silicosis.[20] In 1887, another report on 34 cutlery factory workers described a chronic airway disease and stated that 23 workers died of silicosis.[21] Around 1900, another report appeared in Utah, which described 30 men dying after only 1 to 2 years of dust exposure from crushing quartz ore in a gold assaying mill in Nevada.[22]

Silicosis emerged as a major industrial scourge to the American workers with the industrial revolution and technological innovations. David Rosener and Gerald Markowitz described the awareness of this disease and its political, social, and economic impact in a book, *Deadly Dust, Silicosis and the Politics of Occupational Disease in Twentieth-century America*.[23] The introduction of power equipment in the mines, mills, factories, and other work places significantly increased a widespread hazard because of the generation of greater quantities of fine dust in confined spaces of worksites. This led to the re-emergence of silicosis as a major occupational threat in North America with the complicating companion disease, tuberculosis.[24]

Dust produced at work sites was well recognized as a problem for hard-rock miners, sandblasters, potters, foundry workers, and cutters. Nonetheless, silicosis was not considered a major health hazard in America until the U.S. Bureau of Mines and the U.S. Public Health Service, in the early 19th century, documented the widespread occurrence of silicosis in various worker groups.[25–28] The first major effort in this regard was undertaken from 1913 to 1915 in the lead and zinc mining industry of Joplin, MO, by these two agencies.[25] In a study of 720 miners, 46% were found to have silicosis, with another 14% exhibiting silicosis complicated by tuberculosis. In a report to the "Committee on Mortality from Tuberculosis in Dusty Trades," as quoted by Hosey et al.,[29] it is stated that 93% of the workers examined in the Vermont granite industry in 1920 were affected with silicosis. This was followed by the studies of 427 granite workers in Barre, VT, from 1924 to 1926 by local physicians and the U.S. Public Health Service. In these studies, all Vermont granite workers exposed to high concentrations of dust were found to have silicosis, tuberculosis, or silico-tuberculosis.[24,26] In 1928, Smith reported silicosis in 57% of the 208 workers employed in rock tunneling in New York.[27] There were several other studies conducted in metal grinding, cement processing, porcelain enameling, sandstone grinding, and abrasive grinding in which evidence of silicosis was documented as high as 67%, which provided incriminating evidence of a widespread silicosis hazard and the dire need for dust control.[30]

V. SIGNIFICANT EVENTS IN U.S. HISTORY OF SILICOSIS

In the 1930s, public health awareness grew and silicosis was well recognized as a serious health hazard in North America. This public health awareness and the economic depression precipitated a large number of lawsuits based on both legitimate and spurious claims. Sappington[31] estimated that approximately $100 million in damage suits were outstanding for settlement in 1933. This public awareness was further galvanized by the Gauley Bridge disaster in West Virginia, which initiated a congressional hearing in 1936. This incident, described in detail by Martin Cherniack in his book *The Hawk's Nest Incident,* was America's worst industrial disaster in silicosis. Within two years, 476 workers died of acute silicosis, while an additional 1500 suffered impairment from lung disease they contracted while digging a rock tunnel to divert water to a hydroelectric plant from 1932 to 1934.[32,33] This disaster provided an impetus for industrial health reform in the U.S. from 1936 to 1939, several states passed compensation laws and industrial hygiene dust control standards. In the Vermont granite industry, dust concentrations were regulated to 10 million particles per cubic foot of air sampled (mppcf) from a previous average dust level of 60 mppcf.[30]

Almost 25 years after the first report of silicosis in Joplin, MO, Alice Hamilton wrote "A Mid-American Tragedy," an article on lead and zinc mining.[34] A preliminary survey of industries in 1940 by the U.S. Public Health Service established a silica exposed population of 1.5 to 2 million.[35] Cummings, at the Fourth Saranac Laboratory Symposium on Silicosis, presented estimates of about 50,000 workers having definite silicosis.[36] Uncontrolled dust exposures and silicosis incidence continued to occur in isolated industries. The implementation of dust control measures was impaired since ventilation equipment was scarce due to World War II between 1941 and 1945. As a result, inadequate dust control measures were common in several North American industrial workplaces. For example, in Georgia it was revealed that the granite industry exceeded recommended limits for silica exposure many-fold.[37]

After World War II, widespread mechanization and technological advances for new uses of crystalline silica increased the possibility of several new dusty operations. So the mounting concern of silicosis as a continuing problem led to a second Congressional hearing on silicosis as an occupational hazard in 1956.[38]

In the U.S., during 1950 to 1956, approximately 2000 deaths were recorded due to occupational pneumoconioses.[39] About 60% of these deaths were attributed to silicosis and 28% to silicosis combined with tuberculosis.[39] In a 1958 study of Vermont granite industries, Trasko reported that silicosis was a continuing problem with a substantial increase in silicosis and anthracosilicosis in spite of appreciable declines in tuberculosis since 1949.[30] Although changes in the international coding of disease were partially responsible for this increase in deaths attributed to silicosis, the steady rate at which silicosis occurred was very evident. Follow-up studies of the Vermont granite industry by the Public Health Service and the Vermont Industrial Hygiene Division documented a high degree of success achieved in controlling dust levels and a low incidence of silicosis in workers. Hosey et al.[39] documented the prevalence of silicosis in a group of 1112 workers who began employment in the industry before 1937 and compared this to 1134 workers employed after dust control measures were instituted. The prevalence of silicosis, as determined by chest radiographic survey, had decreased to 15% from a pre-1937 prevalence of 45% in workers with comparable work histories. In 1964, Ashe and Bergstrom[40] re-evaluated chest x-rays of 1478 granite workers and confirmed earlier findings of Hosey et al.[39] showing a decline in the prevalence of silicosis. Data from these studies suggest that dust control measures implemented in 1937 had successfully reduced the exposure to granite dust and decreased the rate of silicosis in workers exposed to granite dust for up to 26 years.

VI. CURRENT STATUS OF SILICOSIS IN U.S.

In 1937, one of the first recommendations for upper limits of exposure to quartz-containing industrial dusts was made by Russell for the Vermont granite industry. Russell based his recommendation on the

TABLE 1
Significant Events in the History of Silicosis

Hippocrates	Described clinical Pneumoconiosis	400 B.C.
Pliny	Described "Fatal Dust"	70 A.D.
Agricola	Described lung disease	1556
Ramazzini	Described dust lung diseases and lesions	1700
Benson	Granted patent for wet grinding	1726
Knight	Described silicosis	1830
Greenhow	Demonstrated silica (polarized light) in lungs	1865
Visconti	Used the term silicosis	1885
Canedy	Case reports of silicosis in U.S.	1887
Leyner	Introduced the pneumatic drill	1897
Haldane	Worker's Compensation Act, U.K.	1918
West Virginia	Gauley Bridge Disaster	1934
U.S. Congress	Gauley Bridge Disaster Hearings	1936
Vermont	Granite Industry Set Dust Limits	1937
U.S. Congress	Mine Safety Hearings	1940
U.S. Congress	Mine Safety Hearings	1956
U.S. Congress	Occupational Safety and Health Act	1970
NIOSH	Recommended 50 μg/m^3	1974
IARC	Implication of silica as a carcinogen	1987

prevalence of silicosis reported earlier in different dusty exposure groups.[29] Understanding of the dose-response relationships has led to the formulation of exposure limits in occupational workplaces. From 1971 to the present, crystalline silica standards have been under continual re-evaluation, and decremental changes in permissible exposure limits at work sites were set by the Occupational Safety and Health Administration (OSHA). The current OSHA permissible exposure limit (PEL) for respirable crystalline silica is 100 μg/m^3 for an 8-hour work exposure. The National Institute for Occupational Safety and Health (NIOSH), charged with developing scientific information on the types and extent of exposures to occupational hazards, has recommended an exposure limit of 50 μg/m^3 for up to 10 h/d during a 40-hour work week.[41]

Despite the existence of remarkable engineering controls and protective devices, cases of silicosis continue to occur. In 1983 NIOSH estimated that approximately 3.2 million workers at 238,000 worksites were potentially exposed to crystalline silica and a significant number of workers in several industries had elevated rates of silicosis. The U.S. Department of Labor estimates that nearly 60,000 workers are at risk of developing some degree of silicosis. If these estimates are considered conservative and valid, the outlook for silicosis as a continuing threat is substantial and burdensome to health care management. NIOSH published several intelligence bulletins in recent years documenting the occurrence of acute silicosis in silica flour mills, sandblasting operations, surface coal mine drilling, and railway bed workers.[42–45] A 1992 NIOSH ALERT described 23 cases of silicosis from exposure to rock drilling.[46] In addition to this, the relationship between silica exposure, silicosis, and the development of lung cancer has received increased attention in recent years.[47,48]

In conclusion, silicosis as an occupational disease has long been recognized (Table 1). In spite of major advances in our understanding of its etiology and the importance of dust control, major pockets of overexposure and disease still exist.

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