Section I
Chapter 4

CLINICAL FEATURES OF SILICOSIS

Daniel E. Banks

CONTENTS

I. INTRODUCTION

Silicosis was first reported by the ancient Greeks and is as old as the history of man. The prevalence of this illness apparently peaked in the last half of the 19th century and the early part of this century. The aggressive industrialization of America, the development of powered tools which generated massive amounts of respirable size dust particles, the failure to recognize a latency period between exposure and development of disease, an inadequate (or perhaps careless) understanding of the relationship between dust exposure and disease, and respiratory protection proven to be inadequate has resulted in a series of epidemics of silicosis which have caused the premature death of many workers.[1–11]

Since the recognition of the dose-relatedness of this disease and the initiation of and compliance with effective dust control measures, the prevalence of silicosis has decreased dramatically.[12–14] The current U.S. Occupational Safety and Health Administration permissible exposure limit (PEL) for respirable free silica is 100 $\mu g/m^3$.[15] In some mines and quarries, compliance with this level has been achieved by instituting wetting techniques and improving ventilation; yet, in industries such as sandblasting, silica mining, and rock drilling, workers remain potentially exposed to many times the acceptable limit of respirable free silica (Table 1). In the U.S., particles much less toxic than silica are available for blasting, and since 1974, the National Institute for Occupational Safety and Health (NIOSH) has recommended substitution of non-silica containing particles in abrasive blasting.[16,17] Where silica sand is used in blasting, epidemics of very aggressive silicosis have recurred, even in developed countries where safe exposure limits are defined and respiratory protection is available[18] (Figure 1).

0-8493-4709-2/96/$0.00+$.50

TABLE 1
Major Industries with Silica Exposure

Occupation	Exposure
Mining	Silica contaminants in mined material.
Milling	Dry, finely ground silica (silica flour) is used for abrasives and filler.
Quarrying and stone work	Slate, granite, and sandstone exposures.
Foundry work	Silica is used as a mold; fettling and chipping produce fine particles but are necessary to make a better molded product.
Sandblasting	Ship building, oil rig maintenance, and preparing steel for painting are major sources of exposure.
Pottery making	Crushing flint and fettling are major sources of exposure.
Glassmaking	Sand is used to polish and as an abrasive.
Boiler work	Cleaning boilers may result in exposure to fine particulate quartz dust derived from coal and refractory brick dust.

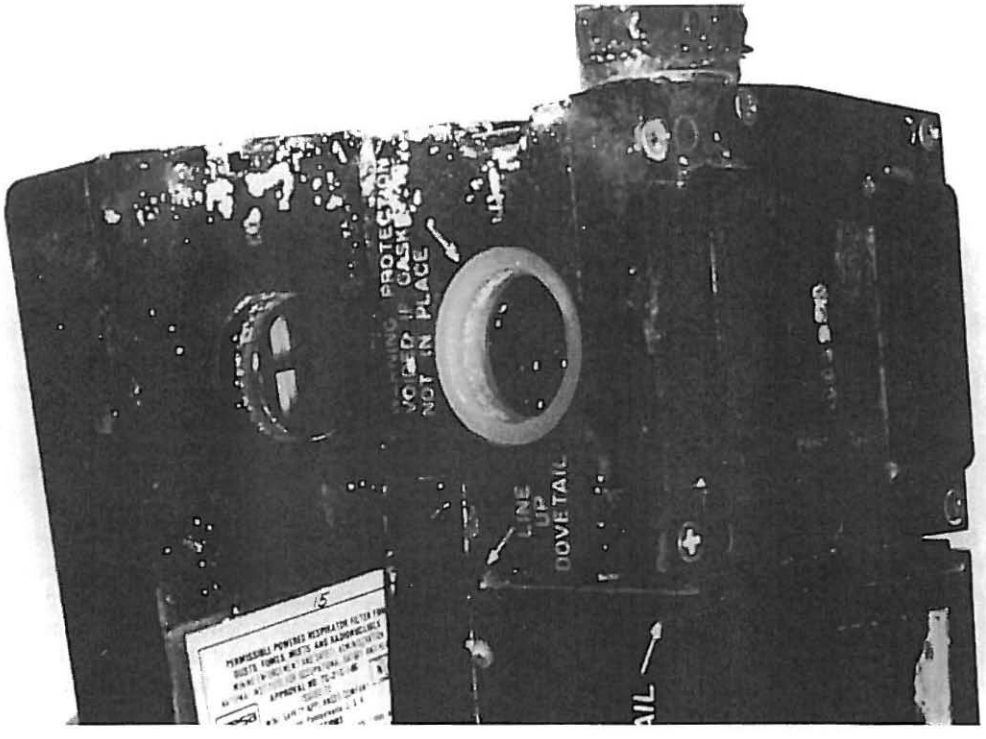


FIGURE 1 This respiratory protective device shows fine silica dust within the apparatus. Technologically sophisticated attempts such as this to protect the worker from excessive dust exposure are inadequate when faced with very fine particles.

II. DETERMINANTS OF SILICOSIS

Silicon dioxide, or silica, is the earth's most abundant mineral. Silicosis, the pulmonary disease caused by the inhalation of silica particles, occurs in workers consistently exposed to respirable size (0.5–10.0 microns in diameter) silica particles at levels exceeding those recognized to be safe. Although host factors such as genetics, smoking, and underlying disease may play roles in the development of silicosis, the primary determinants of whether a worker develops disease are the silica dose in ambient air (including the percentage of free crystalline silica in the dust particles), the duration of dust exposure, whether the silica is crystalline, and the particle size.

For example, hard-rock mining exposures appear to affect smokers and nonsmokers differently.[19,20] In never-smokers with dust exposure, a restrictive effect may occur, as reflected by decreasing total lung capacity

and residual volume. Yet, in smokers with this same dust exposure, the mean effect was increasing total lung capacity and residual volume and a decreasing diffusion capacity when adjusted for alveolar volume. Furthermore, greater airflow limitation occurred in dust-exposed compared to nondust-exposed smokers.

There is little information concerning the effect of cigarette smoking on the lung function of workers with silicosis. Most studies have attempted to identify the effects of silicosis by radiographic category on lung function and have been unable to identify clear differences attributable to cigarette smoking.[21–24] Irwig and Rocks[25] compared lung function parameters in 110 pairs of silicotic and nonsilicotic miners matched for numerous parameters including smoking category. There was no significant difference in the spirometric values between the two groups, implying that dust exposure was the most important determinant of outcome in lung function.

In a more recent report, an additive effect of cigarette smoking and silica inhalation has been recognized. In a population of black South African gold miners with a very high percentage of silicosis (857/1197 miners) studied in a cross-sectional manner, 67% of the workers had bronchitis, including 45% of the nonsmokers.[26] When adjusted for smoking and other demographic parameters, there was a dose-dependent decline in FEV_1 (forced exploratory volume in 1 second), FVC (forced vital capacity), and DLCO (diffusing capacity of the lung for carbon monoxide). In this group, the effect of 25 years of dust exposure was associated with an additional decline of 8 ml per year in FEV_1, while the effect of 25 "pack years" (years smoking times packs smoked per day) of cigarette smoking resulted in an additional decline of 7 ml per year. In this report, the loss in lung function appeared dependent on silicosis category and not current dust exposure.

Only respirable size particles are deposited in the alveoli. Particles less than 1 μm in diameter are believed to be the most fibrogenic. Depending on particle size, up to 80% of the silica dust deposited in the alveoli is quickly cleared. It is the small fraction of retained particles which initiates the fibrogenic process. It is not surprising, therefore, that the duration of exposure, the amount of exposure, and the content of free crystalline silica in the dust are the most important determinants of progression of this disease.[27] Although there are numerous jobs where the risk for silicosis exists, exposures to finely milled silica in the production of silica flour (a material used as a filler or as an abrasive), sandblasting, drilling into siliceous rock (Figure 2), and grinding and chipping among foundry workers are examples of jobs where potentially life-threatening exposure to respirable size particles occurs, where the silica content of the particle is high, and where silicosis can develop unless action is taken to protect the worker.

In the Vermont granite workers' industry engineering controls have been implemented and the prevalence of silicosis nearly eradicated. The effect of silica dust (from granite grinding and chipping) exposure on this population serves as the backbone of the federal regulations for permissible silica exposure.[28–32] Investigators who have studied this industry have recorded a legacy of workers with premature death from silicosis and mycobacterial disease beginning in the early parts of this century. Earlier this century, two studies were performed on this population within a 20 year span. Each showed serious granite dust-induced lung disease with the premature death of workers employed in the granite industry.[4,5] However, beginning in the 1950s, there was evidence that institution of dust control measures had produced a safer workplace.[6,33,34] A recent landmark paper by Graham, et al., showed that the mandated current levels of permissible granite dust exposures in these workers are protective.[35] In the same year a report from England detailed aggressive silicosis in stone masons, showing how severe silicosis can be in the same industry when adequate respiratory protection is not used.[36]

III. THE EFFECT OF SILICA DUST ON LUNG FUNCTION

There are two primary respiratory risks to the worker who has silica exposure. The first is the development of simple silicosis — typically with no or little impairment — with the potential to progress to conglomerate silicosis (also known as progressive massive fibrosis) — a disease capable of inducing severe respiratory disability and a shortened life span.[37,38] In silicosis, depending on the overall dust burden and the rate of disease development, silicotic nodules, the histologic hallmark of this disease, may begin *de novo* or continue to enlarge even after exposure has ceased. For this reason, the physician's initial response to the diagnosis of silicosis should be to recommend that the worker cease silica exposure. Even if the worker ceases exposure, this does not guarantee that the disease will not progress. Attempting to lessen dyspnea is an important part of the treatment for this illness. This includes therapy with inhaled bronchodilators and theophylline preparations. There are no proven methods for halting the progression of this illness or reversing silica-induced impairment.

FIGURE 2 This is a photo of a surface coal mine drilling machine in action. After these holes are bored, explosive charges are inserted and detonated. In this way, the overburden is disturbed so that it may be removed and the underlying coal seam exposed. The risk of exposure in this occupation is not to coal but to siliceous rock overlying the coal seam. An operator is required to sit in the cab adjacent to the drilling apparatus in order to monitor drilling progress. Such work can be exceedingly dangerous because of the dust hazard. These machines generate overwhelming amounts of respirable size dust, typically with a high free crystalline silica content.

The second risk is the development of mycobacterial infection with ensuing respiratory impairment. It appears that silica exposure, even in the absence of silicosis, predisposes the worker to mycobacterial infection.[39] In some silica exposed workers, silicotic nodules are present but of an inadequate profusion to allow for a radiographic diagnosis of silicosis.[40] Yet, the mechanisms of fibrogenesis are underway and cellular and humoral features which alter immunologic mechanisms and which may also be a result of silica-induced immunologic impairment are present.[41–51] Mycobacterial infection in a silicotic worker has the very great potential to hasten the development of respiratory impairment and shorten the worker's life. Identifying changes in the chest radiograph of an individual with silicosis over a relatively short time period means superimposed mycobacterial infection until proven otherwise. A new infiltrate, coalescence of nodules in the upper lung fields, an acute chest illness, or cavitation of a pre-existing lesion are reasons for great concern and demand an aggressive search for mycobacterial organisms.

Although not studied in a systematic fashion, it is clinically reasonable to undertake yearly chest radiographs and application of a purified protein derivative (PPD) skin test in those with silicosis. If the PPD becomes positive without clinical evidence of active tuberculosis, at least a year of isoniazid therapy is indicated.[52–54] Many advocate longer, even life-long, antituberculous prophylactic therapy in this setting because of the very real possibility of extensive irreversible damage to the silicotic lung by this organism.[55]

Few reports have described the effect of silica dust inhalation on respiratory function among workers without silicosis. Among South African gold miners with silica dust exposure, Hnizdo[56] showed that cigarette smoke and dust exposure resulted in an additive loss of the FEV_1. For example, the expected 5 year loss in FEV_1 in a 50-year-old underground miner with a 60 pack year cigarette smoking history was 689 ml. In the same miner without a smoking history, the FEV_1 decline was 236 ml. In this population, the contribution of tobacco smoking was substantially greater than that of dust. Nonsmoking miners in the highest dust concentration had greater lung function than non-miners who smoked a

package of cigarettes per day. Yet, the nonsmoking silicotics group had a significantly lower FEV_1 than nonsmokers without silicosis. Furthermore, in this same group of miners, workers exposed to silica dust who smoke were at a higher risk of dying from chronic obstructive lung disease than those not exposed to silica dust.[57]

In Cowie's previously cited work,[26] underground miners with silica exposure, but without silicosis, had supranormal values for FVC, FEV_1, and DLCO. In autopsied lungs from silica-exposed miners, emphysema was exceedingly rare unless concomitant cigarette smoking had been a part of the miner's lifestyle.[58] Overall, unless silicosis is recognized, the effects of silica exposure in the work place is thought unlikely to cause clinically important lung function declines.

IV. THE DIAGNOSIS OF SILICOSIS

There are three requisites for the diagnosis of silicosis. First, the worker must provide a history of silica exposure sufficient to cause this illness. Second, the chest radiograph must show opacities consistent with silicosis. Third, no underlying illnesses should be present which mimic silicosis. The most common diseases which might mimic silicosis include a miliary distribution of mycobacterial or fungal organisms, or in an unusual circumstance, sarcoidosis. Although respiratory symptoms and lung function impairment commonly are present, neither is necessary for the diagnosis of silicosis.

The first diagnostic criterion which must be addressed by the physician, determining whether the worker's occupational silica exposure is sufficient to cause silicosis, can be difficult. This requires knowledge about the work place environment and the worker's exposures. The most important information includes knowledge regarding the length of employment, exposure measurements (if available), and a recognition of whether the worker was provided effective respiratory protection. A reasonable starting point is the view that silicosis occurs in association with industrial processes where the silica particle is made respirable size. This is the common thread among occupations where silicosis is a clearly recognized risk (e.g., sandblasting, drilling into siliceous rock, or exposure to finely milled silica [silica flour]). Without manipulation of the silica particle so that it becomes respirable size, the particle is trapped in the upper airway defenses, and silicosis does not occur. Furthermore, the adequacy of respiratory protection devices are highly variable.[7] Silicosis can certainly occur in workers even though they use personal respiratory protection. To begin with, since each respirator has a limit beyond which the dust level exceeds the protective capacity, the worker may be using a respiratory protective device which is inadequate for the amount of dust present. Second, the worker may be using the wrong type of respirator. As an example, respiratory protective devices are designed to protect for the specific exposure. If a solvent mask were used for dust protection, the effectiveness would be compromised. Third, the respiratory protection may be ill-fitting. Finally, because these protective devices are associated with an increased work of breathing, which makes work more difficult, the worker may not use effective available protection. How some workers develop silicosis (or greater or lesser extents of this illness) while employed in the same general work area may be related to the adequacy of respiratory protection. Information regarding the silica dust levels in the work place can also provide important information. However, this approach cannot be guaranteed to represent the complete picture. Cases of acute silicosis in surface coal mine drillers have been reported where the measured dust levels were within normal limits.[9] In this report, the measurements were not representative of the overwhelming dust exposures which induced very aggressive silicosis, nor were there measurements of the percentage of free crystalline silica in the measured mine dust. Therefore, although dust measurements can be helpful, understanding the conditions under which the samples were collected and having confidence that these measurements accurately represent the work place environment is essential. Sometimes this information can only be understood by going to the work place and observing how the work is performed.

When the three clinical requirements for the diagnosis of silicosis are met, additional evaluation of the worker is not necessary to make the diagnosis. On occasion, the diagnosis cannot be made clinically. In these instances, histologic examination of lung tissue is necessary. Situations when this might be necessary occur when the exposure history is uncertain or when the differential diagnosis includes a malignant tumor (sometimes considered when the coalesced lesions of progressive massive fibrosis are recognized as unilateral or asymmetric). This consideration may arise more frequently because of the interest in silica as a potential carcinogen.[59–64] Other illnesses have radiographic features which mimic silicosis. These include rheumatoid nodules[65] (referred to as Caplan's syndrome when this occurs in the

presence of pneumoconiosis), infectious processes, or sarcoidosis. Each may need to be ruled out by histologic assessment. When tissue for diagnostic analysis is required, the traditional view has been that an open lung biopsy is preferred in order to lessen the chance of complication of pneumothorax induced by a transbronchoscopic lung biopsy.[66] The increased risk for pneumothoraces may be explained by the knowledge that the upper zones are stiff while emphysematous changes are present in the lower zones. Although the small sample attained by transbronchial lung biopsy may sometimes be inadequate for diagnosis, bronchoalveolar lavage and transbronchial biopsy with energy dispersive x-ray analysis have been used together to aid in the diagnosis of silicosis. In one reported example, a worker employed as a sandblaster, the bronchoalveolar lavage fluid showed a neutrophilic alveolitis, silica was shown to be present in the macrophages by energy dispersive x-ray analysis (although no birefringent crystal particles were found), and the transbronchial biopsy showed fibrocellular nodules in the parenchyma consistent with silicosis.[67]

A. APPROACHES TO CATEGORIZING SILICOSIS

Silicosis can be categorized in two ways. Most commonly, the disease is categorized by findings on the chest radiograph.

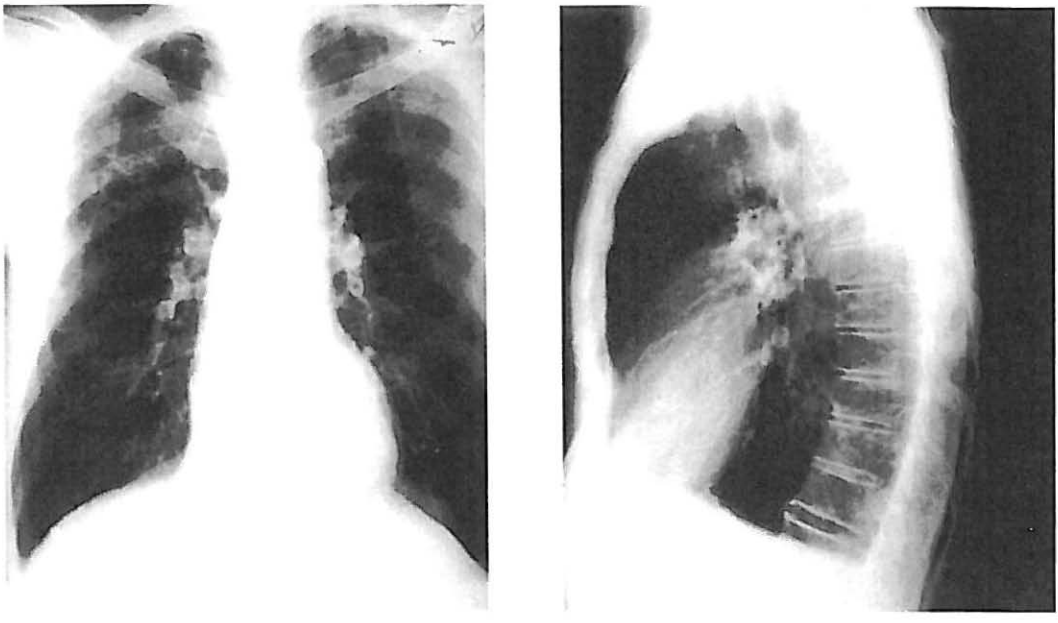
B. CLASSIC SILICOSIS

Classic silicosis is separated into simple silicosis and progressive massive fibrosis. These two presentations are radiographically different, but are grouped together under the category of classic silicosis because they are a part of the radiographic spectrum of this illness. On the chest radiograph, simple silicosis is recognized as a profusion of small (less than 10 mm in diameter) rounded opacities (nodules) predominant in the upper lung zones (for an unknown reason, retention of silica particles are favored in the upper lung zones). In some, these small opacities gradually enlarge and coalesce to form larger, usually bilateral, upper zone opacities of similar sizes (more than 10 mm in diameter) recognized as conglomerate silicosis or progressive massive fibrosis.

In some workers with relatively low silica exposure, inhaled silica is more efficiently cleared from the alveolar spaces into the regional chest lymph nodes. In these instances, the chest radiograph reveals peripheral calcification of the hilar (and sometimes mediastinal) lymph nodes with only a minimal or no background of small rounded opacities (figures 3, 4). These are described as "eggshell calcifications" and appear to be a radiographic pattern seen consistently in silicosis. Workers with such radiographic findings are without symptoms attributable to these calcified nodes. An unusual case of such mediastinal nodes eroding through the airway wall and causing tracheobronchial obstruction has recently been reported.[68] In most workers with silica exposures who develop silicosis, however, the lung cannot effectively clear dust and nodules form in the pulmonary parenchyma, sometimes in association with "eggshell calcifications" in the lymph nodes.

In addition to the radiographic features of classic silicosis described above, the other radiographic presentation occurs rarely and is referred to as acute silicosis.[69–71] This is the result of an overwhelming exposure to free crystalline silica over several years. In addition, it may also be that excessive exposure to "freshly fractured silica," material shown to have considerably more free radical oxygen species than "stale" or "old" fractured silica, is more fibrogenic.[72] In some with acute silicosis, the chest radiograph appears as a basilar alveolar filling pattern (identical to that seen in pulmonary alveolar proteinosis) without rounded opacities or lymph node calcifications. This is termed silico-proteinosis. With time, these features progress from a pattern of lower zone alveolar filling to large masses of coalesced parenchymal tissue, typically bilateral but not always symmetrical, in the mid- and lower zones. In others with very excessive silica exposures, the radiographic features are those of simple silicosis which progresses to conglomerate silicosis in a very short time frame, which is consistent with acute silicosis. The explanation for the very different radiographic response of an alveolar filling pattern vs. a very aggressive nodular silicosis in the face of an overwhelming silica exposure is not known but might reflect an important difference in an individual's pulmonary lymphatic drainage.[73]

Silicosis can also be categorized by the duration from initial exposure to the recognition of the disease. The time frames are imprecise, but this approach is useful because of its relevance to prognosis. Classic silicosis develops slowly. Usually 10 to 30 years (a working lifetime) are required from the beginning of exposure to the recognition of radiographic manifestations. In a minority, the nodules of simple silicosis coalesce to become progressive massive fibrosis. Accelerated silicosis occurs infrequently and appears radiographically as simple silicosis which develops after less than 10 years of excessive silica exposure.



FIGURES 3 AND 4 These postero-anterior and lateral chest radiographs are of a 58-year-old man who worked on the railway for 23 years. His job was to load coal onto trains powered by steam and then to load the coal by shovel into the firebox of the steam engine. He had other duties which required him to spread sand on the tracks, a feature necessary to provide adequate traction for trains travelling the mountains of Eastern Pennsylvania.

These radiographs show a minimal amount of small rounded opacities consistent with pneumoconiosis. What is most remarkable about these films are the peripheral "eggshell" calcifications outlining the hilar and mediastinal nodes. (Radiographs courtesy of Edwin J. Morgan, M.D.).

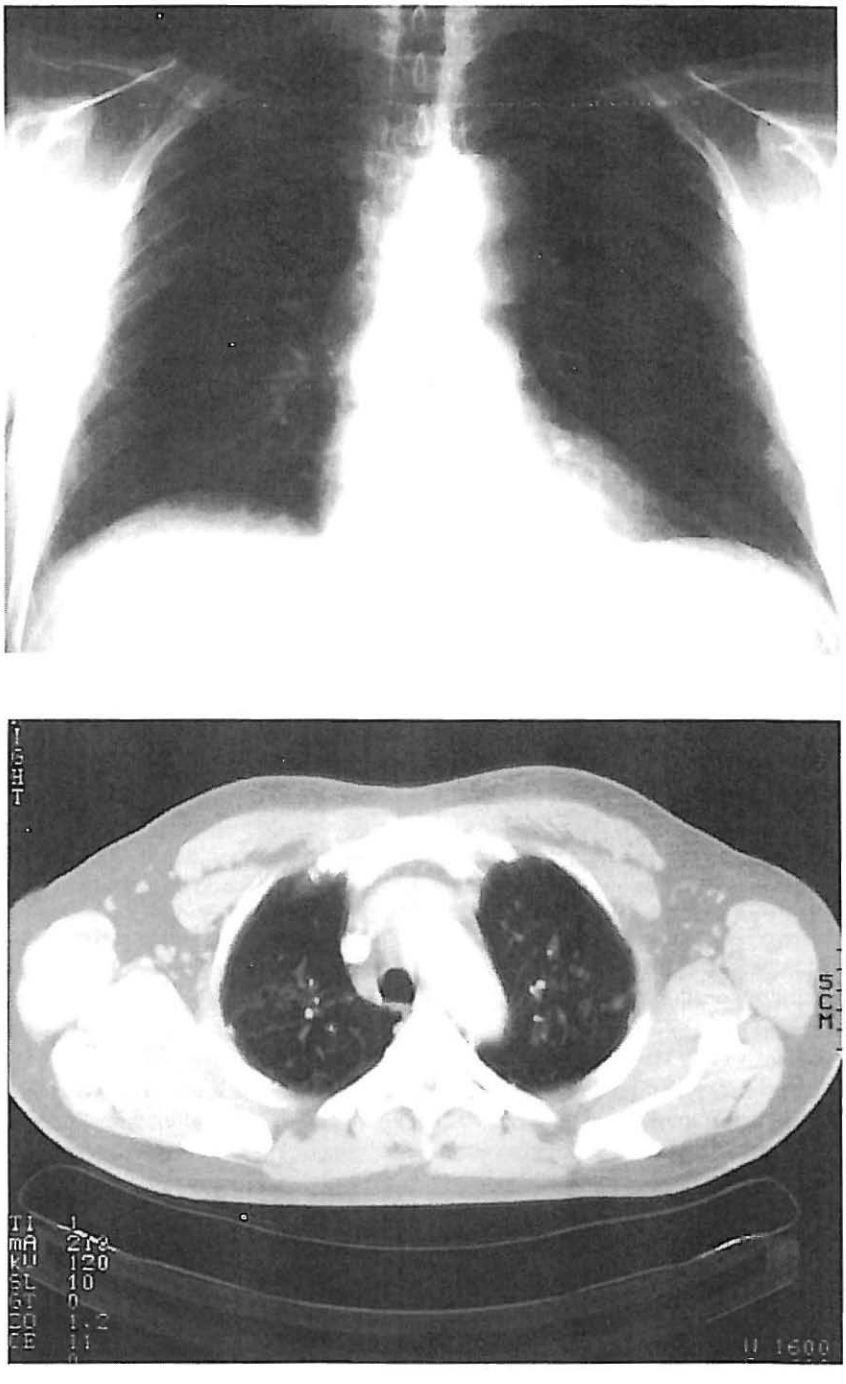
The development of silicosis after such a short time signals that the worker is at great risk for the immunologic sequelae of silicosis[74–77] and for the development of progressive massive fibrosis. Finally, acute silicosis occurs over a fewer number of years and is associated with an inexorable progression towards a respiratory death.

1. Simple Silicosis

Workers with simple silicosis are usually without chest symptoms. Some, however, report a chronic productive cough, a feature likely due to dust-induced bronchitis. Physical examination of the chest is usually unremarkable. Coarse adventitious sounds which may be present are the result of co-existing bronchitis.

Roentgenographically, simple silicosis presents itself as an upper zone distribution of rounded opacities less than one centimeter in diameter. In miners, these opacities have the same distribution as the nodules described in simple coalworkers' pneumoconiosis but are generally larger and more dense.[78] Peripheral "eggshell" calcifications are sometimes present in the hilar and mediastinal lymph nodes.

Attempts have been made to identify workers with preclinical silicosis before changes are evident on chest radiographs. A high-resolution computerized tomographic (CT) scan of the chest, a technique which has increased the effectiveness of imaging in interstitial lung diseases, has been used to detect parenchymal nodulation before these features are evident on plain chest films. Compared to the usual CT scan of the chest, a high-resolution CT scan allows for high-definition, thin-section scans (1–2 mm compared to 8–10 mm "cuts" by the usual technique of CT scanning) (Figures 5,6). The result is much finer resolution of parenchymal detail, better recognition of the type and distribution of regular and irregular opacities[79–82] and thus, a better understanding of the most likely interstitial lung disease. Importantly, when compared to the chest radiograph, the use of high-resolution CT scan has not added to our ability to diagnose silicosis earlier in a worker's career,[83,84] however, the use of high-resolution CT scans has allowed for the recognition of early "coalescence" of nodules and an earlier diagnosis of progressive massive fibrosis.



FIGURES 5 AND 6 The chest radiograph is from a 51-year-old, non-smoking underground miner who served as a roof bolter for approximately half of his 24 years of underground mining. The chest radiograph shows prominent parenchymal nodulations in the upper lung zones consistent with simple silicosis. The lung function tests were within normal limits. The CT scan shows these parenchymal nodules to be located in the posterior part of both upper zones. The nodules are discrete and without evidence of coalescence. A high-resolution CT scan was not available.

Pulmonary function studies in simple silicosis do not usually demonstrate functional impairment. Yet, there is a trend towards restriction in total lung capacity and compliance as the extent of profusion of small rounded opacities increases. This is most clearly manifested as overt restriction in some workers with progressive massive fibrosis. As the disease progresses, reduction in compliance usually precedes the

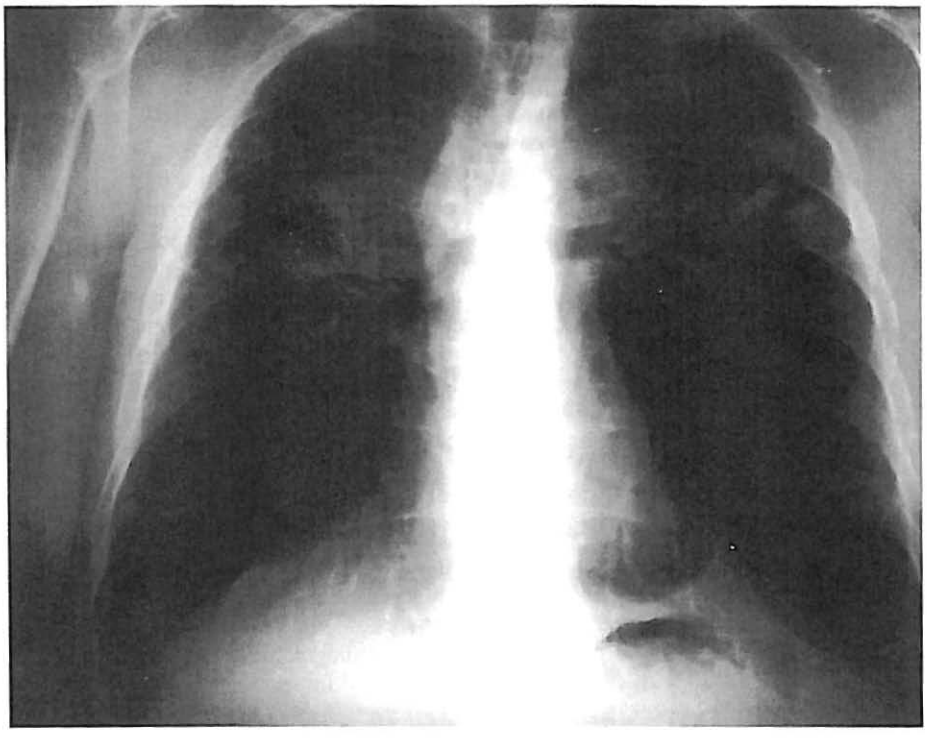


FIGURE 7 This chest radiograph is of a 38-year-old never-smoker employed as a surface coal mine driller from 1973 to 1986. The radiograph was taken one year after he stopped working at the mine. He has cough, morning sputum production, and dyspnea on exertion. Chest examination revealed a quiet chest without adventitious sounds. Lung function tests showed an FVC of 4.88 L (96% predicted), FEV_1 of 2.09 L (53% predicted), and a ratio of 43%. The diffusion capacity and the diffusion capacity adjusted for alveolar volume (KCO) were within normal limits. The chest radiograph shows large opacities in both upper zones of International Labor Office (ILO) category "B" and a background of minimal small rounded opacities. Although no earlier radiographs were available, the development of such severe disease in such a young man implies an accelerated progression of silicosis. In this case, the lung function tests show obstruction rather than the typical changes of restrictive impairment most commonly associated with progressive massive fibrosis.

reductions in vital capacity or forced expiratory flow rate. Few nonsmoking, dust-exposed workers with simple silicosis develop air flow abnormalities.[85,86] In a sophisticated study using high-resolution computed tomography (CT) scans of the chest to identify the presence of emphysema in workers with simple silicosis, Kinsella, et al., noted that simple silicosis did not cause significant emphysema and that it was the degree of emphysema, rather than the extent of simple silicosis, that determined the level of respiratory function. In those with progressive massive fibrosis, emphysema occurred frequently but was not different among smokers and nonsmokers.[87]

2. Progressive Massive Fibrosis

Progressive massive fibrosis is the result of the conglomeration of small rounded opacities. It has been traditionally recognized that progressive massive fibrosis develops on a background of advanced simple silicosis. Yet, not all coal miners who develop progressive massive fibrosis have an underlying advanced degree of simple coal workers' pneumoconiosis.[88] Whether this is also the case with silicosis has not been described (Figure 7).

The respiratory symptoms present in a worker with progressive massive fibrosis are again variable. They range from only a chronic productive cough to exertional dyspnea and, in some, ultimately to respiratory failure. With time, however, the progressive coalescence of silicotic nodules impairs the function of the underlying pulmonary parenchyma and results in progressive respiratory impairment.

Physical examination demonstrates decreased breath sounds on auscultation (explained by the emphysematous changes associated with progressive massive fibrosis) and, if the illness is extensive, signs of cor pulmonale and impending respiratory failure. Crackles do not occur as a result of the fibrotic changes, and finger clubbing, if present, is attributable to another etiology.

The chest roentgenogram reveals confluent nodules greater than one centimeter in diameter which occur on a background of small rounded opacities, which is recognizable as simple silicosis. The confluence of these nodules begins posteriorly and peripherally and migrates centrally. As with simple silicosis, progressive massive fibrosis develops most prominently in the upper lobes. As these upper lobe fibrous masses progressively enlarge, the hila are retracted upward and the lower zones become hyperinflated and appear as bullous emphysema. The presence of these upper zone opacities is often discussed in the context of possible neoplastic processes in the lung, particularly if they are not symmetrical. Importantly, progressive massive fibrotic lesions are relatively thin and plate-like and are located in the peripheral and posterior aspects of the upper lung zones. These radiographic features are sometimes helpful in separating the lesions of progressive massive fibrosis from a malignant etiology for these pulmonary masses.

Pulmonary function studies initially demonstrate a decrease in compliance followed by decreases in lung volume and diffusing capacity. If bronchial distortion and lower zone hyperinflation are present, the forced expiratory time is likely to be prolonged and airflow obstruction is measurable. Deterioration in lung function commonly occurs despite discontinuing silica exposure. The likelihood of progression directly correlates with the duration and concentration of silica exposure, as well as the presence or absence of mycobacterial infection.

C. ACCELERATED SILICOSIS

Accelerated silicosis is radiographically identical to classic silicosis except that the time from initial exposure to silica to the development of a radiographic diagnosis of silicosis, and the pulmonary function changes attributable to silicosis, occur over a shorter time and are often exaggerated. Accelerated silicosis is associated with a relatively rapid progression from the radiographic changes of simple silicosis to progressive massive fibrosis with ensuing severe respiratory impairment and a shortened life span (Figures 8, 9).

Silicotics have an increased prevalence of autoimmune serology, elevated gamma globulin levels, and an increased frequency of connective tissue disease, particularly scleroderma. Alternatively, concomitant connective tissue disease may also influence the course of pneumoconiosis. In 1953, Caplan noted that the course of coal workers' pneumoconiosis in workers with dust exposure could be influenced by co-existing rheumatoid arthritis. Upper zone large opacities appeared more frequently in the lungs of workers with underlying rheumatoid arthritis.

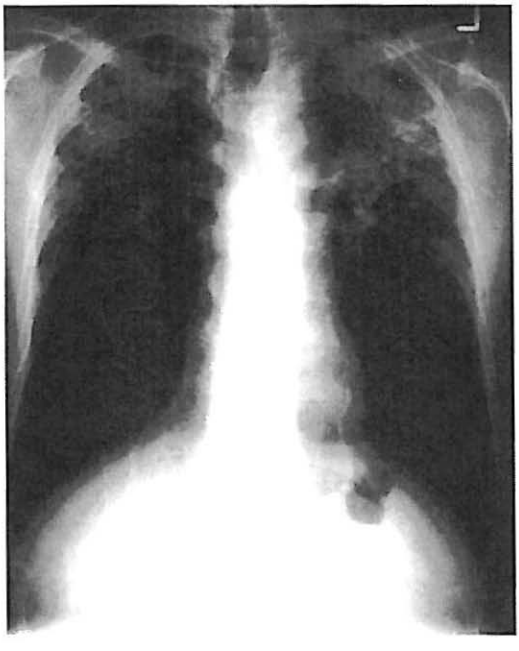
The role of the generally stimulated immune system is not well understood in this disease. Antinuclear antibodies and elevated immunoglobulin levels have not correlated with the baseline profusion category of the chest radiograph, the rate of chest radiographic progression, or the rate of lung function decline in sandblasters with silicosis.

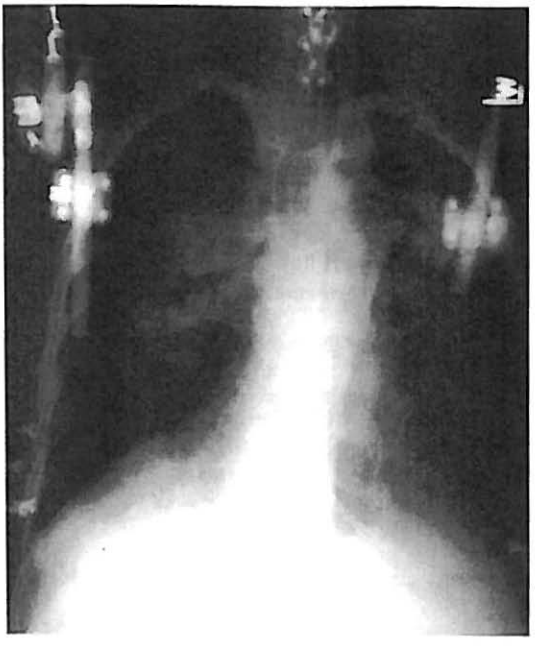
D. ACUTE SILICOSIS

Acute silicosis, the most aggressive form of silicosis, occurs over a short duration of exposure to overwhelmingly high concentrations of respirable free silica. The worker progresses to disabling chest symptoms and severe respiratory impairment, which invariably leads to death due to respiratory failure. Although patients with this form of silicosis may have some features of classic silicosis, there are distinct clinical, radiographic, and histologic differences.

In 1969, Buechner reported four sandblasters with acute silicosis and coined the term silico-proteinosis.[18] At autopsy, these sandblasters had periodic acid Schiff (PAS), positive-staining proteinaceous material filling the alveolar spaces, silica particles in the lung, and histologic changes of alveolar proteinosis. However, a review of the literature prior to Buechner's description suggests that many of the earlier patients had similar histologic features. Suratt reported the same presentation in tombstone sandblasters.[3] Chapman reported the histology to show localized areas of basilar "bronchopneumonia" in which the alveoli were filled with a pink staining edema fluid with a high protein content.[71] Gardner confirmed the uniqueness of this presentation by showing the most common lesion to be an alveolar exudate in which few, if any, cells could be identified.[90] Silico-proteinosis is a descriptive term of the histologic findings of the lungs in those who develop silicosis over a very short period of time (acute silicosis) and not a separate entity.

Workers with acute silicosis have been reported to have an irritative, sometimes productive, cough, weight loss, fatigue, and occasionally, pleuritic pain. Symptoms begin usually one to three years after the initial exposure; however, in very rare examples, symptoms occurring less than a year after beginning





FIGURES 8 AND 9 These radiographs are from a 55-year-old man who was first seen in 1988 and later in 1992. He provided a 75 pack year smoking history and had worked as a rock driller for approximately 20 years. Mycobacterial infection was searched for but not found. A PPD was non-reactive. The dramatic worsening of the radiograph over just four years reflects an accelerated course associated with a shortened life span. The chest radiographs show the way the coalescence of opacities in the upper zones occur and form progressive massive fibrotic lesions. The neck brace in the second radiograph was a part of therapy for a traumatic neck injury.

sandblasting have been reported. Unlike the quiet chest examination of classical silicosis, crackles are usually present and likely reflect alveolar and airway fluid. Patients rapidly develop cyanosis, symptoms of cor pulmonale, and respiratory failure. Survival after the onset of symptoms is typically less than four years. Mycobacterial and fungal infections frequently complicate the clinical course.

The chest radiograph typically reveals bilateral basilar alveolar filling with air bronchograms.[70,91] The diffuse alveolar filling is best described as a ground-glass appearance. Histologically small rounded opacities can sometimes be identified, but they are not easily recognized on the chest radiograph. Progression of the chest radiograph occurs over a relatively short time. Areas of alveolar filling progress to large masses which are similar to those seen in progressive massive fibrosis but somewhat larger and often located in the mid, compared to upper, zones. Tracheal distortion is common and a result of the parenchymal distortion with stress placed on the trachea. The process of radiographic progression is accelerated in these workers by a superimposed mycobacterial infection.

Acute silicosis can usually be diagnosed on the basis of a history of employment in an occupation where the opportunity for overwhelming silica exposure exists and where the clinical features and chest radiograph are consistent with this illness. If a review of the lung tissue becomes necessary for an accurate diagnosis, an open lung biopsy is recommended. Despite appropriate therapy for any underlying chest infection, the worker's lung function continues to deteriorate. Perhaps the best differential diagnosis of these very unusual chest radiographs includes alveolar proteinosis, bronchiolitis obliterans with organizing pneumonia, desquamative interstitial pneumonitis, and lipoid pneumonia. These entities can usually be excluded on a clinical basis.

V. CONCLUSION

The prevention of silicosis has not yet been achieved. If the past leads the way to the future, then it is by no means guaranteed that this disease can ever be totally abolished. Cases continue despite aggressive enforcement policies by regulatory agencies, attempts at dissemination of very readable

materials aimed at workers and management,[92,93] and respiratory dust exposure standards which appear protective and are in place. It is more than frustrating for all parties to see reports of aggressive silicosis in young workers. Perhaps, like the crusade against drunk driving, a determined effort through the contemporary methods of communication might enlighten American society and its workers about the very destructive health effects of silica exposure. Furthermore, both the physician and the affected worker are helpless when a diagnosis of silicosis has been made, and there is no way to affect the natural history of the processes that are underway. What must be developed is a type of "salvage therapy" that has the potential to reverse, or at least dramatically lessen, the aggressive natural history of silicosis that develops over relatively short time periods. Perhaps, if the illness cannot be prevented, an approach to lessen the effect of the fibrosis induced by this dust can be developed.

ACKNOWLEDGMENT

Supported in part by National Institute for Occupational Safety and Health grant #U60/CCU306149–01.

REFERENCES

1. **Betts, W. W.,** Chalicosis pulmonum or chronic interstitial pneumonia induced by stone dust, *JAMA,* 34, 70–74, 1900.
2. **Banks, D. E., Morring, K., Boehlecke, B. A., Althouse, R., and Merchant, J. A.,** Silicosis in silica flour workers, *Am. Rev. Respir. Dis.,* 124, 445–450, 1981.
3. **Suratt, P. M., Winn, W. C., Brody, A. R., Bolton, W. K., and Giles, R. D.,** Acute silicosis in tombstone sandblasters, *Am. Rev. Respir. Dis.,* 115, 521–529, 1977.
4. **Russell, A. E., Britten, E. H., Thompson, L. R., and Bloomfield, J. J.,** The health of workers in dusty trades — II. Exposure to siliceous dust (granite industry), Public Health Bull. No. 187, U.S. Treasury Department, Public Health Service, 1929.
5. **Russell, A. E.,** The health of workers in dusty trades — VII. Restudy of a group of granite workers, Public Health Bull. No. 269, Federal Security Agency, U.S. Public Health Service, Washington, D.C., 1941.
6. **Hosey, A. D., Trasko, V. M., and Ashe, H. B.,** Control of silicosis in the Vermont granite industry: progress report, Publication 557, U.S. Department of Health, Education and Welfare, Washington, D.C., 1957.
7. **Glindmeyer, H. W. and Hammad, Y.,** Contributing factors to sandblaster's silicosis: inadequate respiratory protection equipment and standards, *J. Occup. Med.,* 30, 917–921, 1988.
8. **Davies, C. N.,** Inhalation risk and particle size in dust and mist, *Br. J. Ind. Med.,* 6, 245–253, 1949.
9. **Banks, D. E., Bauer, M. A., Castellan, R. M., and Lapp, N. L.,** Silicosis in surface coalmine drillers, *Thorax,* 38, 275–278, 1983.
10. **Fleming, D., Maynard, D., McKinney, B., Perrotta, D. M., Schulze, L., and Pichette, J.,** Silicosis: Clusters in sandblasters-Texas, and occupational surveillance for silicosis, *Morbidity and Mortality Weekly Report,* 39, 433–437, 1990.
11. **Amandus, H. E. and Piacitelli, G.,** Dust exposures at U.S. surface coal mines in 1982–1983, *Arch. Environ. Health,* 42, 374–381, 1987.
12. National Institute for Occupational Safety and Health, Criteria for a recommended standard. Occupational exposures to crystalline silica, Washington, D.C., U.S. Department of Health, Education and Welfare, Public Health Service, Centers for Disease Control, 1974, 54–55.
13. Work related lung disease surveillance report, U.S. Department of Health and Human Resources, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1991, 27.
14. **Graham, W. G. B., Ashikaga, T., Hemenway, D., Weaver, S., and O'Grady, R. V.,** Radiographic abnormalities in Vermont granite workers exposed to low levels of granite dust, *Chest,* 100, 1507–1515, 1991.
15. Office of Federal Register, Code of federal regulations: occupational safety and health standards. Subpart Z: Air contaminants-permissible exposure limits. Table-1-A, Washington D.C., Office of the Federal Register, National Archives and Records Administration, 1989 (29 CFR 1910.1000).
16. **MacKay, G. R., Stettler, L. E., Kommineni, C., and Donaldson, H. M.,** Fibrogenic potential of slags used as substitutes for sand in abrasive blasting operations, *Am. Ind. Hyg. Assoc. J.,* 41, 836–842, 1980.
17. **Stettler, L. E., Procter, J. E., Platek, S. F., Carolan, R. J., Smith, R. J., and Donaldson, H. M.,** Fibrogenicity and carcinogenic potential of smelter slags used as abrasive blasting substitutes, *J. Toxicol. Environ. Health,* 25, 35–36, 1988.
18. **Buechner, H. A. and Ansari, A.,** Acute silico-proteinosis, *Dis. Chest,* 55, 274–284, 1969.

19. **Manfreda, J., Sidwall, G., Maini, K., West, P., and Cherniack, R. M.,** Respiratory abnormalities in employees of the hard rock mining industry, *Am. Rev. Respir. Dis.*, 126, 629–634, 1982.
20. **Kreiss, K., Greenberg, L. M., Kogut, S. J. H., Lezotte, D. C., Irvin, C. G., and Cherniak, R. M.,** Hard rock mining exposures affect smokers and nonsmokers differently, *Am. Rev. Respir. Dis.*, 139, 1487–1493, 1988.
21. **Teculescu, D. B., Stanescu, D. C., and Pilat, L.,** Pulmonary mechanics in silicosis, *Arch. Environ. Health*, 14, 461–468, 1967.
22. **Becklake, M. R., DuPreez, L., and Lutz, W.,** Lung function in silicosis of the Witswatersrand gold miner, *Am. Rev. Tuberc.*, 77, 400–412, 1958.
23. **Ehrlich, R. I., Rees, D., and Zwi, A. B.,** Silicosis in the non-mining industry on the Witswatersrand, *S. Afr. Med. J.*, 73, 704–708, 1988.
24. **Jones, R. N., Weill, H., and Ziskind, M.,** Pulmonary function in sandblasters silicosis, *Bull. Physiopath. Respir.*, 11, 589–595, 1975.
25. **Irwig, L. M. and Rocks, P.,** Lung function and respiratory symptoms in silicotic and nonsilicotic gold miners, *Am. Rev. Respir. Dis.*, 117, 429–435, 1978.
26. **Cowie, R. L. and Mabena, S. K.,** Silicosis, chronic airflow obstruction, and chronic bronchitis in South African gold miners, *Am. Rev. Respir. Dis.*, 143, 80–84, 1991.
27. **Hughes, J. M., Jones, R. N., Gilson, J. C., Hammad, Y. Y., Samimi, B., Hendrick, D. J., Turner-Warwick, M., Doll, N. J., and Weill, H.,** Determinants of progression in sandblasters' silicosis, *Ann. Occup. Hyg.*, 26, 701–712, 1982.
28. Criteria for a recommended standard...Occupational exposure to crystalline silica, U.S. Department of Health, Education and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, HEW publication No. (NIOSH) 75–120, 1974.
29. **Theriault, G. P., Burgess, W. A., DiBernardinis, L. J., and Peters, J. M.,** Dust exposure in the Vermont granite sheds, *Arch. Environ. Health*, 28, 12–17, 1974.
30. **Theriault, G. P., Peters, J. M., and Fine, L. J.,** Pulmonary function in Vermont granite shed workers, *Arch. Environ. Health*, 28, 18–22, 1974.
31. **Theriault, G. P., Peters, J. M., and Johnson, W. M.,** Pulmonary function and roentgenographic changes in granite shed exposure, *Arch. Environ. Health*, 28, 23–27, 1974.
32. **Graham, W. G. B., O'Grady, R. V., and Dubuc, B.,** Pulmonary function loss in Vermont granite workers, *Am. Rev. Respir. Dis.*, 123, 25–28, 1981.
33. **Ashe, H. B.,** Silicosis and dust control, *Public Health Rep.*, 70, 983–985, 1955.
34. **Ashe, H. B. and Bergstrom, D. E.,** Twenty six years experience with dust controls in the Vermont granite industry, *Ind. Med. Surg.*, 33, 73–78, 1964.
35. **Graham, W. G. B., Ashikaga, T., Hemenway, D., Weaver, S., and O'Grady, R. V.,** Radiographic abnormalities in Vermont granite workers exposed to low levels of granite dust, *Chest*, 100, 1507–1514, 1991.
36. **Seaton, A., Legge, J. S., Henderson, J., and Kerr, K. M.,** Accelerated silicosis in Scottish stonemasons, *Lancet*, 337, 341–344, 1991.
37. **Westerholm, P.,** Silicosis: Observations on a case register, *Scand. J. Work Environ. Health*, 1–86 (suppl 6), 1980.
38. **Finkelstein, M., Kusiak, R., and Suranyi, G.,** Mortality among miners receiving compensation for silicosis in Ontario: 1940–1975, *J. Occup. Med.*, 24, 663–667, 1982.
39. **Sherson, D. and Lander, F.,** Morbidity of pulmonary tuberculosis among silicotic and nonsilicotic foundry workers, *J. Occup. Med.*, 32, 110–113, 1990.
40. **Craighead, J. E. and Vallyathan, N. V.,** Cryptic pulmonary lesions in workers occupationally exposed to dust containing silica, *JAMA*, 244, 1939–1941, 1980.
41. **Craighead, J. E., Kleinerman, J., Abraham, J. L., Gibbs, A. R., Green, F. H. Y., Harley, R. A., Ruettner, J. R., Vallayathan, N. V., and Juliano, E. B.,** Diseases associated with exposure to silica and nonfibrous silicate minerals, *Arch. Pathol. Lab. Med.*, 112, 673–720, 1988.
42. **Bowden, D. H. and Adamson, I. Y. R.,** The role of cell injury in the continuing inflammatory response in the generation of silicotic pulmonary fibrosis, *J. Pathol.*, 144, 149–161, 1984.
43. **Adamson, I. Y. R. and Bowden, D. H.,** Role of polymorphonuclear leukocytes in silica-induced pulmonary fibrosis, *Am. J. Pathol.*, 117, 37–43, 1984.
44. **Lugano, E. M., Dauber, J. H., and Daniele, R. P.,** Acute experimental silicosis, *Am. J. Pathol.*, 109, 27–36, 1982.
45. **Callis, A. H., Sohnle, P. G., Mandel, G. S., and Mandel, N. S.,** The role of complement in experimental silicosis, *Environ. Res.*, 40, 301–312, 1986.
46. **Christman, J. W., Emerson, R. J., Graham, W. G. B., and Davis, G. S.,** Mineral dust and cell recovery from the bronchoalveolar lavage of healthy Vermont granite workers, *Am. Rev. Respir. Dis.*, 132, 393–399, 1985.
47. **Lowrie, D. B.,** What goes wrong with the macrophage in silicosis? *Eur. J. Respir. Dis.*, 63, 180–182, 1982.
48. **Schmidt, J. A., Oliver, C. N., Lepe-Zunige, J. L., Green, I., and Gery, I.,** Silica-stimulated monocytes release fibroblast proliferation factors identical to interleukin 1, *J. Clin. Invest.*, 73, 1462–1472, 1984.
49. **Martin, T. R., Altman, L. C., Albert, R. K., and Henderson, W. R.,** Leukotriene B_4 production by the human alveolar macrophage: a potential mechanism for amplifying inflammation in the lung, *Am. Rev. Respir. Dis.*, 129, 106–111, 1984.

50. **Bitterman, P. B., Wewers, M. D., Rennard, S. I., Adelberg, S., and Crystal, R. G.,** Modulation of alveolar macrophage driven fibroblast proliferation by alternative macrophage mediators, *J. Clin. Invest.*, 77, 700–708, 1986.
51. **Davis, G. S.,** Pathogenesis of silicosis: current concepts and hypotheses, *Lung*, 164, 139–154, 1986.
52. **Snider, D. E.,** The relationship between tuberculosis and silicosis, *Am. Rev. Respir. Dis.*, 118, 455–460, 1978.
53. **Baras, G.,** Silico-tuberculose en Suisse, *Schweiz. Med. Wochenschr.*, 100, 1802–1805, 1970.
54. **Cowie, R. L., Langton, M. E., and Becklake, M. R.,** Pulmonary tuberculosis in South African gold miners, *Am. Rev. Respir. Dis.*, 139, 1086–1089, 1989.
55. **Morgan, E. J.,** Silicosis and tuberculosis, *Chest*, 75, 202–203, 1979.
56. **Hnizdo, E.,** Loss of lung function associated with exposure to silica dust and with smoking and its relation to disability and mortality in South African gold miners, *BJIM*, 49, 472–479, 1992.
57. **Hnizdo, E.,** Combined effect of silica dust and tobacco smoking on mortality from chronic obstructive lung disease in gold miners, *Br. J. Ind. Med.*, 47, 656–664, 1990.
58. **Hnizdo, E., Sluis-Cremer, G. K., and Abramowitz, J. A.,** Emphysema type in relation to silica dust exposure in South African gold miners, *Am. Rev. Respir. Dis.*, 143, 1241–1247, 1991.
59. **Hessel, P. A., Sluis-Cremer, G. K., and Hnizdo, E.,** Silica exposure, silicosis, and lung cancer: a necropsy study, *Br. J. Ind. Med.*, 47, 4–9, 1990.
60. **Wu, K.-G., Fu, H., Mo, C.-Z., and Yu, L.-Z.,** Smelting, underground mining, smoking, and lung cancer: a case-control study in a tin mine area, *Biomed. Environ. Sci.*, 2, 1–8, 1989.
61. **Heppleston, A. G.,** Silica, pneumoconiosis, and carcinoma of the lung, *Am. J. Ind. Med.*, 7, 285–294, 1985.
62. **Lynge, E., Kurppa, K., Kristoferson, L., Malker, H., and Sauli, H.,** Silica dust and lung cancer: results from the Nordic occupational mortality and cancer incidence registers, *J. Nat. Cancer Inst.*, 77, 883–889, 1986.
63. **Goldsmith, D. F., Guidotti, T. L., and Johnston, D. R.,** Does occupational exposure to silica cause cancer?, *Am. J. Ind. Med.*, 3, 423–440, 1982.
64. **McDonald, J. C.,** Silica, silicosis, and lung cancer, *Br. J. Ind. Med.*, 46, 289–291, 1989.
65. **Caplan, A.,** Certain unusual radiologic appearances in the chest of coalminers suffering from rheumatoid arthritis, *Thorax*, 8, 29–30, 1953.
66. **Ziskind, M., Jones, R. N., and Weill, H.,** Silicosis, *Am. Rev. Respir. Dis.*, 113, 643–665, 1976.
67. **Nugent, K. M., Dodson, R. F., Idell, S., and DeVillier, J. R.,** The utility of bronchoalveolar lavage and transbronchial lung biopsy combined with energy-dispersive x-ray analysis in the diagnosis of silicosis, *Am. Rev. Respir. Dis.*, 140, 1438–1441, 1989.
68. **Cahill, B. C., Harmon, K. R., Shumway, S. J., Mickman, J. K., and Hertz, M. I.,** Tracheobronchial obstruction due to silicosis, *Am. Rev. Respir. Dis.*, 145, 719–721, 1992.
69. **Dee, P., Suratt, P., and Winn, W.,** The radiographic findings in acute silicosis, *Radiology*, 126, 359–363, 1978.
70. **Sampson, H. L.,** The Roentgenogram in so-called "acute" silicosis, *Am. J. Pub. Health*, 23, 1237–1239, 1933.
71. **Chapman, E. M.,** Acute Silicosis, *JAMA*, 98, 1439–1441, 1932.
72. **Vallyathan, V., Shi, X., Dalal, N. S., Irr, W., and Castranova, V.,** Generation of free radicals from freshly fractured silica dust: potential role in acute silica-induced lung injury, *Am. Rev. Respir. Dis.*, 138, 1213–1219, 1988.
73. **Eden, K. G. and von Seebach, H. B.,** Atypical quartz dust induced pneumoconiosis in SPF rats. Aspects of the role of the lymphatic system in the pathogenesis of silicosis, *Virchows Arch. A.* 372, 1–9, 1976.
74. **Lippman, M., Eckert, H. L., Hahon, N., and Morgan, W. K. C.,** Circulating antinuclear and rheumatoid factors in coal miners. A prevalence study in Pennsylvania and West Virginia, *Ann. Intern. Med.*, 79, 807–811, 1973.
75. **Jones, R. N., Turner-Warwick, M., Ziskind, M., and Weill, H.,** High prevalence of antinuclear antibodies in sandblasters silicosis, *Am. Rev. Respir. Dis.*, 113, 393–395, 1976.
76. **Rodnan, G. P., Bendek, T. G., Medsger, T. A., and Cammarata, R. J.,** The association of progressive systemic sclerosis with coal miners' pneumoconiosis and other forms of silicosis, *Ann. Intern. Med.*, 66, 323–329, 1967.
77. **Haustein, U. F., Zeigler, V., Hermann, K., Melhorn, J., and Schmidt, C.,** Silica-induced scleroderma, *J. Am. Acad. Dermatol.*, 22, 444–448, 1990.
78. **Anderson, W. H., Hamilton, G. L., and Dorsett, B. E., Jr.,** A comparison of coal miners exposed to coal dust and those exposed to silica dust, *Arch. Environ. Health*, 1, 540–547, 1960.
79. **Muller, N. L. and Miller, R. R.,** Computed tomography of chronic infiltrative lung disease. Part 1, *Am. Rev. Respir. Dis.*, 142, 1206–1215, 1990.
80. **Muller, N. L. and Miller, R. R.,** Computed tomography of chronic infiltrative lung disease. Part 2, *Am. Rev. Respir. Dis.*, 142, 1440–1448, 1990.
81. **Mathieson, J. R., Mayo, J. R., Staples, C. A., and Muller, N. L.,** Chronic diffuse lung disease: comparison of diagnostic accuracy of CT and chest radiography, *Radiology*, 171, 111–116, 1989.
82. **Bergin, C. J., Coblentz, C. L., Chiles, C., Bell, D. Y., and Castellino, R. A.,** Chronic lung disease: specific diagnosis by using CT, *Am. J. Roentgenol.*, 152, 1183–1188, 1989.
83. **Begin, R., Bergeron, D., Samson, L., Boctor, M., and Cantin, A.,** CT assessment of silicosis in exposed workers, *Am. J. Roentgenol.*, 148, 509–514, 1987.
84. **Bergin, C. J., Muller, N. L., Vedal, S., and Chan-Yeung, M.,** CT in silicosis: correlation with plain films and pulmonary function tests, *Am. J. Roentgenol.*, 147, 477–483, 1986.

85. **Teculescu, D. B., Stanescu, D. C., and Pilat, L.,** Pulmonary mechanics in silicosis, *Arch. Environ. Health,* 14, 461–468, 1967.
86. **Jones, R. N., Weill, H., and Ziskind, M.,** Pulmonary function in sandblasters silicosis, *Bull. Physiopath. Resp.,* 11, 589–595, 1975.
87. **Kinsella, M., Muller, N., Vedal, S., Staples, C., Abboud, R. T., and Chan-Yeung, M.,** Emphysema in silicosis. A comparison of smokers with non-smokers using pulmonary function testing and computed tomography, *Am. Rev. Respir. Dis.,* 141, 1497–1500, 1990.
88. **Hodous, T. K. and Attfield, M. D.,** Progressive massive fibrosis developing on a background of minimal simple coal workers' pneumoconiosis. Proc. VIIth International Pneumoconiosis Conference. National Institute for Occupational Safety and Health, Department of Health and Human Services, (NIOSH) Publ. No. 90–108, Part 1, 123–126.
89. **Middleton, E. L.,** The present position of silicosis in industry in Britain, *Br. Med. J.,* 2, 485–489, 1929.
90. **Gardner, L. U.,** Pathology of so-called silicosis, *Am. J. Public Health,* 23, 1240–1249, 1933.
91. **Sampson, H. L.,** The Roentgenogram in so-called "acute" silicosis, *Am. J. Public Health,* 23, 1237–1239, 1933.
92. Centers for Disease Control Alert: Request for assistance in preventing silicosis and deaths from sandblasting, U.S. Department of Health and Human Services, Public Health Service, CDC, National Institute for Occupational Safety and Health, Department of Health and Human Services (NIOSH) Publ. No. 92–102.
93. Centers for Disease Control Alert: Request for assistance in preventing silicosis and deaths in Rock drillers, U.S. Department of Health and Human Services, Public Health Service, CDC, National Institute for Occupational Safety and Health, Department of Health and Human Services, (NIOSH) Publ. No. 92–107.

Silica and Silica-Induced Lung Diseases

Edited by

Vincent Castranova
Val Vallyathan
William E. Wallace

CRC Press
Boca Raton New York London Tokyo

Library of Congress Cataloging-in-Publication Data

Silica and silica-induced lung diseases / edited by Vincent
Castranova, Val Vallyathan, William E. Wallace.
p. cm.
Includes bibliographical references and index.
ISBN 0-8493-4709-2
1. Silicosis. I. Castranova, Vincent. II. Vallyathan, Val.
III. Wallace, William E., 1942– .
[DNLM: 1. Lung Diseases--chemically induced. 2. Lung Neoplasms-
-chemically induced. 3. Silicon Dioxide--adverse effects.
4. Silicosis--etiology. 5. Occupational Exposure. WF 600 S583
1995]
RC774.S52 1995
616.2'44--dc20
DNLM/DLC
for Library of Congress 95-6538
CIP

International Standard Book Number 0-8493-4709-2
Library of Congress Card Number 95-6538
Printed in the United States of America 1 2 3 4 5 6 7 8 9 0
Printed on acid-free paper