SECTION II
PNEUMOCONIOSES

SILICOSIS

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INTRODUCTION

In 1556 Agricola wrote in *De re metallica,* "It remains for me to speak of the ailments and accidents of mines, and of the methods by which we can guard against them, for we should always devote more care to maintaining our health, that we may freely perform our bodily functions, than to making profits." This advise is no less true today and underlines one of the reasons silicosis is prevalent. While our medical, engineering, epidemiologic, and toxicologic knowledge concerning silica and silicosis is incomplete, it is sufficient to render silicosis a rare disease where appropriate action is taken to reduce workplace exposure to the many dusts containing silica (SiO_2). Further research will undoubtedly refine our knowledge of: silica's mechanism of action; its interaction with other environmental agents (such as cigarette smoking); its genetic predisposition; its pathophysiology; and its dose-response relationships. But the elimination of silicosis, while relying on technology, will be sociological. How do we achieve "safe" dust levels in the workplaces in which exposure to silica is taking place? Regulation and enforcement are obviously important, but what are the best laws? The best economic incentives for control? The best political solutions? The best technical solutions? And what research needs to be conducted to provide "policy makers" with the right information to make policy compatible with both occupational health and production? In large part, this chapter is devoted to considering the scientific issues inextricably bound with policy issues which must be addressed before silicosis is a thing of the past.

DEFINITION

Silicosis is a fibrotic disease of the lungs produced by the inhalation and deposition of dust containing silicon dioxide or silica (SiO_2). It can take the acute form under conditions of intense exposure but usually takes the chronic form, requiring several to many years to develop. It has frequently been associated with tuberculosis (silicotuberculosis) and other mycobacteria which synergistically increase its pathogenicity. The interested reader is referred to several useful sources (19)(22)(33). Much of the following material is derived from these sources.

Chronic Manifestations

Chronic reactions, occurring over 20 to 45 years, usually involve exposure to dusts containing a relatively small proportion of quartz (30% or less). Lesions are usually nodular and are likely to be more prominent in the upper lobes. In this *simple* stage of silicosis, nodules are usually small (5mm or less). Normally this stage has little effect on pulmonary function.

Complicated silicosis (progressive massive fibrosis) also usually develops in the upper lobes. In this case fibrotic nodules coalesce and encompass blood vessels and airways. Function may be severely compromised under these conditions. In the past, tuberculosis was a common accompaniment of this condition.

Caplan's syndrome was first described in coal miners with rheumatoid arthritis (3). It was subsequently found in other mining occupations. Its most characteristic feature is larger, more rapidly developing nodules than those seen in simple silicosis. An increased prevalence of progressive massive fibrosis is seen in these individuals.

Acute and Accelerated Silicosis

Very high exposure to silica can result in acute silicosis. This disease may appear one to three years after the onset of exposure. The distinguishing feature of acute silicosis is intra-alveolar deposits (similar to those seen with alveolar proteinosis), appropriately termed "silicoproteinosis." Additionally, in contrast to the nodular fibrosis seen in the chronic form, diffuse

interstitial fibrosis is found. Silicosis developing in less than 10 years has been described most often in sandblasters. In these cases, massive fibrosis is likely to develop and locate in the middle and lower lobes. (See Acute Silicosis subsection, page 239).

CAUSATIVE AGENTS

Silicon dioxide or silica (SiO_2), the agent responsible for silicosis, occurs in three different mineralogical forms. These are quartz, cristobalite, and tridymite. Quartz has hexagonal crystals; cristobalite, cubic crystals; and tridymite, hexagonal. The noncrystalline forms of silica (amorphous silica) are considered to have little fibrotic potential. Heating, however, can change their structure into crystalline form.

The primary source of silica is quartz. Quartz is a mineral found in nearly all mineral deposits and is an important component of common rocks such as granite and sandstone. This is the principle reason workers are exposed to it in various occupations. Sand contains large amounts of quartz and is used in the glass and pottery industry as well as in brick, mortar, and abrasives production. In finely pulverized form (silica flour), it is added to soaps, paints, and porcelains. Since crystalline silica occurs in various colors, these materials find use as gems or for other decorative purposes.

Cristobalite and tridymite are two minerals that usually occur together, a major source being volcanic rock in California, Colorado, and Mexico (31). These two minerals can also be man-made by heating silica (either crystalline or amorphous) to high temperatures. This process occurs in calcining diatomaceous earth; resulting products find use in insulation, filters, and furnace linings.

Flints contain free silica and have been used for centuries because of their hardness and heat resistance.

OCCUPATIONS AND INDUSTRIES INVOLVED

Abrasives
Abrasive blasting
Boiler scaling
Cement production workers
Ceramics
Coal mining and milling
Fillers (paints, rubber, etc.)
Foundry work (ferrous and nonferrous)
Glass manufacture
Insulation production and installation
Metal mining and milling
Mining, quarrying and tunneling
Nonmetallic mining and milling
Plastic manufacturing
Pottery making
Refractories
Road working
Rubber manufacturing
Sandblasting
Scouring soap manufacturing
Tile and clay production
Tunneling
Vitreous enameling

EPIDEMIOLOGY

One definition of epidemiology is the study of the distribution and determinants of disease. The classical process consists of examining a series of variables to ascertain causation. Variables usually include age, sex, race, socioeconomic status, ethnicity, religion, etc. Since it is known that silica causes silicosis, and that a certain level of exposure produces disease in humans regardless of sex, age, or race, this section will deal primarily with evidence relating dose to response in human beings.

There are several major difficulties involved in attempting to do this. They fall primarily into three categories:

1) difficulties in the accurate determination of dose,
2) difficulties in the accurate determination of health effects (or disease) and,
3) difficulties in dealing with competing variables (such as cigarette smoking and host susceptibility).

These three problems will be generically discussed. Available literature will then be reviewed, and the final segment of this chapter will summarize the current state of knowledge and questions that must be addressed by further research.

Dose

It is obviously important to know how the silica particle exerts its toxic effect. Is it related to the number of particles? To the size of the particles? To the concentrations? Any attempt to make meaningful exposure measurements should involve consideration of these factors.

There is another set of questions relating to the deposition and clearance of particles. What are the characteristics of particles that penetrate

to the large airways? The small airways? The alveoli? What role do host factors, such as the immune system or anatomy, play? Are there special characteristics about particles that are cleared versus particles that destroy macrophages?

The measurement of airborne dust in the workplace must bear some general relationship to the toxic amount of silica delivered to the lung, but there are clearly important questions that can be raised about this relationship. We can accurately measure silica dust levels, but how do we assess the chronic, long-term effects of "low-level" exposure to silica? Assuming workplace exposure relates somehow to delivered dose, we still have the difficulty of ascertaining a working-lifetime exposure to silica. Over the years actual measurement techniques have changed, work processes have changed and environmental hygiene has changed.

Personal habits also affect exposure. Two persons working the same jobs may position themselves differently, may use the local exhaust ventilation differently, and may take their breaks in different places. All of these sorts of personal work characteristics make area sampling highly suspect in representing true exposure. Personal sampling techniques are obviously needed. Ideally we would like a personal sampling result for every worker every day. This is, of course, not possible. Even if it were, accurate personal sampling techniques have only been available for the past few years and in the case of silica, we need exposure data for the previous 20 to 40 years in order to determine lifetime exposure levels. Other components of dust besides silica may be important. It is uncommon in industries in which silica exposures occur for the exposure to be purely to SiO_2. Other materials are present in mines, foundries, quarries, construction sites, etc. These other exposures may be important either by directly influencing the toxicity of silica or by exerting effects on the respiratory system themselves. This raises the question of whether a certain amount of exposure to silica in a foundry produces the same effect as the same amount of silica in a granite shed.

Response—Health Effects

Silica has the potential to damage the lung. There are several ways to monitor silica-induced lung damage. First, on an individual basis, detectable effects on both chest x-ray and pulmonary function occur prior to frank clinical symptoms. Despite this, pulmonary impairment due to silica inhalation may proceed undiagnosed because (a) the disease usually takes several years to develop; (b) the affected individual ages during that interim and may smoke cigarettes or be exposed to significant air pollution; (c) the clinician may fail to inquire about the individuals' occupational history. It is also probable that since the lung normally has reserve capacity, significant damage could occur before it became clinically manifest.

Second, individual versus group effects must be considered. The acute loss of 200 or even 500 ml of $FEV_{1.0}$ in a male with a normal $FEV_{1.0}$ of 4.0 liters might clinically pass unnoticed. However, this same loss in a population exposed to silica would be epidemiologically significant. Since the population would contain individuals with already compromised pulmonary function, this additional loss could be critical.

Choosing the appropriate test to determine subtle effects from silica is important. While chest x-ray is the old standby, it has recently become clear that silica nodules, invisible to x-ray, can be found in workers with relatively mild exposures who have died from other causes (32). Pulmonary function tests may show effects earlier than x-ray, but this is unclear because unfortunately, it has not been common practice to measure baseline pulmonary function in workers prior to their exposure to silica.

Although it is well known that simple silicosis (diagnosed by x-ray) exists without profound clinical symptoms, it is not known how much silica (or silicosis) in the lung predisposes an individual to the development of massive fibrosis. This is an important question because progressive massive fibrosis (PMF) can occur in the absence of further silica exposure. At the present time, factors determining progression remain unclear.

Confounding

Apart from personal differences in work practices, smoking habits, etc., there are innate differences in individuals and their response to silica. This is clearly the case in Caplan's syndrome which probably occurs at a subtler level of disease and by different, little understood mechanisms. Parkes provides reference to a study of fluorspar miners in Sardinia in which

resistance and predisposition to silicosis may be genetically determined (22).

The study of health effects from silica inhalation in smokers has revealed a fairly wide variation in response. When the smoking habit is considered in workers exposed to silica, the problem is obviously compounded. The potential outcome of cigarette smoking is chronic obstructive pulmonary disease (COPD). In a cigarette smoker exposed to silica, the result is frequently mixed pulmonary disease. Given any such individual, apportioning risk to the two factors is difficult if not impossible. On a group basis, however, proper statistical techniques should be capable of allocating risk (28).

Despite the fact that silicosis has been a common, occupationally related disease for many years, only a few studies have been directed toward its epidemiologic aspects. This is probably due to the many difficulties associated with monitoring the health of a population over a period of time; the paucity of epidemiologists interested in occupational disease; and the difficulty of determining a lifetime dose of silica. On the other hand, data have existed for years that are relevant to standard setting, and "old" studies are remarkably compatible with newer "sophisticated" studies in pointing to a safe level of exposure.

Several old studies conducted by the Public Health Service revealed tragic amounts of silica exposure and silicosis in hard rock mining (12) (15). In addition, Harrington and Lanza demonstrated very high rates of silicosis in Butte copper miners (10). Later, Dreesen et al. reported silicosis with concentrations of silica dust ranging from 2 to 37 million particles per cubic foot (mppcf). No cases were seen in workers whose exposures were 10 years or less in duration with concentrations averaging 18 mppcf (5). Vitally needed are studies that look at exposures over a working lifetime and that also consider the health of the worker after exposure stops, i.e., after retirement.

Flinn et al. studied metal miners during the period 1958 to 1961 (6). This study, involving 50 mines, included over 14,000 employees and 14,480 impinger samples. The quartz content varied from a reported 2% - 95%. Dust levels ranged from 0 to over 50 mppcf. The health assessment consisted of a medical history, occupational history, pulmonary function tests, and chest x-rays. Not surprisingly, a relationship was found between duration of exposure and prevalence of silicosis. Workers whose exposure had not exceeded 5 years duration were unaffected. Workers who had been exposed for 30 or more years had prevalence rates exceeding 60%.

All studies of work populations exposed to silica raise the problem of previous exposures to higher levels. No exception was the study by Renes et al. involving iron foundries (24). Almost 2,000 men were examined and over 9% were found to be affected. Those who had worked for 20 or more years had a prevalence rate of 25.8%. Over 80% of the air samples were below 6.9 mppcf. But the silicosis cases were attributed to past exposures which allegedly were considerably higher.

Few studies give reliable lifetime estimates of dust exposure. An exception is the study of Flinn et al., which focused on 9 West Virginia potteries (7). Over 2,500 individuals received physical examinations and had chest x-rays taken. From this study, 189 were diagnosed as having silicosis. Impinger samples were collected to assess breathing zone exposure to silica. Quartz concentrations were measured in settled dust and ranged from 1%-39%. Table II-1 presents some of the results.

Their justifiable conclusion was that exposures should be kept below 4 mppcf if new cases were to be avoided.

The early devastation caused by silica exposure seems to have caused some people to be satisfied with improved conditions. Few rigorous attempts to prove safety have been made, and much reliance has been placed on old techniques and clinical diagnoses. An example of this is the study by Rajhans and Budlovsky in which workers in an Ontario brick plant were studied (23). While they claim no cases of silicosis have appeared in this industry, it is not clear that this group is entirely free of respiratory disease. First, they relied primarily on 70 mm chest x-rays which provide less definition than the standard 14 × 17″ x-ray. Second, there were few workers who had long employment histories. Perhaps the most interesting question raised by their paper is that of the possible interaction of other dusts in the environment. Certainly the physical-chemical qualities of dusts are different. Exactly how these different qualities effect pulmonary response is not clear. A study done earlier by Keatinge and Potter revealed similar results in a British brickworks (14).

Table II-1

RELATION OF DUST CONCENTRATION AND LENGTH OF EMPLOYMENT IN THE POTTERY INDUSTRY TO SILICOSIS*

Dust Concentration million particles/cu ft	Years in Pottery Industry 0-9	10-19	20-29	30-39	Over 40
0-3.9:					
Cases of silicosis	-	1	1	-	-
Workers exposed	481	223	65	21	8
Percentage	0	0.4	1.5	0	0
4-7.9:					
Cases of silicosis	1	6	26	27	29
Workers exposed	321	198	110	53	34
Percentage	0.3	3	24	51	85
8-15.9:					
Cases of silicosis	-	8	5	10	10
Workers exposed	176	119	25	17	14
Percentage	0	7	20	59	71
Over 16:					
Cases of silicosis	13	33	10	5	4
Workers exposed	363	174	21	7	5
Percentage	4	19	48	71	80

*Includes 1st, 2nd, and 3rd stage cases.

Fulton et al. came up with different results in a Pennsylvania brickworks (8). The material used to make the brick was significantly different from that in Ontario; it contained more quartz and less aluminum. Very high prevalence rates of silicosis were found in this population. Silicosis was found at all levels of exposure, except below 2 mppcf. It was also found to be more prevalent in workers involved with burned brick (which contained tridymite and cristobalite) than with "green" brick. The silica content of both was high.

While it appears the Flinn et al. study (7) considers safe levels to be below 4 mppcf and the Fulton et al. study (8) considers safe levels to be below 2 mppcf, it is important to note the proportion of silica in these dusts is different.

The granite industry in Vermont has been the source of much useful data relevant to the public health questions about silica. This industry, no different from others involving silica exposures, produced its share of disability and death. The cemetery at Barre, Vermont provides dramatic reminders of the early tragedies associated with this industry. It is said that granite cutters, learning of their affliction with silicosis or silicotuberculosis, spent the last months of their lives producing their own gravestones.

The original study of Vermont granite workers was carried out by Russell et al. They found "universal occurrence of silicosis among the workers" and "appallingly high death rates from tuberculosis" (26). A later study by Russell et al. documented cases in occupations where the dust exposure averaged 3-9 mppcf (25). As in most studies, the question of earlier exposure to higher levels was raised. They recommended a safe limit of 9-20 mppcf for dust containing 35% silica. Russell later recommended a standard of about 10 mppcf.

It is clear that measures to control dust in Vermont had a tremendous impact. Wet methods and local exhaust ventilation established about 1937-1940 have greatly diminished the health risk.

Hosey et al. conducted an environmental study of the Vermont granite industry and demonstrated that few exposures exceeded 5 mppcf (13). The prevalence of silicosis diagnosed by x-ray decreased from 45% in 1937 to 15% in

1956. This group reported that only one new—but questionable—case of silicosis occurred in a worker beginning work after "dust control" (1940).

Ashe and Bergstrom found no new cases of silicosis in workers exposed for up to 20 years at levels of between 3-5 mppcf (1). However, they wisely suggested continued environmental and medical surveillance. This entire population was again restudied cross-sectionally by Theriault et al. (27)(28)(29) and longitudinally by Musk et al. (20).

In 1969, the Harvard School of Public Health joined with the Industrial Hygiene Division of Vermont in a comprehensive study of the relationship between exposure to granite dust, percent quartz content of the dust, and lung disease among granite shed workers exposed for many years to low levels of granite dust. To estimate current dust exposure in the granite sheds of Vermont, 784 personal respirable dust samples collected from 13 occupational groups in 49 granite sheds; 483 of these samples were analyzed for quartz content (27). A lifetime estimate of exposure to granite dust and quartz was calculated for each worker from the dust concentration data and a complete occupational history. Five indices of exposure were developed, and dust-year was selected by a multiple regression analysis as the index most highly correlated with changes in vital capacity (FVC). (Current dust and quartz concentrations in the granite sheds differ from previous estimates due to differences in sampling and analytical techniques.) Important conclusions reached from these studies are: 1) It was determined that 10 mppcf of granite dust was the rough equivalent of 0.1 mg/m³ of quartz. 2) The quartz content of the granite dust was estimated at 9%. 3) Average one year exposure was to 523 $\mu g/m^3$ of granite dust and 50 $\mu g/m^3$ of quartz.

The granite dust and quartz dust concentrations are presented in Tables II-2, II-3, and II-4. During the period of the study, the average granite dust concentration was 523 $\mu g/m^3$; quartz averaged 50 $\mu g/m^3$. Exposures were adjusted to these quantities and called a dust-year. It must be stressed that in the last decade, major changes in dust sampling and dust analysis have taken place. Personal lapel sampling has replaced fixed location sampling, thereby providing a better estimate of the dust actually breathed by the workers. Mass respirable sampling now provides mass dust concentration data rather than the count concentration from the impinger sampling technique used earlier. Although these changes have improved the accuracy of estimating dust exposure, they have presented a challenge in establishing a lifetime dust and quartz exposure.

In calculating lifetime dust exposures, workers employed prior to 1940 were assumed to have had dust exposures 10 times higher than present levels. This factor was derived from a study of a shed without dust controls (2).

Seven hundred and ninety-two active granite shed workers from Barre, Vermont were studied to estimate the effect of granite dust inhalation on pulmonary function. Based on a complete occupational history and a comprehensive evaluation of the past and present environment, a total lifetime dust and quartz exposure for each worker was established.

Effects of granite dust on pulmonary function were reported by Theriault et al. (28). When the workers were seen for their annual chest roentgenograms (provided through a comprehensive health program instituted in 1937 by the Industrial Hygiene Division of Vermont), they were asked to participate in a study of their pulmonary function and to answer a brief questionnaire on their smoking habits. Ventilatory capacity was measured with a spirometer. The forced vital capacity (FVC) and the forced expiratory volume in one second (FEV_1) were measured. Total lung capacity (TLC) was estimated from anteroposterior and lateral chest roentgenograms and the residual volume (RV) was obtained by the subtracted difference between TLC and FVC. Workers' smoking histories were grouped as follows: those who had never smoked; ex-smokers (those who had stopped smoking for six months or more); or current smokers (those who smoked one or more cigarettes a day). The amount of smoking was quantified in cigarettes per day and in years smoked.

There were three principal results based on the evaluation of lung function. The first is related to multiple regression techniques and is shown in Table II-5. While the loss attributable to dust exposure is small, it is statistically significant and could represent a significant loss if multiplied by many years and a higher than average dustiness. It also must be remembered that this loss is the average loss for the entire population which means that some individuals would be losing less and some more.

The second major finding related lifetime

Table II-2

OCCUPATIONAL CLASSIFICATION AND AIR SAMPLING FREQUENCY

Occupation (Classification No.)	No. of Dust Samples	No. of Quartz Samples
Cutter, letter cutter (1)	258	202
Sculptor, carver (2)	32	19
Polisher, surface machine operator (3)	104	65
Sandblast operator (4)	52	30
Carbo-saw operator, contour planer, grinder, diamond-saw operator, circular-saw operator (5)	48	38
Gang-saw operator, wire-saw operator (6)	97	44
Hydraulic splitter operator (7)	17	11
Crane operator (8)	4	2
Grouter, lumper, bedsetter (9)	10	5
Stencil cutter (10)	13	8
Boring mill operator, lathe operator, tool grinder (11)	9	4
Finisher, plug drill operator (12)	40	26
Boxer, derrickman, foreman, maintenance, general air, stone washer, torch burner (13)	100	29
Total	784	483

Table II-3

GRANITE DUST CONCENTRATION BY OCCUPATION AND SHED*

		Occupation						
		1	2	3	4	5	—	14
Granite Shed	1	634	644†	583	270	212	—	476
	2	515	398†	259	230	268†	—	293
	3	653	696	762	392†	368	—	478
	4	576†	679†	640	411†	438	—	502
	50	601	708	565	429	480	—	523

*All concentrations expressed as micrograms per cubic meter.

†Calculated by method described in text.

Table II-4

QUARTZ CONCENTRATION BY OCCUPATION AND SHED*

		Occupation						
		1	2	3	4	5	—	14
Granite Shed	1	93	95†	54	13	19	—	48
	2	54	35†	11	12†	12†	—	18
	3	72	104†	111	17	29	—	53
	4	99	166†	86	58	60	—	84
	—	—	—	—	—	—	—	—
	50	61	99	46	35	36	—	50

*All concentrations expressed as micrograms per cubic meter.
†Calculated by method described in text.

dust exposure to pulmonary function. By generating a prediction equation from individuals who had had no dust exposure, a percent predicted was calculated for workers with increasing dust exposure. Figure II-1 presents the results.

Table II-5

EFFECT OF AGE, HEIGHT, SMOKING, AND DUST ON FVC* AS ESTABLISHED BY MULTIPLE REGRESSION ANALYSIS

Independent variables	b ± SE	p
Age	29.9 ± 0.244	<.001
Height	54.7 ± 0.314	<.001
Years smoked	8.8 ± 0.183	<.01
Dust-years	1.6 ± 0.057	<.01

*FVC is dependent variable. Total R square = 0.532; b represents regression coefficient; −4072.6 is constant used.

Quartz Effect on Pulmonary Function

It is clear that both total dust and quartz are correlated with adverse pulmonary function. While the midpoint 50% occurs 2.5 years later for quartz than it does for granite dust, these are small differences; we must be hesitant in attributing an additive effect to total dust. However, the fact that the effect of quartz on the lung is different from that of granite dust reinforces the suggestion that mass respirable quartz dust should have a TLC of its own.

The third important finding relates to the relative effects of cigarette smoking and dust exposure. If regression equations are calculated for each dependent variable of pulmonary function, the data in Table II-6 result. Using models of restrictive and obstructive pulmonary disease, one would expect dust to produce the former and cigarettes the latter. If the table is examined, this indeed is the case. Dust causes the TLC, FVC, and $FEV_{1.0}$ all to be decreased, consistent with restrictive disease. Cigarette smoking causes a decrease in $FEV_{1.0}$ greater than the decrease in FVC and also causes an increase in RV and TLC. All these changes are compatible with obstructive disease.

Roentgenographic Changes

Chest roentgenograms of 784 granite shed workers were classified according to the UICC/Cincinnati classification, and their relationships to lifetime dust exposure, ventilatory function, and smoking habits of the workers were studied (29). Increase in dust exposure correlated with an increase in the profusion (number of opacities per unit area) and in the size of rounded opacities. Irregular opacities were related more to smoking than to dust. Forced vital capacity (FVC) was lower for people with abnormal roentgenograms and decreased with greater profusion. Residual volume (RV) increased with smoking but not with dust exposure. No trend was shown for total lung capacity (TLC). A dose-response curve for the effect of dust on ventilatory function and on roentgenograms showed that ventilatory capacity was affected earlier than the roentgenograms.

Table II-6

EFFECT OF AGE, HEIGHT, SMOKING, AND GRANITE DUST ON PULMONARY FUNCTIONS AS ESTABLISHED BY MULTIPLE REGRESSION ANALYSIS

Pulmonary Functions	Constant	Age	Height	Years Smoked	Dust-Years	Multiple R
FVC, ml	−4,073	−30	+55	−9	−1.6	0.73
$FEV_{1.0}$, ml	−2,100	−27	+40	−12	−1.6	0.71
FEV/FVC%	115	−0.07	−0.15	−0.13	−0.02	0.35
RV, ml	−1,316	+33	+10	+24	None	0.57
TLC, ml	−5,212	None	+65	+16	−1.8	0.46

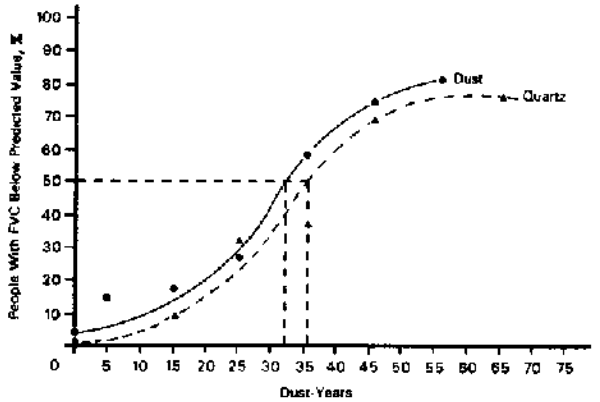

Figure II-1. Dose-response curve between granite dust and quartz and FVC.

Dose-Response for Dust on Roentgenograms

By plotting the percentage of people with opacities on their chest roentgenograms against an increasing dust exposure, one derives a dose-response which gives an estimate of the toxicity of the dust (Figure II-2). On the y axis are the percentages of people with radiographic opacities, and on the x axis is the increasing exposure expressed in dust-years. One dust-year is defined as an exposure for one year to the average dust concentration in the granite sheds—523 μg/cm of granite dust. The curve shows a plateau at around 30% of workers with exposure from 0-35 years. After that, the curve increases dramatically; at an exposure of 55 dust-years, 60% of the granite shed workers have opacities. The point at which 50% of the active workers are affected may be the best estimate of the toxicity of the dust and could serve as a means of comparison with other toxic materials. In this study the 50% dose-response is estimated at 46 dust-years of exposure.

Dose-Response for Roentgenograms and Ventilatory Function

In Figure II-3, we have plotted the proportion of people with abnormal roentgenograms against the dust exposure. The curve obtained from a similar study establishing dust effect on pulmonary function has been superimposed (28). The comparison is weakened by the fact that the curve starts at 30% for roentgenograms; nonetheless, by comparing the midpoint 50% of the two curves, it allows us to compare the effects.

While it takes 32.5 dust-years of exposure to affect the ventilatory function of 50% of the workers, it takes 46 dust-years to produce opacities on 50% of the worker's radiographs. There is a delay of about 13.5 years between the appearance of signs of dust effect on pulmonary functions and on roentgenograms. It can be argued that early detection of the effects of dust in *groups* of workers is better accomplished by pulmonary function tests than roentgenograms.

Morgan has criticized these results because only one reader read the x-ray films and because there was a high rate (~30%) of radiographic abnormalities at zero dust readings (17). Both criticisms are valid, but it should be pointed out that had the x-rays not been "over-read," the discrepancy between pulmonary function and x-ray results would have been even greater.

Morgan also minimized with some justification, the significance of 2 ml loss of ventilatory capacity for each dust-year of exposure (17). It is important to note, however, that cross-sectional studies have certain inherent weaknesses. Usually the population is a "survivor" population in that persons who contract serious pulmonary disease leave because they cannot work.

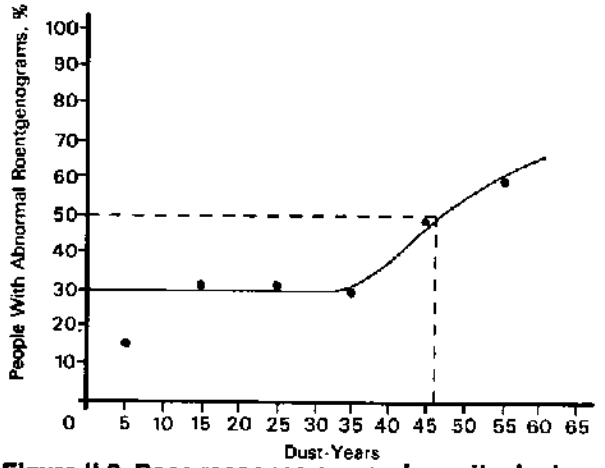

Figure II-2. Dose-response curve of granite dust on roentgenograms.

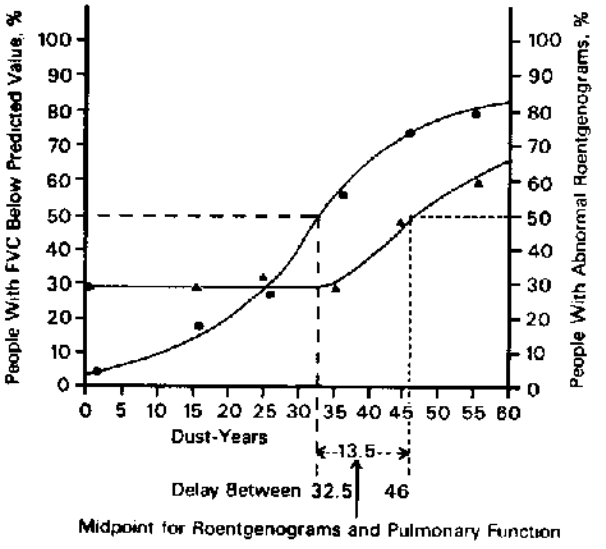

Figure II-3. Dose-response curve of dust on roentgenograms (triangles) and FVC (circles).

Also, since many of the independent variables of interest (age, years of smoking, years on job, dust-years) are inextricably correlated, statistical tests have difficulty in giving valid attribution to each.

These problems argue for the use of prospective studies which, among other advantages, provide the opportunity to assess the impact of dropouts from populations. The prospective model measures change in lung function over time and correlates the magnitude of change with the level of exposure. Instead of estimating changes from cross-sectional data, real changes are measured. Such a study was conducted by Musk et al. on the same Vermont granite workers (20). Pulmonary function studies were performed on 974 workers in Vermont granite sheds in 1974. Of these subjects, 668 had been studied 4 years earlier and had remained in jobs in which their exposure to granite dust was assumed not to have changed, based on dust concentrations measured during 1970. The yearly decrement in pulmonary function observed in the 668 granite shed workers was excessive (0.07 to 0.08 l/yr for FVC and 0.05 to 0.07 l/yr for FEV_1). This exceeded the expected decrement derived from several other occupational and population groups. Published cross-sectional and longitudinal data usually indicate a decrement of no more than 0.03 l/yr. in both FVC and FEV_1 (20). The observed decrements were independent of exposure groups and not accounted for by cigarette smoking. In 528 additional granite shed workers, decrements in ventilatory capacity had been measured for 1, 2, or 3 years and were consistently of the same order of magnitude. Dust concentrations within defined jobs and between granite sheds showed great variability. Despite this, a suggestive relationship between exposure and decrement in ventilatory function was demonstrated at the end of 2 years; however, at the end of 4 years, the relationship could no longer be shown with these exposure groupings. The difficulty in characterizing individual dust exposures and projecting dust concentrations for several years is considered to account for the absence of a dose-response relationship at the 4 year follow-up. The most important result of this study is evidence that previous estimates of lung volume loss among granite shed workers (2 ml/dust-year loss in lung capacity) were underestimates. It was concluded that present dust concentrations in Vermont granite sheds cause excessive deterioration of lung capacity.

A recently published article (9) challenged the findings of Musk et al. (20). This study concluded that technical deficiencies in the Musk study "led to exaggerated and erroneous estimates of loss." They correctly pointed out that the FVC's taken between 1970-1974 did not meet current ATS criteria. The "Harvard group" is currently reanalyzing their tracings and applying ATS criteria to determine how much, if any, their conclusions will be changed.

Discounting the "Harvard group" studies involving pulmonary function still leaves several studies, old and new, that suggest exposure to silica at levels below 100 $\mu g/m^3$ causes x-ray (7)(8)(21)(25)(29) and pathological changes (32).

Morgan presents a case for (what he calls)

industrial bronchitis; he considers it relatively harmless (18). Morgan suggests the observed losses could be a "non-specific response to dust alone"(17). This is certainly possible, but some recent evidence raises doubt about this being the complete explanation.

Vallyathan and Craighead examined the lung sections of 19 deceased Vermont granite workers (32). Examinations were accomplished with light and polarized light microscopy, scanning and backscattered electron microscopy, x-ray energy and x-ray fluorescent spectrometry, and x-ray crystallography. Medical histories, chest x-rays, and pulmonary function tests were available for all 19 indiviuals. Fifteen of the 19 had begun work after dust control (1937). All of the workers had *clinically normal* x-rays and pulmonary function tests, but dust-related fibrotic lesions were present in all lungs. None had confluent of severe fibrosis, but all had varying degrees of focal fibrosis. The quantity of silicon in each lesion was approximately the same as that found in lesions of persons with clinical silicosis. The authors concluded fibrosis can clearly begin before being detectable by standard clinical approaches (chest roentgenograms and pulmonary function tests).

All of the studies described in this section provide evidence for adverse pulmonary effects at levels of exposure above 10 mppcf or 0.1 mg/m^3. Some showed that foundry workers exposed to the equivalent of 0.05 mg/m^3 of quartz developed silicosis while those with less exposure did not (21). All the Vermont findings were seen with an average exposure around 0.05 mg/m^3 of quartz. It is possible, however, that since this was the average exposure, individuals whose exposure exceeded this level accounted for the noted effects. [The "no effect" level was probably below 0.05 mg/m^3, but available data did not allow accurate determinations.]

ESTIMATE OF POPULATION AT RISK

In the United States, occupational exposure to silica occurs in several large categories of industry; in particular, mining, manufacturing, construction, and agriculture. The U.S. Bureau of Census Statistical Abstracts for 1971 provides the statistics in Table II-7.

A recent NIOSH estimate reveals that a more accurate number for metal mining is 300,000 instead of 76,000 (16). This Table also leaves out 2.5 million agricultural workers; 0.6 million workers in the chemical and allied products industry; and 0.6 million workers in heavy construction who may be at risk. There are many other miscellaneous industries in which silica exposure may take place.

The current state of our knowledge does not permit accurate estimates of the incidence and prevalence of silicosis. Systematic studies of all the above industries would need to be conducted. If either extensive information on silica exposures or health effects were available, the magnitude of the problem could be estimated. This lack of information is not unique to the silica problem. Attempts to estimate the prevalence of all of the occupational diseases results in similar frustration.

Table II-7

EMPLOYMENT IN INDUSTRIES HAVING POTENTIAL EXPOSURE TO FREE SILICA 1970

Metal mining	76,000
Coal mining	125,000
Nonmetallic minerals (except fuels)	95,000
Stone, clay, and glass products	507,000
Iron and steel foundries	188,000
Nonferrous foundries	69,000
Cement production	3,212,836
Total	4,272,836

PATHOLOGY

It is beyond the scope of this chapter to review the details contained in many sources. The interested reader is referred to Parkes (22), Morgan and Seaton (19), and books on the pathology of the lung.

Findings on Gross Examination

Fibrous adhesions are commonly found in the pleural cavity with plaques visible over the pleural surface. The hilar nodes are frequently enlarged and sometimes calcified. The lung may be hyperpigmented. On cut sections, the lung reveals grayish nodules, usually in the superior-posterior aspects of the respective lobes. These can range in size from small to large (3 mm to 1 cm) and can also vary in profusion. Nodules in both the lung parenchyma and in the lymph

nodes show a whorled appearance (Figure II-4). In severe cases, nodules may coalesce and be associated with tuberculous infection or necrosis and cavitation (Figure II-5). Associated with this coalescence is prominent contraction of the upper lobe(s)—a striking radiographic feature of the advanced disease. Evidence of congestive heart failure may exist with corresponding enlargement of the right side of the heart.

Microscopic Findings

The classical silicotic nodule is usually located in the area of the respiratory bronchiole. The nodule is composed of reticulin fibers in the periphery and collagen fibers in the center. Fibroblastic activity is usually evident around the periphery of the concentric lesion (Figure II-6).

The airways and blood vessels are frequently destroyed by being entrapped in the fibrotic nodule. Silica particles are difficult to identify in tissue sections by polarized light microscopy. Therefore, special techniques involving high resolution microscopy are required. It appears that the extent of the lesion bears little association with the amount of silica present.

In cases of massive fibrosis the normal pulmonary structure may be distorted or destroyed. When there is coexistent mycobacterial infection, the characteristic histological feature of tuberculosis may not be observed. It is, therefore, important to stain and/or culture the lesion for acid fast bacilli in all cases showing massive fibrosis.

Acute Silicosis

Since this disease develops so rapidly, the pathologic picture is very different from the chronic form. Please see the Acute Silicosis monograph, page 239.

Pathogenesis

Much experimental work has been done to elucidate the mechanism of silica's action. The work of Heppleston has been particularly noteworthy in revealing several important steps (11):

1. Destruction of macrophages by the ingested silica particles.
2. The production of more macrophages to continue ingesting silica particles.
3. Stimulation of collagen formation.
4. Hyalinization of the collagen.

After lung macrophages have been damaged, additional macrophages appear to engulf liberated silica particles and they die too. Then a reticulin network is formed. Finally, the collagen is laid down and is hyalinized. There is evidence that silica somehow stimulates lipid factors which in turn promote the production of more macrophages. A nonlipid material is also produced by macrophage death; this material stimulates fibroblasts to produce collagen. The hyalinization step may be related to immunoglobulins.

While the information described above has been derived primarily from animal studies or studies of cell cultures, the implications for man are fascinating. For example, we currently measure silica exposure primarily as a mass measurement, partially ignoring the distribution of particle size. Depending on particle size, there can be many more or fewer particles for the same mass. Does the macrophage recognize a particle's size? Is the number present more impor-

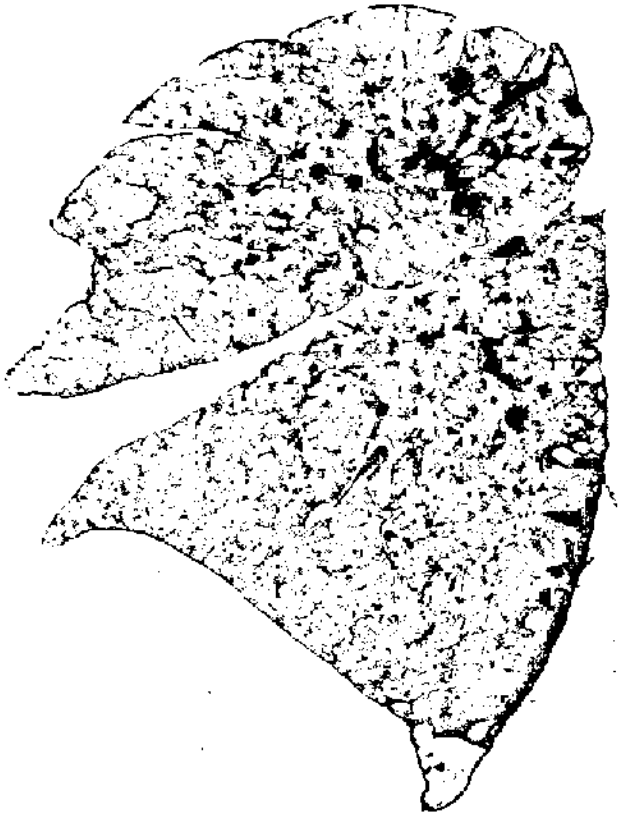

Figure II-4. *Simple Silicosis*—The whole lung section shows numerous discrete rounded nodules, typical of simple silicosis. The nodules are more numerous in the upper lobes and have pale centers with darkly pigmented outer borders.

Figure II-5. ***Conglomerate Silicosis (progressive massive fibrosis PMF)*****—The whole lung section shows a large central area of fibrosis composed of multiple coalescent rounded nodules with pale centers typical of conglomerate silicosis. The patient was a coal miner and the lung also shows the macular lesions of coal workers' pneumoconiosis.**

tant? Macrophages engulf silica particles of respirable size; could there be differential treatment based on particle size? We know the same silica mass might be associated with differing numbers of particles because of different size and potentially have an effect on varying the number of macrophages. The answers are important so as to know the most meaningful way of assessing exposure for humans facing the risk. Although differences in gravimetric and impinger sampling are known to be poorly correlated, differences in particle size and surface properties are clearly important in terms of biological effects.

It is not fully understood why some individuals develop massive fibrosis while others do not. Several factors may be important. The dose of silica is, of course, important and relates to both the induction period as well as the severity of disease. Cigarette smoking is also an important factor and will be considered later. Immunological status is another important factor. Persons with rheumatoid arthritis react differently to silica exposure both in quality and quantity. Other immunological host factors are probably important too. Concurrent infection is likewise important. The relationship to mycobacterial infection has been known for years. Other microbiological infections are likely to be important as well.

Acute silicosis is primarily related to massive silica exposure. The general pathological findings that have been described differ from chronic silicosis. This may relate to the usual pulmonary defense mechanisms being overwhelmed.

CLINICAL DESCRIPTION

Symptoms

Dry cough may be an early manifestation of silicosis. As the disease advances, the cough may become more prolonged and distressing and be associated with sputum production. Hemoptysis is not common. Breathlessness is a common symptom as the disease progresses. Initially, shortness of breath occurs during heavy exercise: less and less exercise is required to induce dyspnea as the disease progresses. Wheezing is not common unless the disease is accompanied by asthma, or if there is significant large airway distortion (such as the distortion occasionally seen in PMF). There is a variable association between shortness of breath and either pulmonary function results or opacities seen on chest x-ray. Usually general health is not impaired in simple silicosis. Pneumothorax is common in advanced stages and respiratory failure is an important consequence of progressive massive fibrosis (PMF). If silicosis is associated with tuberculosis or congestive heart failure, general health obviously deteriorates. In advanced silicosis, respiratory failure precedes cor pulmonale and ultimately, congestive heart failure.

Physical Signs

In late stages, physical findings similar to those found in other chronic fibrotic and obstructive lung diseases occur. Bronchitic symptoms and signs are common. Hypoxemia is usually present in advanced cases even without cyanosis or congestive heart failure. The ability to expand the chest is not impaired until late in

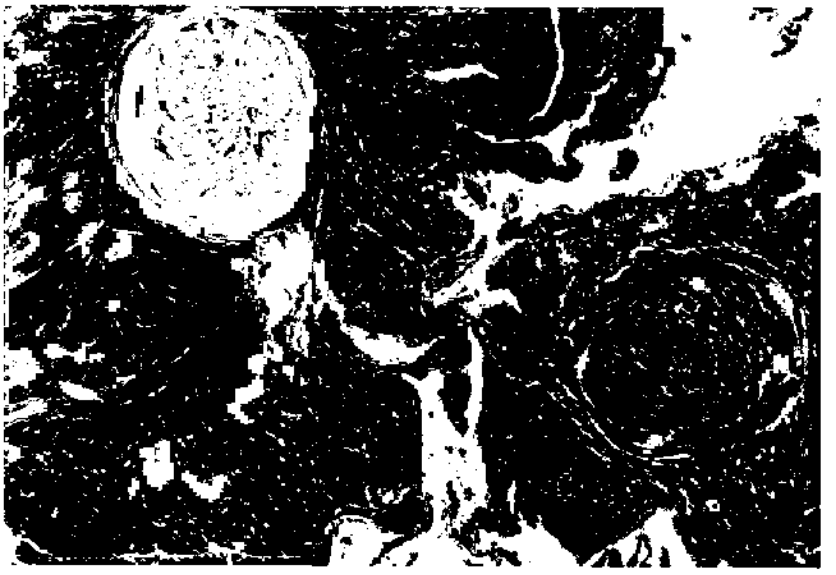

Figure II-6. ***Silicotic Nodules*****—Three silicotic nodules in a coal worker's lung. Note concentric arrangement of collagen fibers. Pigmented macrophages are present in the silicotic center and in stellate mantle surrounding the lesion. The paucity of pigment in the remainder of lesion is characteristic. Hematoxylin and Eosin × 200.**

the disease. For the most part, percussion is not affected until late in the disease, and this is usually related to pleural fibrosis. Unless the disease is complicated by chronic obstructive pulmonary disease or tuberculosis, adventitous sounds are not commonly heard on auscultation. In the late stages of silicosis, signs of right ventricular hypertrophy and eventually right heart failure will appear, generating cor pulmonale and ultimately congestive heart failure.

Lung Function

Until the disease is fairly far advanced, effects on pulmonary function are minimal to moderate. There may be some slight reduction in vital capacity and some depression of arterial oxygen tension (hypoxia), particularly on exercise. It is difficult to characterize individual cases, however, as one rarely has baseline pulmonary function values available for comparing the current state of an individual.

The correlation of pulmonary function tests with radiographic classification is weak on a clinical basis, unless progressive massive fibrosis is present. The disease is manifested by a restrictive pulmonary pattern on pulmonary function tests. For example, TLC, VC, and RV are all likely to be decreased. In the absence of massive fibrosis, oxygen desaturation is rare at rest or during mild exercise but may be observed on greater effort in some individuals. Although defects in diffusing capacity are not common, as the disease progresses there may be some effect on gas transfer.

Radiographic Appearance

The most common x-ray evidence of silicosis is the appearance of small discrete round opacities, which usually occur in the upper halves of the lung fields and vary in size from approximately 1 - 3 mm in diameter (Figure II-7). As the disease progresses, the opacities increase in size and number and begin to affect the lower parts of the lung as well as the upper lung fields. Nodular coalescence, upper lobe contraction, hilar retraction toward the apex, and basilar emphysema form an important diagnostic radiographic pattern in complicated silicosis (Figure II-8). With further progression of the disease, larger opacities are seen which could be described as conglomerate masses. In some severe cases these opacities can occupy the greater part of the lung field on a radiograph. The progression is variable in speed and obviously relates to dust exposure and host factors. Sudden progression or sudden worsening of the x-ray picture often

Figure II-7. *Simple Silicosis*-Posteroanterior radiograph showing multiple discrete rounded nodules (1-3 mm in diameter) primarily in the upper mid-zones.

heralds the onset of a superimposed mycobacterial or fungal infection. In some cases, the sudden worsening of the disease may be associated with the rheumatoid factor being present. This is referred to as Caplan's syndrome and may be associated as well with the onset of rheumatoid arthritis (3). Calcification can sometimes complicate the picture on x-ray. This calcification can not only affect the silicotic nodule itself, but can also affect the fibrous lymph nodes. Frequently, lymph node calcification is characterized by a very thin, dense ring of calcification known as eggshell calcification. Pleural fibrosis, while uncommon in early stages of silicosis, may occur—particularly in advanced cases.

Other Tests

Ordinarily, if other clinical tools are available, acquisition of pulmonary tissue is not necessary to make the diagnosis of silicosis. In the face of rapidly advancing silicosis, it is important to acquire sputum samples for culture and to rule out mycobacterial and fungal infections. Tests for rheumatoid and antinuclear factors should be performed. Electrocardiography may be useful in advanced cases to establish or refute the presence of right sided heart failure.

Clinical Complication

From the previous discussion, one should also think of the possibility of superimposed mycobacterial infection. In advanced cases, the possibility of cor pulmonale exists. Bronchitis is a frequent accompaniment of silicosis and may be the result of concurrent cigarette smoking. It is not certain whether silica itself causes bronchitis. Emphysema is sometimes seen in lungs of silicotics; it may be more related to cigarette smoking than to dust exposure, although the basilar emphysema characteristic of advanced silicosis is probably specifically related to the upper lobe contracting from massive fibrosis. The rapid progression of silicosis in the presence of rheumatoid arthritis or the rheumatoid factor has already been mentioned and should be kept in mind. Bronchial carcinoma is sometimes seen in individuals with silicotic lungs, but with our

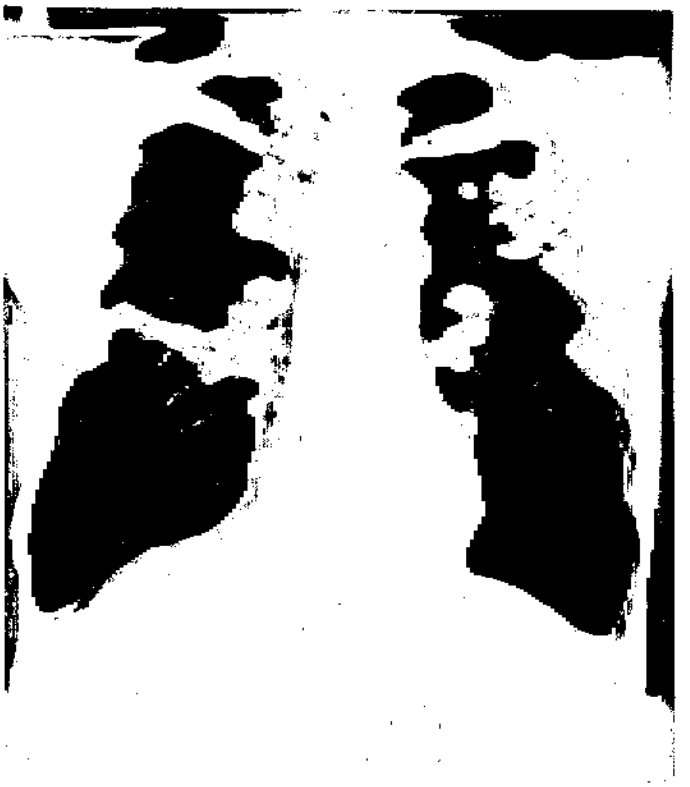

Figure II-8. ***Progressive Massive Fibrosis (PMF)*— Posteroanterior radiograph showing a significant loss of lung parenchyma. Basilar bullae, bilateral upper lobe conglomerate lesions, with compensatory emphysema and elevated hila are seen.**

present knowledge, there is little evidence to suggest it occurs more frequently in silicotics than in nonsilicotics (33). In a miner with lung cancer, the possibility of exposure to radon daughter products should be entertained.

Treatment

There is no specific treatment for silicosis. Corticosteroids do not appear to affect the progression of the disease. Detecting and treating concurrent tuberculosis is essential. In addition, appropriate treatment for congestive heart failure should be begun if this complication exists.

DIAGNOSTIC CRITERIA

In the presence of an adequate occupational history revealing work exposure to silica, the diagnosis of simple silicosis is usually straightforward. Roentgenographic changes are relied on by most clinicians for the diagnosis. While the argument is frequently made that changes on chest x-rays precede other clinical findings, there is now some reason to doubt that. Mild restrictive disease detected by pulmonary function may occur in workers with early x-ray manifestations. It must be remembered by the clinician that an FVC of $\geq$80% predicted is considered normal. On the other hand, if there are 100 men who have early x-ray evidence of silicosis whose average FVC is 90% of predicted, this comprises an abnormal population. Clinicians are usually at a disadvantage seeing individuals one at a time, and they frequently have no baseline chest x-ray or pulmonary function test results for comparing current clinical findings.

Given an individual with exposure to silica, a mild restrictive defect would suggest early silicosis; likewise small round opacities on the chest x-ray would support the diagnosis. With minimal loss of pulmonary function or minimal

chest x-ray abnormality, symptoms such as shortness of breath are unlikely to be a clinical feature unless associated with underlying chronic airways disease.

When it comes to complicated silicosis, PMF, or Caplan's syndrome, the diagnosis is more difficult. The possibility of a lung tumor or tuberculosis must be considered. Bacteriological testing of sputum usually reveals mycobacterium tuberculosis. A lung biopsy may be necessary to diagnose carcinoma.

Abnormalities of diffusing capacity are not common or profound in early silicosis. Similarly, clubbing and physical signs in the chest, while not ruling out silicosis, suggest other diseases. Hyperinflation, reduced breath sounds, prolonged expiratory phase, and reduced expansion of the chest are among the most common physical findings in advanced silicosis.

Acute silicosis should be suspected in a worker with massive exposure to silica, e.g., an unprotected sandblaster. Mycobacterial infection occurs in about one-quarter of these cases. Symptoms of progressive shortness of breath are common. Weakness, weight loss, diffuse rales, and even cyanosis can be seen. Usually there is evidence of massive disease on chest x-ray, with the diaphragm frequently being high. Pulmonary function is severely compromised.

PREVENTION

The theoretical approach to preventing silicosis is simple: reduce airborne dust concentrations to safe levels. This implies cognizance of safe levels and feasible utilization of technical means to control dust exposures. While there remain some questions about the safe level, technology is available to achieve at least the 50 $\mu g/m^3$ level in most situations. The ACGIH *Industrial Ventilation Manual* provides ventilation designs appropriate for foundries, ceramics industries, crushing, grinding, and screening operations. Since sandblasting generates so much dust and is so difficult to control, and since other substitute techniques are available, this practice should be prohibited.

Other existing techniques such as enclosure, isolation, local exhaust or dilution ventilation, and wet processes are highly effective methods of controlling dust exposure. As a last resort, in unusual and temporary situations, protective respirators can be employed effectively.

Since perfect knowledge does not exist as to safe levels of exposure, medical surveillance techniques should be continued, not as control methods, but to verify the adequacy of the standard and the meeting of the standard. Both pulmonary function tests and chest x-rays should be employed at appropriate intervals. [NIOSH recommends chest x-rays and pulmonary function studies be utilized prior to employment placement and at least once each 3 years thereafter. (Criteria for Recommended Exposure to Crystalline Silica, NIOSH Publication No. 75-120)]

RESEARCH NEEDS

Research should be directed toward the elimination of silicosis. In addition, questions concerning the subclinical effects of silica exposure must be answered, e.g., at what level of exposure do fibrotic lesions appear in the lung? If exposure at that point is stopped, do the lesions remain static or do they progress?

Aside from the questions concerning health effects, questions about environmental assessment are important. For example, does mass respirable sampling provide a better or worse way to assess biologically meaningful exposure? Are there ways to measure and compile worker exposure to silica so that accurate lifetime exposure can be assessed? Is the effect of silica modified (antagonized or enhanced) by concomitant exposure to other dust?

The role of animal, tissue, and cell studies must also be considered. Are there nonhuman tests that allow accurate prediction of human toxicity? Are there ways of simulating 40-year human exposures in animals?

Engineering approaches to problem solution need to be considered. Are there better ways to process these materials so as to minimize dust exposure? Are better engineering techniques available to control existing processes?

Policy issues likewise need research. What are the best ways to create a climate that facilitates both occupational health and productivity? Are there economic incentives that would promote this? Is regulation and enforcement the best method?

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ACUTE SILICOSIS

Daniel E. Banks

Acute Silicosis, or silico-proteinosis, is a rare presentation of silica-induced lung disease (Table II-8) (10). This form of silicosis is associated with massive exposures to respirable size particles of high free crystalline silica content over a short period of time. Invariably, this disease is untreatable with a lethal outcome. Betts, in 1900, first described this acute presentation of silicosis in the United States and his description is relevant today (4):

> After coughing has continued for some time there will be. . . loss of appetite, loss of weight and shortness of breath, the respirations running as high as 38 to 42 breaths per minute on the slightest exertion. As the weeks pass, the patient suffers general malaise and soon finds it impossible to get about. . . in (the disease's) later stages, the temperature may rise to 102 or 104. . .

Early reports related acute silicosis to miliary tuberculosis because of the similar rapid downhill course (11). Other early reports described similar presentations in workers who mixed silica and alkali in the production of industrial abrasives (7)(9). Postmortem examination of the lung in these cases revealed (6):

> The presence in every alveolus of large amounts of pink staining fluid, (and) an extreme grade of edema with a very high protein content. Another interesting finding is the presence of epithelium in all the alveoli.

The pathologic and radiographic features of acute silicosis were fully described in 1969. Buechner and Ansari described 4 sandblasters with a mean silica dust exposure of only 4 years, and relentlessly progressive dyspnea, cough, fatigue, weight loss, and pleuritic chest pain (5). Despite the prompt diagnosis and treatment of tuberculosis in 3 cases and appropriate therapy of suspected tuberculosis in the 4th, mean survival time from onset of symptoms was only 7.5 months. All died from respiratory failure.

In all, chest radiographs showed air bronchograms and an alveolar filling pattern. Each man had significant restriction of lung volumes.

Pathologically, the alveolar septae were thickened and infiltrated with mononuclear cells. The lungs were firm and heavy with a pinkish, proteinaceous PAS positive staining alveolar exudate (Figure II-9) identical to that seen in idiopathic alveolar proteinosis and has resulted in the use of the term silico-proteinosis. Typical silicotic nodules were seen in 2 cases, but these were smaller than nodules noted in the chronic form of silicosis.

More recently, Suratt et al reported acute silicosis in 4 tombstone sandblasters (12). These cases were similar to those above in both mean duration of exposure (4 years) and mean survival from onset of symptoms (6 months). Two had pneumothoraxes complicating their clinical course. One developed focal glomerulonephritis and another systemic lupus erythematosis (both had positive anti-nuclear antibodies, a common finding in sandblasters' silicosis (8)). All 4 had a restrictive impairment on spirometry with a significant decrease in diffusing capacity. No chest radiographs revealed the pattern of silico-proteinosis described above. Instead, 2 showed bilateral upper lobe opacities, and 2 showed a reticulonodular pattern.

Despite the absence of a radiograph appearance of silico-proteinosis, postmortem lung examinations in two sandblasters revealed a PAS positive exudate filling the alveoli. Discrete hyalinized and cellular nodules were present in alveolar walls and within the wall of small pulmonary blood vessels. The authors considered these pathologic changes as intermediate between silico-proteinosis and chronic nodular silicosis.

Evidently, then, there is variability in chest radiographs of workers with massive silica dust exposures over a short period. Classically, alveolar infiltrates with air bronchograms are present and correlate with PAS positive proteinaceous

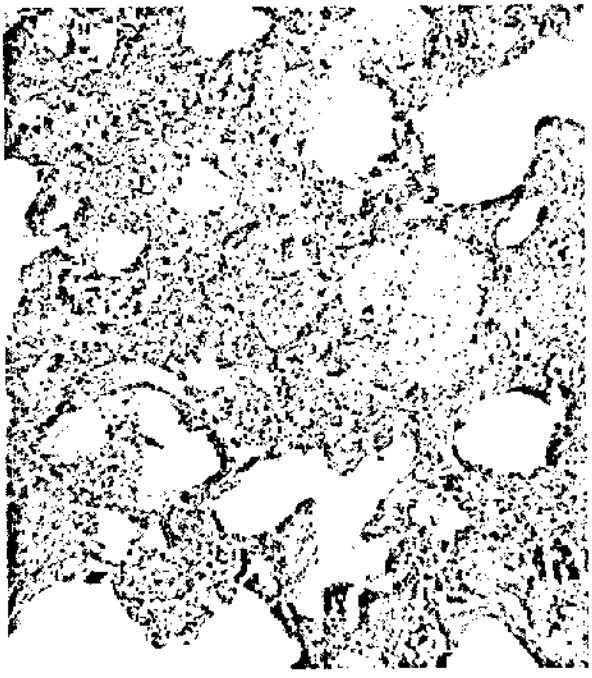

Figure II-9. Photomicrograph (Hematoxylin & Eosin, 50X) from a lung biopsy specimen of a surface coal miner driller who died 26 months after the diagnosis of acute silicosis was made. The photomicrograph shows distorted pulmonary parenchyma, interstitial inflammation and fibrosis and filling of the alveolar spaces with a relatively acellular material with some epithelial cells present. This material gave a positive reaction when stained with Periodic acid-Schiff reagent. (3)

Figure II-10. Chest roentgenogram of a silica flour worker showing diffuse small opacities with a lower lobe predominance, a large opacity in the right mid-lung field, and a right sided air-bronchogram.

alveolar exudate. Alternatively, simple nodular silicosis, which rapidly progresses to progressive massive fibrosis, may be present in those with short-term massive exposures.

Silicotics are particularly prone to mycobacterial infections. Bailey et al found 22 of 83 silicotic sandblasters in New Orleans developed complicating mycobacterial infections, both with typical and atypical (M. kansasii and M. intracellulare) organisms (1). All 18 (diagnosed antemortem) converted positive sputum to negative status under treatment. Control of tuberculosis did not prevent progressive respiratory impairment and 4 of these patients died of respiratory failure. Of the total of 8 deaths in the entire group, 3 occurred in sandblasters with silico-proteinosis.

Recently NIOSH representatives evaluated the health of miners and mill workers at 2 silica flour mills in Southern Illinois (2). Of 61 workers and ex-workers with 1 or more years of exposure to silica, 16 (26%) developed simple silicosis and 7 (11%) had conglomerate silicosis. Four of these 7 had 6 or less years of silica dust exposure. One workman developed the radiographic picture of silico-proteinosis (associated with a mid-lung conglomerate lesion) after only 2-½ years of dust exposure (Figure II-10).

As we enter the decade of the 1980's, it is vexing to acknowledge that silicosis—perhaps the oldest occupationally related disease—exists despite sophisticated control technology. Sandblasters and silica flour mill workers are 2 groups still at high risk of developing acute silicosis. Adequate compliance with current standards and continued surveillance of workers exposed to free silica is essential to prevent severe health effects.

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Table II-8

IMPORTANT CHARACTERISTICS OF THE DIFFERENT CLINICAL FORMS OF SILICOSIS (8)

Clinical Type	Pathology	Exposure Levels	% Silica in Dust	Usual Exposure Duration	Time from 1st Exposure to Disease Development (X-ray Findings)
Chronic ("Classical") Silicosis	Fibrotic nodules located near respiratory bronchioles	"moderate"	<30%	20-40 yrs.	20 yrs.
Accelerated Silicosis	Fibrotic nodules smaller than those in "classical" silicosis PMF in mid-zones	"moderate-high"	47-84%	5-15 yrs.	4-8 yrs.
"Silico-proteinosis" Acute Silicosis	Diffuse interstitial fibrosis and alveolar lipo-proteinosis	"heavy"	90-100%	3-6 yrs.	1-3 yrs.

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