

FILE NAME: New York State & NY Times (NY)

DATE: 1938

DOC#: NY081

DOCUMENT DESCRIPTION: NY Dept of Labor Special Bulletin - Silicosis & Its Prevention

Special Bulletin No. 198
1938



SILICOSIS AND ITS PREVENTION

By

Adelaide Ross Smith, M.D.

Division of Industrial Hygiene



NEW YORK STATE DEPARTMENT OF LABOR
STATE OFFICE BUILDING
ALBANY—NEW YORK CITY

3-23-38-1000 (6-9476)

VORWALD COLL
BOX 42

Price 35 Cents

State of New York

DEPARTMENT OF LABOR

Albany Office
State Office Building

New York Office
80 Centre St.

ELMER F. ANDREWS

Industrial Commissioner

MICHAEL J. MURPHY

Deputy Industrial Commissioner

ROSE SCHNEIDERMAN

Secretary



THE INDUSTRIAL BOARD

Richard J. Cullen, *Chairman*

Edward W. Edwards

John J. Carroll

Nelle Swartz

James A. Corcoran



THE BOARD OF STANDARDS AND APPEALS

William J. Picard, *Chairman*

Joseph P. Craugh

Raymond M. Fisher

TABLE OF CONTENTS

	Page
Introduction.....	7
Chapter I — Significance of Silicosis.....	9
Pneumoconiosis.....	9
Definition of silicosis.....	9
Public health aspect of silicosis.....	9
Number exposed.....	9
Silicosis and tuberculosis.....	9
England and Wales.....	9
South African miners.....	9
Life insurance statistics, U. S.....	10
Anthracite miners.....	10
Ax grinders.....	10
Granite workers.....	10
Silica exposure and high mortality from other causes.....	11
Chapter II — Cause of Silicosis.....	12
Chemical properties of silica.....	12
Structure.....	12
Thermal properties.....	12
Solubility.....	12
Occurrence of silica in nature.....	12
Free silica.....	12
Silicates.....	13
Industrial uses of silica.....	13
Silicosis-hazardous industries.....	15
Chapter III—Characteristics of Silicosis.....	16
Factors in the development of silicosis.....	16
Type of silica in dust.....	16
Per cent of free silica in dust.....	16
Size of dust particles.....	16
Length of exposure to dust.....	16
Degree of dustiness.....	16
Standards of permissible dustiness.....	17
Individual susceptibility.....	17
Action of silica on body tissues — experimental studies.....	18
Toxicity.....	18
Solubility.....	18
As a colloid.....	18
Effect on tubercle bacilli.....	18
Behavior of silica in the body.....	18
Urinary excretion.....	18
Silica in blood and sputum.....	19
Reaction of peritoneal tissue to injection of silica and other dusts.....	19
Reaction of lymph nodes to silica and other dusts.....	19
Behavior of silica in the lungs.....	19
The dust disposal mechanism of the lungs.....	19
The lymphatic filter and the pulmonary lymphatic system.....	20
"Dust" cells.....	20
The pathology of silicosis.....	20
Growth of connective tissue.....	20
The silicotic nodule.....	21
Tuberculosis with silicosis.....	22
Post mortem appearance of silicotic lungs.....	24
Examination of lung tissue for silica.....	25
Symptoms and course of silicosis.....	25
Rate of development.....	25
Predisposing factors.....	25

ILLUSTRATIONS

Frontis-piece Granite surfacing—with and without dust control.

Fig.

1. Microphotograph of section of lung tissue showing silicotic nodules.
2. Silico-Tuberculosis cross-section of lung showing extensive consolidation from silicosis and tuberculosis with marked emphysema.
3. Third stage silicosis in a blaster—exposure 23 years.
4. Silicosis with tuberculosis in a rock driller—exposure 31 years.
5. Advanced silicosis with tuberculosis in a rock driller—exposure 31 years.
6. Rock drilling—no dust control. Serious silicosis hazard.
7. Rock drilling—dust removed. Silicosis hazard under control.

INTRODUCTION

Disease of the lungs due to the inhalation of dust has recently assumed a position of outstanding interest in the field of industrial hygiene. This is in part due to the light of publicity which has played upon the subject in recent years but it is also due to a realization, somewhat tardy, of the real importance of the subject. There can be no doubt whatever that the effects of dust play a far-reaching role in the health of the industrial population.

Articles and reports of investigations in this field have so flooded the press, both popular and medical, that it would seem unnecessary to add anything further. However, repeated requests for information on silicosis are made to the Division of Industrial Hygiene and it is with the idea of furnishing a reference manual which in a convenient form will cover the essentials that this review of silicosis has been prepared.

ADELAIDE ROSS SMITH, M.D.



Granite Surfacing—With and Without Dust Control

CHAPTER I

SIGNIFICANCE OF SILICOSIS

Pneumoconiosis

Originally the word pneumoconiosis was used for lung disease caused by dust. There were subdivisions, as anthracosis, siderosis, chalicosis and silicosis to denote the dust concerned, i.e., coal, iron, stone and silica, respectively, but no distinction in their effects was recognized. Gradually it became apparent that one dust, silica, was far more injurious than others and finally it was realized that when the other dusts had apparently produced lung disease it was due to the presence of silica in them. Silicosis according to Gardner¹ is, with the exception of asbestosis, the only true pneumoconiosis. Dusts which do not contain silica will cause changes in the lungs recognizable upon X-ray, but they are not progressive changes and of themselves they do not produce disabling disease.

Definition of Silicosis

Silicosis is defined by the American Public Health Association as follows:

"Silicosis is a disease due to breathing air containing silica (SiO_2) characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present) and by characteristic X-ray findings." It is more or less arbitrarily divided into three stages.

Public Health Aspect of Silicosis

Before describing the properties of silica itself and the pathology of the disease it will perhaps give a better understanding of its importance to consider it from the public health aspect in terms of the number exposed to the possible hazard of contracting it and the effect of exposure upon mortality statistics.

Number Exposed to Silica Dust in United States and New York State.

Lanza and Vane² estimate that there are approximately 450,000 workers exposed to silica dust in the chief mining, quarrying and manufacturing industries of the country with a good many more thousand previously exposed but now engaged in other work. This is probably a conservative estimate.

In New York State the estimate of the number of people exposed to definitely injurious dust is tentatively put at 7,500; with a middle group of between 13,000 and 14,000 exposed to dust of a moderately harmful type, from the aspect of both concentration and nature; and a third group of about 35,000 exposed to a questionable dust hazard, the extent of which cannot at the moment be appraised.

Silicosis and Tuberculosis

All analyses of mortality statistics among those exposed to silica point to one thing: The increased frequency of deaths from tuberculosis among them.

This practically unvarying association with tuberculosis constitutes by far the most serious feature of the disease; one which has been recognized for hundreds of years. Modern statistical evidence of the association is abundant.

England and Wales

The following table presents the standardized respiratory tuberculosis mortality rates for the years 1921-1923 for England and Wales from the report of the Registrar General.³

Occupation	Rate per 100,000
Tin and copper miners, all underground workers.....	1,886
Tin and copper miners (only).....	1,323
Cutlery grinders	1,178
Metal grinders.....	637
Stone workers and slate workers.....	512
Potters	411
Barmen	402
File cutters	365
All occupied and retired males	150

In this table are presented the eight occupations having the highest mortality rates from tuberculosis as compared with the rate for all occupied and retired males. Of the eight occupations listed, seven are associated with the inhalation of quartz dust. It will be observed that tin and copper underground miners have a tuberculosis rate more than 12 times that for all occupied or retired males; and file cutters who work on sandstone grinding wheels, largely composed of quartz, have a tuberculosis rate more than twice that of all occupied and retired males. Silicosis with recognized tuberculosis, accounted for 68-69 per cent of 543 deaths in a group of Rand miners reported by Irvine, Simson and Strachan.⁴

Life Insurance Statistics

In a study made by 12 life insurance companies in this country in 1928 quoted by Lanza and Vane,² "the occupations showing the highest ratios of actual to expected deaths from tuberculosis were, in order: Underground lead and zinc miners, granite and sandstone cutters, copper miners and gold and silver miners.

"All of these workers were exposed to a serious silica hazard. The ratio of actual to expected deaths from tuberculosis were respectively 1,833 per cent, 976 per cent, 913 per cent and 804 per cent. There were actually 60 deaths from tuberculosis among these men as against six expected. Tuberculosis was responsible for one-half of all the deaths of lead and zinc miners, 29 per cent of the deaths of copper miners, and 20 per cent of those among gold and silver miners. In all these three mining classes, deaths from tuberculosis exceeded deaths from accidental injuries. Among iron miners who are, on the whole, probably less exposed to silica dust than are the other mining classes mentioned, we find that the ratio of actual to expected tuberculosis deaths is only 260 per cent. There were 16 deaths from tuberculosis among granite cutters as against 1.7 expected."

Anthracite Miners

In a study of anthraco-silicosis among hard coal miners made by the United States Public Health Service,⁶ the control group, not exposed to dust, was found to have a tuberculosis rate of less than one per cent, whereas among those with early and advanced anthraco-silicosis it was 15.3 and 43 per cent respectively.

Ax-Grinders

Winslow and Greenburg⁷ quote an interesting study of tuberculosis in an ax-grinding plant in Connecticut made by Drury which covered the death certificates of the workers for the previous 20 years. Sandstone wheels with a high percentage of quartz had been used. The rate among grinders and polishers was 1,900 per 100,000. Among other persons in the mill it was 160, and for the State of Connecticut as a whole 150 per 100,000.

Granite Workers

In the U. S. Public Health Service study of granite workers it was found that tuberculosis was recorded as the cause in 65 per cent of 631 deaths among cutters, occurring between 1900 and 1925. During the period of the study the death rate for tuberculosis was 14 per 1,000 granite workers,

a rate, as the authors point out, in excess of the death rate for all causes in the general population.⁵

Illustrations of the high death rate from tuberculosis in silica-dusty industries can be multiplied many times over. While it is possible, in fact probable, that many cases of death due to silicosis alone or with other complications have been mistakenly reported as tuberculosis, errors could not begin to account for the excess tuberculosis rate. Where it is possible to check diagnoses carefully by sputum examination as in tuberculosis sanatoria the association with a history of exposure to silica dust is still apparent. In all reports of autopsies upon cases of silicosis, the number showing tuberculosis has been outstanding.

Silica Exposure and High Mortality from Other Causes

It seems possible in view of the statistical analysis made by Collis and Yule⁶ that the presence of silica in the body in undue amounts not only predisposes to pulmonary tuberculosis but has an unfavorable effect on other organs. Comparing mortality statistics in a large group of industrial workers exposed to silica dust with another industrial group not so exposed and with the general population, they found definitely increased rates for diseases of the heart, gastro-intestinal tract and kidneys among the former. So striking was the difference that they declare silica to be a general body poison as is lead. Gardner⁷, however, does not find such a conclusion justified by his experimental studies.

CHAPTER II

CAUSE OF SILICOSIS

Chemical Properties of Silica^{10, 11, 12}

Silica has the chemical formula SiO_2 , indicating that it is a combination of the element silicon (Si) and oxygen (O). It occurs by itself in the "free" form, and as such, is variously known as silicon dioxide, or "free" silica. In combination with other elements, it is known as "combined silica," but it is free silica which concerns us especially because in this form it causes more damage to the lungs than when it is in the combined state.

Structure

Free silica, silicon dioxide, may be either crystalline or noncrystalline (amorphous) in structure. Crystalline silica, which is simply quartz, is a white or colorless, extremely hard substance with a specific gravity of 2.66. When heated for a long time at about $1,000^\circ \text{C}$. it forms a second variety known as tridymite with a specific gravity of 2.33. A third variety, cristobalite, specific gravity 2.34, occurs when quartz is heated for periods not long enough to form tridymite. Non-crystalline (amorphous) silica occurs as a fine white powder.

Thermal Properties

Silica assumes the vitreous, molten form more easily than any other mineral substance, melting to colorless quartz glass in the oxyhydrogen blowpipe beginning at about $1,600^\circ \text{C}$. The coefficient of thermal expansion of quartz glass is very small, so that it possesses in a high degree the property of being able to withstand rapid cooling without cracking.

Solubility

Silica is reduced by carbon in the electric furnace to form silicon carbide, and by magnesium to amorphous silicon. Crystalline and vitreous silica are insoluble in water and in all acids except hydrofluoric, but they are slowly soluble in aqueous solutions of alkaline hydroxides and carbonates. Fused silica is readily soluble in phosphoric acid and the alkalis.

Silica is chemically inactive at ordinary temperatures but at high ones acts as an acid anhydride and combines with the bases and many metallic oxides to form silicates. When soluble alkaline silicates are treated with acids, silicic acid, an amorphous, gelatinous substance is obtained. This is soluble in water and acids and is readily dissolved by dilute solutions of alkali hydroxides and carbonates.

Occurrence of Silica in Nature

Silica is one of the commonest minerals. In one form or another it enters into 60 per cent of the earth's crust and forms whole mountain ranges in certain countries.

Free Silica

Examples of free silica in the pure crystalline form are quartz and rock crystal but there are many other minerals whose essential composition is quartz but which have been changed in color or form by the addition of small amounts of other substances, usually oxides of other elements. Many of these are used chiefly for ornaments and jewelry but some, such as buhrstone and flint, have important industrial uses as well. A list of these quartz rocks includes the following:

Agate	Cairngorm	Needle stone
Aventurine	Chrysoprase	Onyx
Amethyst	Certine	Opal
Bloodstone	Flint	Plasma
Brazilian pebble	Hairstone	Phrase
Buhrstone	Hyalite	Rose quartz
Carnelian	Jasper	Sard
Cat's eye	Jaspilite	Smoky quartz
Chalcedony	Lydian stone	Tiger's eye
Chert	Milk quartz	

Common rocks containing a high proportion of free silica are:

Sand-stone, essentially grains of quartz with some feldspar, mica and other materials added.

Quartzite, a product of sandstone.

Ganister, a sedimentary rock with a very high percentage (about 98 per cent) of free silica.

Granite, composed essentially of quartz and feldspar, containing 25 to 35 per cent of free silica.

Pegmatite, a form of granite.

Ordinary sand is made up almost entirely of grains of quartz.

Amorphous (non-crystalline free) silica forms the composition of opal and is associated with quartz in certain other stones such as chert, flint and chalcedony. It is chiefly represented by diatomaceous earth and tripoli or "rotten stone."

Diatomaceous earth is a soft earthy rock composed of the skeletons of small aquatic plants, resembling chalk in appearance. It is capable of holding four times its weight of water and is a poor conductor of heat, sound and electricity. Tripoli is a porous rock which results from the natural decomposition of sandstone.

Silicates

Silica combined with other minerals, the so-called silicates, enter into the composition of every rock species and form the essential substance of such common materials as clay, mica, feldspar and slate.

The only silicate which is generally recognized as capable of causing a disabling disease is asbestos (most common type is chrysolite, hydrous-magnesium-silicate) which gives rise to the condition known as asbestosis, differing in several important respects from silicosis. This will be described later.

Sericite, a hydrous silicate of aluminum and potassium together with other fibrous silicates, sillimanite and tremolite, occurring in quartz bearing rock, has been put forward by Jones¹³ as mainly responsible for silicosis but this theory has not been substantiated.

Other silicates such as talc, somewhat similar to asbestos chemically, shale, kaolin, feldspar and pure mica, have been carefully studied but have not been found to produce significant changes in the lungs in industry except under conditions of very intense exposure. Experimentally these dusts do not cause any progressive changes.¹⁴ The term "silicatosis" has been used to describe the nodular fibrosis resulting occasionally from severe exposure to silicates other than asbestos. Silicatosis, when it occurs is probably the result of some admixture of free silica.

Industrial Uses of Silica

Silica has many properties which give it industrial value as for example its hardness, resistance to acid and to quick temperature change, its crystalline and decorative properties and, in the amorphous form, its value as a filtering medium and non-conductor.

It is used in a variety of different forms, i.e., in the massive form, both roughly broken and cut to different sizes and shapes; in the original condition as pebbles or as grains; crushed to a coarsely granular form or ground to a fine powder.

Its chief uses and corresponding states have been tabulated by Ladoo¹⁵ as follows:

Uses of Silica	Types of Silica Used
Abrasive uses:	
In scouring and polishing soaps and powders.	Quartz, quartzite, flint, chert, sandstone, sand, tripoli, and diatomaceous earth; all in finely ground state.
In sandpaper.....	Quartz, quartzite, flint, sandstone and sand; coarsely ground and closely sized.
In sand-blast work.....	Quartz, quartzite, sandstone and sand, crushed into sharp angular grains uniform in size.

Uses of Silica	Types of Silica Used
Metal buffing, burnishing and polishing.	Ground tripoli and other forms of ground silica.
For sawing and polishing marble, granite, etc.	Sharp, clean sand graded into various sizes.
As whetstones, grindstones, buhrstones, pulpstones, oilstones.	Massive sandstone from very fine to moderately coarse grained.
Tube-mill lining.....	Chert, flint, and quartzite in dense, solid blocks.
Lithographers' graining sand.....	Medium to fine sand or rather coarsely ground silica and tripoli.
Tube-mill grinding pebbles.....	Rounded flint pebbles.
In tooth powders and paste.....	Various forms of pure silica finely ground.
Wood polishing and finishing.....	All forms of silica ground to medium fineness.
Refractory uses: In making silica fire brick and other refractories.	Fairly pure quartzite known as ganister; not less than 97 per cent. SiO_2 nor more than 0.40 per cent alkalis. Tightly interlocking grains desired.
Metallurgical uses:	
In making silicon, ferro-silicon, and silicon alloys of other metals, such as copper.	Moderately pure sand, massive crystalline quartz, sandstone, quartzite, or chert.
As a flux in smelting basic ores.....	Massive quartz and quartzite.
Foundry-mold wash.....	Ground sandstone, quartz, and tripoli.
Foundry parting sand.....	Fine sand and ground tripoli.
Chemical industries:	
As a lining for acid towers.....	Massive quartz or quartzite.
As a filtering medium.....	Massive diatomaceous earth and tripoli, sand, finely granular quartz or quartzite, finely ground tripoli, diatomaceous earth, and other forms of silica.
In the manufacture of sodium silicate..	Pure pulverized quartz sand, pure tripoli, and diatomaceous earth.
In the manufacture of silicon carbide...	Pure quartz sand.
Paint: As an inert extender.....	Finely ground crystalline quartz, quartzite and flint; also finely ground sandstone, sand, and tripoli.
Mineral fillers: As a wood filler.....	Finely ground crystalline quartz, quartzite, flint tripoli, and other types of ground silica.
In fertilizers.....	As above.
In insecticides.....	As above.
As a filler in rubber, hard rubber pressed and molded goods, phonograph records, etc.	Finely ground silica of all types.
In road asphalt surfacing mixtures.....	As above.
Ceramic uses: In the pottery industry as an ingredient of bodies and glazes.	Flint, tripoli, and chert, amorphous silica preferred; also all other forms of very pure silica, all finely ground.
Building stone.....	Cut granite and sandstone.
Monuments, paving blocks.....	Cut granite.
In the manufacture of ordinary glass.....	Pure quartz sand.
In the manufacture of fused-quartz chemical apparatus, such as tubes, crucibles, and dishes.	Very pure massive quartz preferred.
Decorative materials: In the manufacture of gems, crystal balls, table tops, vases, statues, etc.	Rock crystal, amethyst, rose quartz, citrine quartz, smoky quartz, chrysoprase, agate, chalcedony, opal, onyx, sardonyx, jasper, etc.
Insulation:	
Heat insulation for pipes, boilers, furnaces, kilns, etc.	Massive and ground diatomaceous earth.
Sound insulation in walls, between floors, etc.	As above.

Uses of Silica

Structural materials: Sandline brick.....

Types of Silica Used

Moderately pure, sharp, angular sand, preferably finer than 20-mesh, together with a small percentage of finely pulverized silica.

Optical quartz: For the manufacture of lenses and accessories for optical apparatus.

Clear, colorless, flawless rock crystal or massive crystallized quartz.

Silicosis-Hazardous Industries

It is apparent that exposure to silica dust in industry will occur not only (1) in the use of siliceous materials as listed above but also (2) in obtaining or preparing siliceous materials, such as in:

Quarrying sandstone, granite, slate or mica; milling sand, flint or other silica-containing substances; cutting and surfacing granite or sandstone;

and (3) in any dust-creating operation where contact with siliceous material is unavoidable, as in mining, excavating and tunnelling in hard rock, or where silica dust is present incidentally, as for instance in the dust of textile mills and grain elevators.

In modern times silicosis was first recognized when a Commission was appointed in 1902 to study conditions in the gold mines on the Rand in South Africa. Since then investigations all over the world have brought to light its very widespread occurrence. The results of many of these investigations were summarized in the Report of the International Conference on Silicosis held at Johannesburg in 1930.¹⁶ The following table shows the industries in which it has been most commonly reported:

Industry	Source of silica exposure	Operations offering exposure
Abrasives: scouring powders, soaps.	Ground sand.....	Grinding, mixing, packing.
Excavating.....	Rock dust.....	Drilling, blasting, hauling.
Foundries.....	Sand used in molds and in sand blasting. Parting compounds.	Sand blasting, cleaning, sand conditioning.
Glass.....	Sand is a constituent of glass; also used in sand blasting.	Mixing, polishing, sand blasting.
Metal hand tools, instruments, etc.	Dust from grindstone when natural stones are used.	Grinding, also dressing surface of stones.
Milling sand, flint, slate.....	Dust from material milled....	Milling, packing, conveying.
Mining (hard rock).....	Rock dust.....	Drilling, blasting, hauling.
Pottery: including tiles, electrical fittings.	Dust from ingredients; china-stone, flint and feldspar.	Mixing, making, mould making, biscuit placing and brushing, fettling, etc.
Quarrying granite, sandstone, etc.	Dust from stone quarried.....	Drilling, blasting, cutting, hauling, etc.
Refractory materials, fire brick, etc.	Dust from materials used: ganister, quartzite, sandstone.	Crushing, grinding, mixing, and subsequent manipulation of dried bricks, etc.
Sandblasting: buildings, railroad cars, etc.	Sand used to clean or remove paint, etc.	Sandblasting.
Stone works: granite, sandstone.	Dust from stone.....	Cutting, surfacing, sandblasting.
Tunnel construction (hard rock)	Rock dust.....	Drilling, blasting, hauling.
Vitreous enamelling of sanitary ware.	Silica in enamel.....	Mixing, spray-coating.

Isolated cases have also been reported among such workers as grave diggers, leather dressers and metallurgists.¹⁷

CHAPTER III

CHARACTERISTICS OF SILICOSIS

Factors in the Development of Silicosis

At this point the question naturally arises: "What exact conditions of exposure to silica dust determine the development of silicosis?"

Unfortunately, the conditions influencing its development are so numerous and variable that a simple answer is impossible. The following all play a part but the chief physical factors are silica content and character, length and intensity of exposure, to which must be added individual susceptibility.

Type of Silica in Dust

By definition, silicosis comes from breathing air containing SiO_2 or free silica which, in the crystalline or quartz form, is most injurious. Amorphous (non-crystalline) silica is capable of producing a less severe degree of silicosis but exposure to this form is limited. The silicates (silica in the combined form) also are, as stated above, with the exception of asbestos, much less harmful than free silica.

Per cent of Free Silica in Dust

Since silicosis comes from breathing free silica, obviously one of the factors in promoting its development in any industry will be the amount or percentage of free silica in the dust under consideration. Dusts with higher percentages of free silica are more dangerous under similar conditions than those with less.

Thus the dust produced in grinding or pulverizing sand for use in abrasives is more dangerous than the dust in a cement plant because pure sand is composed of approximately 99 per cent free silica and cement of only six to eight per cent.

Size of Dust Particles

The smaller the size of particles in any dust, the longer they will stay in suspension in the air, subject to being breathed and, consequently, the more harmful the dust. Particles above 10 microns in size settle with relative rapidity. A micron is approximately one twenty-five thousandth of an inch.

The size of the particles in dusts of various kinds differs of course depending on the material, but in many processes involving exposure to silica dust the particles tend to be extremely small.

The latest data indicate that the majority of particles in an industrial dust and in silicotic lungs are less than one micron in size.¹⁸ This means of course that much of the dust in such operations is invisible in the ordinary microscope. Such dust is so fine that it can be thought of rather as a gas than as a dust. After all visible dust has been removed from an operation, the invisible dust present may still be sufficient to cause silicosis.

Length of Exposure to Dust

Then there is the factor of duration of exposure. The longer the exposure, the greater the risk. Intermittent dust exposure, providing it is not excessive, is less likely to give rise to silicosis than work where contact with dust is continuous. This is probably not true, however, of intermittent high exposures.

Degree of Dustiness

Of great importance is the degree of dustiness or the number of particles in a given amount of air. Naturally the more dust of a harmful character present, the greater will be the liability to silicosis.

Dust is estimated in this country as the number of particles per cubic foot of air. Dust counts in jobs of moderate dustiness range between 20 and 50 million particles per cubic foot, while in intensely dusty work the count may reach a billion particles or more.

Since the harmfulness of a dust depends not only on the number of small particles and percentage of free silica but also on the other factors men-

tioned, particularly length of exposure, it is extremely difficult to say what constitutes a "safe" dust count under these varying conditions.

Standards of Permissible Dustiness

In the granite industry where the granite contains 35 per cent of free silica, a count of 10 million particles per cubic foot is apparently safe. Workers exposed to dust giving counts of that amount or less have shown no increase of respiratory disease. Presumably with dust containing 70 per cent free silica a count half as high would be required for safety, but definite standards must await future investigation and experience.

The study of anthraco-silicosis made by the U. S. Public Health Service⁸ showed that employment in an atmosphere containing less than 50 million dust particles per cubic foot would produce a negligible number of cases of anthraco-silicosis when the quartz content of the dust was less than five per cent.

Where the quartz content was about 13 per cent, a safe limit appeared to be 10 to 15 million particles per cubic foot. The limit of toleration for rock workers exposed to dust with about 35 per cent quartz was set tentatively at five to 10 million particles per cubic foot of air.

Cummings, quoted by Drinker and Hatch, from a consideration of general experience, suggests five million particles per cubic foot as a threshold for dust high in quartz.¹⁸

In the National Silicosis Conference Report^{18a} the following statement is made in regard to "safe" dust concentration:

"In general, dust concentrations of less than five million particles per cubic foot of air are considered safe even in cases where the dust contains a high percentage of free silica. To obtain a more definite correlation of these two factors it has been suggested that the dust count be multiplied by the percentage concentration; then if the result is under five million, one can be almost sure that the conditions are safe. But unfortunately the reverse is not so definite; if the result is more than five million, one cannot say with assurance that the conditions are unsafe."

Silicosis may develop in several months under continuous intense exposure to fine dust of a very high quartz content; but it usually takes many years if there is intermittent exposure to a slightly dusty process where the material is coarse and low in quartz content.

Individual Susceptibility

It is apparent with silicosis as with most diseases, that there is a great difference in individual susceptibility but the chief reasons for this difference are obscure. In some cases early respiratory infection may be a predisposing factor. Relative inefficiency of the nose in filtering dust may be another. The rate of respiration and the type of breathing of a given individual has also been suggested as a possible factor in individual susceptibility.

It is held that there is a general racial susceptibility among negroes to silicosis as there is to tuberculosis. A recent investigation along these lines among 4,066 foundry workers by Greenburg, Siegal and Smith²⁵ is of especial interest in this connection because the silicosis rates found among the 180 colored foundry workers included in this study were lower than those for the group as a whole (1.7 per cent as compared with 2.7 per cent). Several factors, however, appeared to play a part in these low rates: (1) the colored group, as a whole, was somewhat younger than the entire group—55 per cent being less than 40 years of age compared with 42 per cent of all foundry workers in this age group; (2) their duration of foundry exposure was less—86 per cent having been exposed less than 20 years compared with 60 per cent of the entire group; (3) a larger proportion of them were employed in foundries of the "combined" type—55 per cent as compared with 34 per cent of the entire group—wherein the silicosis rate was found to be the lowest of the four types of foundries. The distribution of the colored workers by specific occupation followed in general the distribution of the white workers examined.

Action of Silica on Body Tissues—Experimental Studies

Toxicity

Silica is a definite tissue poison. Gardner,¹⁴ Gye and Purdy¹⁵ and others have found that when a suspension of minute silica particles is injected into the tissues, an acute inflammatory reaction occurs which is followed by necrosis or destruction of cells. This in turn is followed by a healing process characterized by the formation of a nodule of connective tissue fibres. Other dusts such as silicon carbide, emery, diamond, coal and iron may cause an inflammatory reaction but without necrosis or nodule formation.

Solubility

Silica is apparently soluble in body tissues. Mills²⁰ reports that when particles of the fresh water sponge, which is similar in composition to quartz, are introduced into the tissues of animals they are slowly but definitely dissolved. Belt²¹ suggests that when silica dust is inhaled it gradually changes in the lungs from the crystalline form to silica sol and eventually to silica gel. In the intermediate stage it injures lung tissue producing the disease silicosis, and reduces its resistance. This action continues and the disease progresses until all the toxic silica sol is changed to the inert gel.

Support for this idea is furnished by the experiment reported by Mills in which, after particles of fresh water sponge were placed in the lung tissue of a dog, definite fibrosis was found, suggesting a concomitant injury developing as the sponge dissolved. The production of fibrosis, over-development of connective tissue, in the lung is the characteristic feature of silicosis.

The idea that the harmfulness of silica depends on its solubility has not been fully accepted as yet, however. Gardner believes that tissue reaction to it occurs too rapidly to be explained on the basis of solubility alone. He suggests that some other property as yet unknown may be responsible.

As a Colloid

Heffernan and Green²² suggested that the action of silica on animal tissues does not depend on toxic properties but rather upon its properties as a powerful colloid, absorbing body fluids and otherwise interfering with normal processes.

Cummins and Weatherall²³ found that silica sol interfered with the bactericidal action of the blood against bacillus typhosis, though no effect was shown on the growth of tubercle bacilli.

Effect on Tubercle Bacilli

Experiments, interesting in the light of the clinical association of silicosis with tuberculosis, have been performed by numerous investigators. Gye and Kettle²⁴ found that when they injected silica and tubercle bacilli together, the bacilli were apparently protected by some characteristic of the silica abscess and multiplied rapidly. In animal experiments, if a silica abscess were produced in one groin and an abscess from another irritating substance in the other groin, tubercle bacilli injected subsequently by vein showed a tendency to concentrate in the silica abscess.

Gardner,²⁵ working with guinea pigs, found that partially healed areas of tuberculous infection were reactivated and made to progress by the inhalation of quartz dust.

These findings were confirmed by Dowd,²⁶ who concluded from his work, also with guinea pigs, that inhaled silica dust alters tissues so that they become more favorable to the growth of the tubercle bacilli. What the nature of the alteration is remains to be demonstrated.

Behavior of Silica in the Body

In spite of being a tissue poison in concentrated doses, silica in small amounts is a natural constituent of all animal tissue and is present in certain foods.

Urinary Excretion

King and Dolan²⁷ have shown that silica which reaches the blood either by absorption from the intestine or the lungs is rapidly excreted by the kidney.

The amount appearing in the urine varies with diet and also of course with exposure.

Bloomfield and Goldman²⁸ followed up the work of King and Dolan by studies on the urinary excretion of silica in a group of 123 anthracite coal miners and found that it averaged 2.5 milligrams per 100 cc of urine, while in a group of 11 control subjects it averaged 1.0 milligrams. They also found that silica is still excreted in the urine of individuals who have been away from any silica dust exposure for several years.

Goldwater²⁹ studied the urinary excretion of silica in non-silicotic humans and found (1) that there are wide variations in silica concentration depending on the specific gravity of the urine, (2) that the same individual on a constant diet may show wide daily fluctuations in urinary silica and (3) that different individuals on similar diets show great differences in output. He warns therefore that great caution should be observed in interpreting urinary silica findings.

Silica in Blood and Sputum

Silica may likewise be demonstrated in the blood and sputum. Boehme and Kraut quoted by Sweany,³⁰ reported a two- to three-fold increase in the blood of patients with silicosis, while King and Dolan found more silica in the sputum of such persons than among those not exposed.

Reaction of Peritoneal Tissue to Injection of Silica and Other Dusts

It is an interesting fact that a *nodular* reaction of the tissues to silica which in the lungs (shortly to be described) is characteristic of the disease, is not at all limited to pulmonary tissue. Miller and Sayers³¹ injected a suspension of various dusts into the abdominal cavities of guinea pigs and found there, after 90 days, reactions similar to those occurring in the lungs upon inhalation of the same dusts. So characteristic were the reactions that this method is recommended by the authors as a practical means of determining the possible harmfulness of any given dust.

The response of the peritoneal tissue to the injection of dusts was of three kinds, namely, proliferation, absorption and inertness. A reaction of proliferation with the formation of nodules was shown to quartz and chert. A reaction of absorption, where the dust deposits tended to disappear was shown to calcite, limestone, calcium carbonate, gypsum and cement—all dusts, it may be noted, containing calcium. Finally a reaction of inertness, where there was neither proliferation nor absorption was shown to soapstone, silicon carbide, ferric oxide, anthracite and bituminous coal and precipitator ash.

McCord³² and associates confirmed these experiments and determined the peritoneal tissue reaction to a number of other dusts as well.

Reaction of Lymph Nodes to Silica and Other Dusts

The reaction of lymph nodes to suspensions of various dusts was studied by Stüber³³ who found characteristic reactions to dust with a free silica content above 26 per cent. Other dusts such as cornstarch, rouge, manganese, cadmium and silicate dusts with a free silica content of not more than three per cent did not produce these changes. The author suggests that the lymph node injection method may be developed into a rapid means of determining the toxicity of dusts.

Behavior of Silica in the Lungs

The Dust Disposal Mechanism of the Lungs

The body is equipped to deal with inhaled dust in several ways. A good deal of it, especially the larger particles, is caught on the moist mucous membranes of the upper respiratory passages, in the nose, throat, pharynx and trachea and either blown out, coughed out or swallowed. The nose is more efficient in catching dust than the mouth and for this reason mouth breathers suffer more severely than others from the effects of dust.

The finer particles of dust escape retention on mucous surfaces and reach the air spaces of the lungs. There, a more elaborate dust disposal mechanism is brought into play. This depends on two elements, a special type of migrating cell known as a macrophage or phagocyte, and the pulmonary lymphatic system.

The Lymphatic Filter

The latter is composed of a fine network of thin walled lymphatic vessels which accompany all the pulmonary arteries and veins. Through these lymph channels passes a constant stream of lymph from the periphery to the root or hilum of the lungs where it flows through large collections of lymphoid tissue, the tracheobronchial lymph nodes. Small collections of lymphoid tissue, are located at intervals throughout the lymphatic network particularly at the dividing points of bronchi, arteries and veins. Deposits of lymphatic tissue may be thought of as playing the part of filters in the drainage system created by the lung lymphatics. Normally the amount of lymphoid tissue in the lung is least in infancy and tends to increase with age as it is needed. The pleural membrane covering the lungs has its own system of lymphatic channels which communicates freely with that of the lungs proper especially in the septa between lobes.

"Dust" Cells

When dust particles reach the alveoli, the air spaces of the lungs, they are promptly engulfed by the migrating cells already mentioned. Some dusts are engulfed quickly, others slowly. Silica is one of the latter. Some authorities believe that these cells originate from the lining of the air spaces themselves; others from the lining of adjacent blood and lymph vessels. When the "dust cell," as it may be called, has taken up its load of dust it makes its way into the nearest lymphatic vessel and travels along in the lymph stream to a deposit of lymphoid tissue. When the dust cells die the dust particles themselves may be absorbed or they may remain as an inert deposit in the lymph node or they may be picked up by other cells and carried farther. Sometimes when lymphoid deposits lie adjacent to a bronchus, dust cells may pass through its wall and a small amount of dust may work its way out in this way through the bronchi. Unless the lungs are overwhelmed with dust, however, most of the particles reach an ultimate destination in the hilum nodes.

Relatively large amounts of non-irritating dust can thus be taken care of in the lungs without involving much difficulty other than enlargement of the lymph nodes to accommodate it. But with silica dust certain special effects occur.

The Pathology of Silicosis

When silica dust enters the lung its first effect is on the bronchioles, the smallest bronchial passages. It damages their lining cells and causes a slight inflammation which the South African authorities speak of as a dry "bronchiolitis." This condition may be at least partly responsible for the dry cough, so common in silicosis.

Silica particles, after reaching the alveoli, are taken up by dust cells in the usual way and carried into the lymph vessels but now, as the engulfed silica becomes dissolved, its particular injurious action begins to take place. The dust cell containing silica may die, or the dissolved or colloidalized silica may penetrate through it. In either case the latter is brought directly in contact with adjacent tissues, and wherever it comes in contact with connective tissue, it stimulates increased growth and causes a condition of fibrosis or overproduction of connective tissue to develop.

Growth of Connective Tissue

This reaction is conspicuous first in the tracheo-bronchial lymph nodes. The sinuses or spaces through which the lymph flows in the nodes become gradually obstructed by the growth of connective tissue. The flow of lymph is slowed down until an actual backing up may occur. Then the dust cells with their silica loads settle in all the deposits of lymphoid tissue and, wherever they collect an overgrowth of connective tissue follows. As the resulting obstruction increases, the dust cells which Gardner has found are rendered more than usually active by the presence of silica, tend to penetrate through the walls of the lymphatic channels into the lung tissue and here again connective tissue overgrowth is set up.



The result is a generalized fibrosis which affects the alveolar walls, interferes with the elasticity of the lungs and encroaches on air spaces and small blood vessels. The same process is set up in the pleura overlying the lungs when backing up of lymph carries dust cells and silica into the pleural lymphatics. The result here is a dry pleurisy with thickening and adhesions.

The Silicotic Nodule

The form which connective tissue overgrowth produced by silica takes is characteristic. Its feature is the development of a definite nodule sometimes called a pseudotubercle, which is somewhat similar to the tubercle of



Figure 1

Microphotograph of Section of Lung Tissue Showing Silicotic Nodules

(From "Silicosis." International Labor Office, 1930)

tuberculosis but with definite differences. Gardner¹⁴ has described in detail the formation of these nodules in guinea pigs where the lesion is essentially the same as that found in man.

The nodules begin in collections of lymphoid tissue where the phagocytes or dust cells are arrested and the shape of the nodule, whether round, oval or elongated, depends on the configuration of the lymphoid deposit. The irritating effect of the silica stimulates the growth of connective tissue cells which gradually replace the lymphoid cells. These connective tissue cells become arranged in a whorl-like manner at the center, while at the circumference they take on a laminated or layer-like arrangement. Gradually the cellular character at the centre is lost and in the fully developed nodule, the centre is composed of clear hyaline material with a surrounding zone of connective tissue. Caseation or calcification of the centre of the nodule may occur.

Growth of the nodule occurs at its circumference and proceeds as the disease progresses, nodules becoming both larger and more numerous. Individual nodules may reach the size of 2-3 mm. or slightly larger, and be easily palpable and visible to the naked eye. Composite nodules may be formed by the growth of adjacent, individual ones. Contrary to the behavior of tubercles, breaking down of simple silicotic nodules does not occur. When it does it is an indication that infection has been added to the silicotic process.

When nodules form under the pleura they give the appearance of small, whitish, raised areas spoken of as "sub-pleural plaques." Not all the fibrosis in silicotic lungs is nodular. A fine diffuse fibrosis occurs also, and the extent to which the nodular or the diffuse type predominates seems to vary in different occupations depending possibly upon the percentage of free silica in the dust breathed, the rate of development of the disease, or other unknown factors.

Tuberculosis with Silicosis

The same mechanism by which dust is disposed of in the lungs operates to take care of infectious organisms. Tubercle bacilli and silica particles are engulfed by phagocytes in the same way. Consequently the silicotic and tuberculous processes are closely associated, and the lesions of the two show some similarity in their distribution.

The presence of the two combined conditions modifies the characteristic appearance of each. Tuberculous lesions in a silicotic lung show more fibrosis than would ordinarily be expected and silicotic nodules in the presence of a tuberculous infection tend to break down and become caseous as they do not do in simple silicosis. The large areas of massive consolidation found in advanced silicosis are often the result of the combined effect of silica and the tubercle bacillus. Such areas are, as a rule, larger than are formed by the coalescence of adjacent nodules in uncomplicated silicosis. Tubercle bacilli may be found in them, but they are notoriously hard to detect. The nodules may show caseation and cavitation, though excavation is less frequent than in uncomplicated tuberculosis.

Some authorities believe that a low-grade, latent tuberculous infection may be present in certain silicotic lesions from the start when tubercle bacilli are inhaled along with the dust, but that it remains enclosed in fibrosis and quiescent until activity develops later. Such a condition usually occurs in the apical region while the rest of the lung may be either free from obvious silicosis or show it in an early stage. In other cases larger irregular nodules scattered here and there throughout a silicotic lung may be produced by a latent tuberculous infection without any clinical signs. Animal inoculations from such lesions have, according to Strachan and Simson,¹⁴ been positive in a considerable number of cases.

Active tuberculous infection may occur at any stage of silicosis but is more common in the later stages when it usually runs a chronic course. The acute types, tuberculous pneumonia and miliary tuberculosis, are uncommon in the later stages of silicosis probably because of the more extensive fibrosis. In the early stages of silicosis, tuberculosis ordinarily runs its usual course but

appears definitely to aggravate the silicosis. Sometimes the onset of a tuberculous infection, according to the authorities quoted above, brings to light signs of silicosis which were previously not detectable. In such cases the infection seems to select sites in the lung where minute aggregations of



Figure 2

Silico-Tuberculosis

Cross-section of Lung Showing Extensive Consolidation from Silicosis and Tuberculosis with Marked Emphysema

(From "Silicosis." International Labor Office, 1930)

silica are already present and to assume the characteristic miliary distribution of silicosis. Willis³⁵ states that as a rule tuberculous infection with silicosis may occur in two forms, i.e., (a) apical and unilateral with downward spread as in ordinary pulmonary tuberculosis, and (b) non-apical, often bilateral, asymmetrical and often massive. Gardner¹ suggests that the apical lesion represents an infection, present before exposure to dust, which has failed to heal under its influence; while the non-apical form may represent a new, truly industrial infection. While massive areas of fibrosis usually occur late, in a few cases they may be present from the first, especially in the apical regions.

Evidently two factors operate to influence the development of tuberculosis in the silicotic lung. One is the coincident fibrosis which tends to limit and enclose the infection. The other is the tissue injury produced by silica which tends to reduce resistance to infection. Moreover the fibrosis, while it may limit the spread, may also tend to prevent healing and possibly result in more numerous and larger infected areas.

Belt²¹ suggests that tuberculous infection gets its start primarily where collections of silica particles occur in regions of less adequate blood supply. There the tissue becomes devitalized by the action of silica while the blood supply is inadequate for defense or repair. Relatively avascular areas, according to him, become more and more numerous as fibrosis progresses and it is this which accounts for the greater incidence of tuberculosis in advanced than in early silicosis.

Post Mortem Appearance of Silicotic Lungs

The first thing to look for in silicotic lungs according to authorities already quoted, is evidence of fibrosis first in the root glands, second in the pleura and third in the lung substance. Root glands are enlarged, firmer than normal and pigmented according to pigment in the dust breathed. The visceral pleura shows pigmentation and thickening together with a variable number of nodules or plaques which can be felt beneath the surface and appear as fine pin point pearly white areas surrounded by pigment.

On section, the surfaces of the lungs show small pigmented nodules. These can not be felt in the earliest stages. Later they become palpable and project above the cut surface. Their number and size give an indication of the stage of the disease. Thus, according to Strachan and Simson,³⁴ in early silicosis they may be small and moderately numerous or medium sized and sparse; in moderately advanced silicosis they may be small and numerous or large and moderately numerous; while in advanced silicosis they are both numerous and large. Nodules up to two mm. in size are considered small, from two to four mm. medium, and from five mm. to a centimetre, large.

Willis³⁵ summarizes the appearance of the lungs in cases of well developed silicosis as follows:

"The gross appearance of the silicotic lung is characteristic. It is stiff, inelastic, pigmented and adherent. It offers resistance to the knife which cuts it with a grating noise, the incision yielding a mottled surface of pigmented nodule, often grown around a bronchus or blood vessel, much pigment, diffuse fibrosis, thickened interlobar septum and thickened bronchial and vascular walls. Occasionally in the absence of complicating tuberculosis and often in the presence of the latter, there may be large densely fibrotic masses which occupy much of a lobe or a lung and which may be caseous or cavitated at the centre."

The lungs may not be much increased in size but show an increase in weight. Emphysema, particularly in the marginal regions and at the apices, is a more or less constant feature. In some cases plaques occur on the diaphragmatic pleura. Occasionally silicotic fibrosis may be found in neighboring lymph nodes in the posterior mediastinum, at the cardiac end of the stomach, around the pancreas and at the hilum of the liver.

In advanced stages, bronchitis is well marked and the bronchi are obviously thickened as are also the interlobar septa and trabeculae. Alveolar fibrosis is not marked except when infection is present or dust exposure has been overwhelming.

Tuberculous infection superimposed on silicosis is very difficult to detect by appearance in the early stages. Nodules may be more numerous at the usual sites of election for tuberculosis. In well marked cases of tuberculo-silicosis the root glands are much enlarged, of a grayish color, sometimes with foci of caseation, or calcification. There are dense adhesions on the pleura with much scarring and patchy thickening. Caseation can be seen in the nodules which in certain areas, especially under the pleura and toward the upper parts of the lobes, may become confluent with breaking down of tissue and cavity formation.

Examination of Lung Tissue for Silica

The actual presence of silica in the lungs can be determined by chemical analysis after ashing. The results are expressed as the percentage of SiO_2 in the total ash of the lung, or as milligrams per gram of dried lung tissue.

According to McNally,³⁶ more than two milligrams of SiO_2 per gram of dried lung tissue is evidence of undue exposure. The normal level being about one milligram. With two or more milligrams, nodulation is usually present but the degree is not proportional to the amount of silica present.

Silica is present in higher concentration in the lymph nodes. The normal for city-bred adults is approximately six milligrams per gram of dried tissue.

Crystalline silica fragments can also be seen in lung tissue by examination under the microscope with polarized light. But there is no relation between number of particles seen in a given area and the chemical content of SiO_2 , since the silica ceases to be visible when it loses its crystalline form.

Symptoms and Course of Silicosis

Rate of Development

Silicosis develops slowly under ordinary conditions. Pancoast and Pendergrass³⁷ found that the earliest cases of advanced silicosis among granite cutters occurred after 10 to 14 years; among pottery workers, after 24 years; and among metal grinders after five years. On the other hand they found cases of early silicosis in granite cutters after 20 years; pottery workers after 35 years, and metal grinders after 37 years. Under exceptionally severe exposure it develops in less than a year's time. The development of the disease depends on the extent of the exposure and the individual's reaction to dust. Seven years is probably about the usual time it takes to develop silicosis under conditions of moderate severity.

Predisposing Factors

Susceptibility varies with conditions such as nasal obstruction, infection and, possibly, age. When a man breathes through his mouth he loses the advantage of the extensive dust catching mucous surfaces of the turbinate bones and inhales more dust than he otherwise would. South African investigators have found that mouth breathers are definitely more subject to silicosis than nose breathers. Lehmann³⁸ says "the susceptibility of workers with poor nasal filtration is much greater than that of men with good nasal filters."

Chronic lung infection is likely to predispose to silicosis through involvement of the lymphatic system, thus facilitating lymphatic obstruction. A tuberculous infection especially renders an individual more susceptible to the effects of siliceous dust, and its onset may bring to light, in a comparatively short time, evidences of previously unsuspected silicosis. It is possible that silicosis develops more slowly in a young person than in an older one because of the less abundant lymphoid tissue present in the lungs in youth to catch and hold dust, but there is as yet no clear evidence on this point.

Symptoms

An individual who is developing silicosis may not know, even when extensive changes have taken place in his lungs, that there is anything wrong with him. Approximately one-fourth of the men in a group of New York City rock drillers whose chest X-rays showed advanced silicosis had no complaints whatever. When symptoms do occur, shortness of breath is likely to be the first one noticed. This becomes progressively more marked as the disease advances, until in the late stages it may be completely incapacitating. With

the dyspnoea there is likely to be some cough, usually dry, unless infection is present and some pain in the chest from pleural irritation. There may also be some epigastric pain. There is usually no loss of weight in simple silicosis and no fever or malaise.

The frequent association of tuberculosis with silicosis has already been mentioned and it was stated that while it may occur at any stage of development of the disease it is more common in the late ones. Individuals are found whose X-ray pictures show clear evidence of tuberculosis with silicosis who yet remain without symptoms; but eventually, when the infection becomes clinically active, evidences of toxemia such as fever, loss of weight, fatigue and night sweats make their appearance.

Course

The course of silicosis differs greatly in different individuals and under different conditions of exposure. It may develop so slowly that at the end of 40 or 50 years of dusty industrial life an individual may show only the earliest stage. It may develop in as short a time as one or two years under very severe conditions. It may apparently remain quite stationary even with continued exposure of a mild degree. On the other hand it may continue to progress even after exposure has ceased if the total dust dose has been severe enough. When tuberculosis is superimposed or becomes active in an advanced stage of silicosis, progress is usually rapidly down hill. This is borne out by the U. S. Public Health Service experience with the granite cutters at Barre, Vermont, where it was found that the median duration of cases of silico-tuberculosis, from the time of disabling sickness to death, was 15 months.

Physical Signs

Just as there may be few or no revealing symptoms until a late stage has been reached so the physical signs may be indeterminate and unreliable.

The appearance of an individual with silicosis is usually good. These people tend toward a robust type and are as a rule overweight. The first significant sign may be a diminution in the chest expansion which becomes progressively less as the disease develops and may be reduced to as little as two centimeters from the normal nine. The chest tends to be rigid and barrel shaped as in emphysema. In advanced cases there is usually some cyanosis and clubbing of the fingers.

In addition to restriction in chest expansion other evidences of functional impairment are to be found in an increased respiratory rate immediately after exercise; an increased lapse of time before the pulse returns to normal after exercise and diminution in the vital capacity.

Examination of the lungs reveals diminished resonance and distant breathing. The frequent presence of emphysema, however, often results in increased resonance. British investigators especially describe a harshened, thinned and shortened inspiratory murmur as characteristic. Râles are not heard as a rule unless infection is present. The chest is spoken of as characteristically "dry."

With Superimposed Tuberculosis

The first sign of active tuberculosis may be a definite and progressive loss of weight. The patient's "temperature rises and his pulse rate accelerates, his cough becomes definitely increased and his sputum more abundant and often streaked with blood; pleurisy is common; asthenia and exhaustion become extreme and the patient dies."³⁵ Gardner⁹ finds that the sputum in silico-tuberculosis is usually negative for tubercle bacilli until late in the disease.

There are no characteristic changes in the blood picture, blood pressure or urine, except possibly increased excretion of silica. The red cell count and the hemoglobin may show a compensatory increase.

X-ray Appearances

Early

In order to understand the X-ray appearances of silicosis, it is important to remember the underlying condition, the increase of fibrous tissue in the lung. It is this which is responsible for all that the X-ray shows.

At the very beginning of the process there is an increase in the normal lung markings similar to that found in chronic bronchitis or chronic passive congestion of the lungs. The hilum shadows are increased in extent and prominence; there is an increase in the trunk shadows and an increased prominence of the linear markings in the peripheral zone. While these changes will be found to develop under observation in a normal lung upon exposure to silica, they are not characteristic of that condition, occurring as they do from other causes and hence they have no general diagnostic value. The British speak of this appearance as "large branch fibrosis," likening the lung markings to the outline of a tree. As the disease progresses the fibrosis becomes more generalized. It is apparent beyond the main branches of the bronchial tree, and suggests the appearance of fine branches and twigs. This has been called small branch fibrosis, or "arborization." It is still not definitely diagnostic of silicosis.

Nodulation

It is not until the next development, the appearance of definite mottling due to the formation of silicotic nodules large enough to cause definite shadows

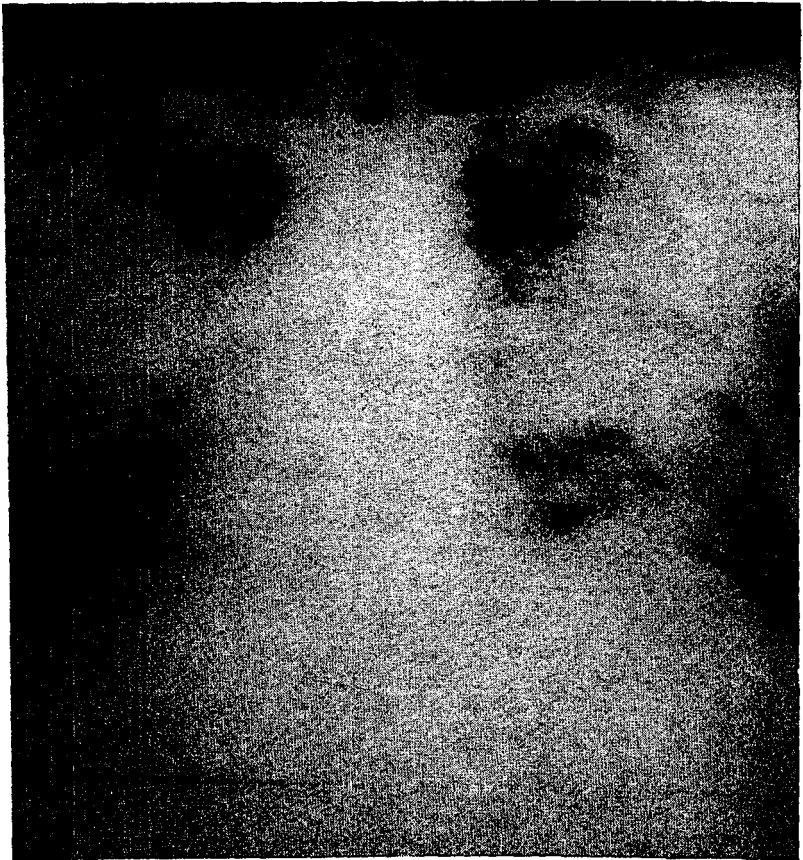


Figure 3

Third Stage Silicosis in a Blaster—Exposure 23 Years

that the picture can be considered characteristic. This appearance is the first specific sign of silicosis. It has been likened to a "leafless tree putting on leaves."

The mottling in silicosis usually appears first on the right side about the root of the lung. Later it becomes generally distributed throughout both lungs, though still with a tendency to be more marked at the roots and less in the apical regions and bases. The nodules vary in size from two to six millimeters and increase in size and number as the disease progresses. In the most advanced form of silicosis, nodules coalesce forming large, irregular masses. Massive, conglomerate lesions probably develop, according to Gardner,⁹ in areas where the lung tissues have been injured by previous infections that have subsequently healed.

Interstitial Fibrosis

This is the typical, though not the invariable, appearance of silicosis. A type with much interstitial fibrosis as well as nodulation may occur and is

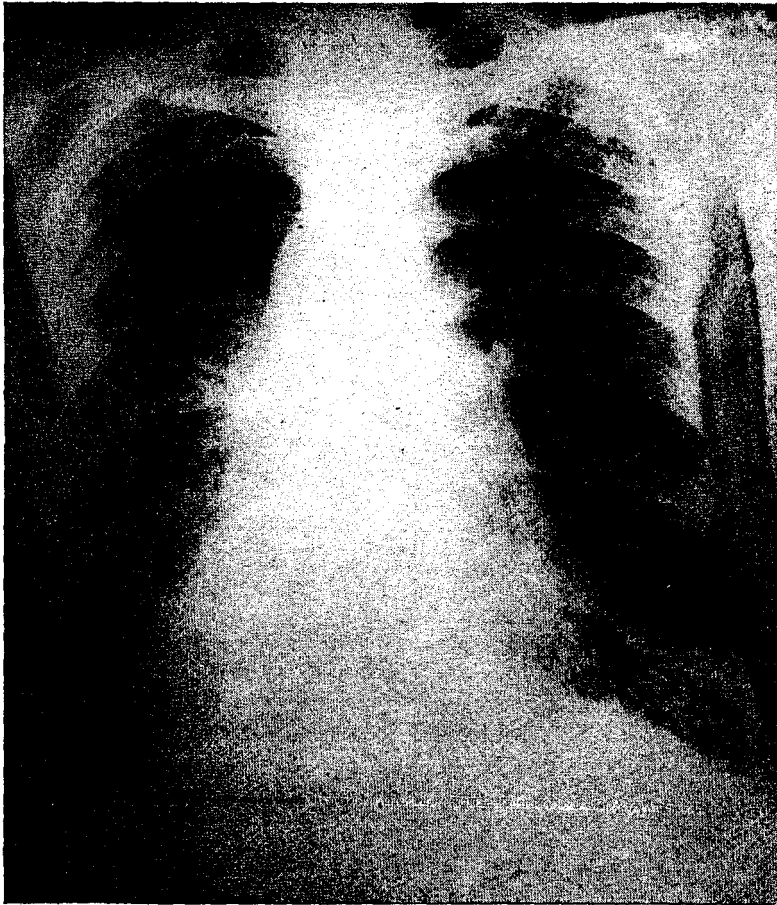


Figure 4

Silicosis with Tuberculosis in a Rock Driller—Exposure 31 Years

apparently commoner in some types of industrial exposure than others. Pancoast⁴² relates variations in X-ray appearances to the underlying pathology, pointing out that the linear fibrosis follows blockage of the lymph channels; the nodular type depends on fibrosis occurring in lymphoid deposits, and the interstitial type on penetration of interstitial tissue by dust cells, presumably occurring when the lymphatic block is rapid.

Silico-Tuberculosis

Gardner⁴³ describes the superimposition of tuberculosis upon silicosis as follows:

"It may manifest itself as simple superimposition or as a new type of disease known as silico-tuberculosis. The primary complex of tuberculosis can be detected on a background of generalized nodulation. The silica rarely has any effect upon these foci as they are generally sterile by the time persons begin to work in dust. Very rarely they have been reactivated. The scars of healed apical tuberculosis are recognizable in the silicotic lung. After prolonged exposures to dust such lesions have been seen to break down and give rise to very chronic silico-tuberculosis, a

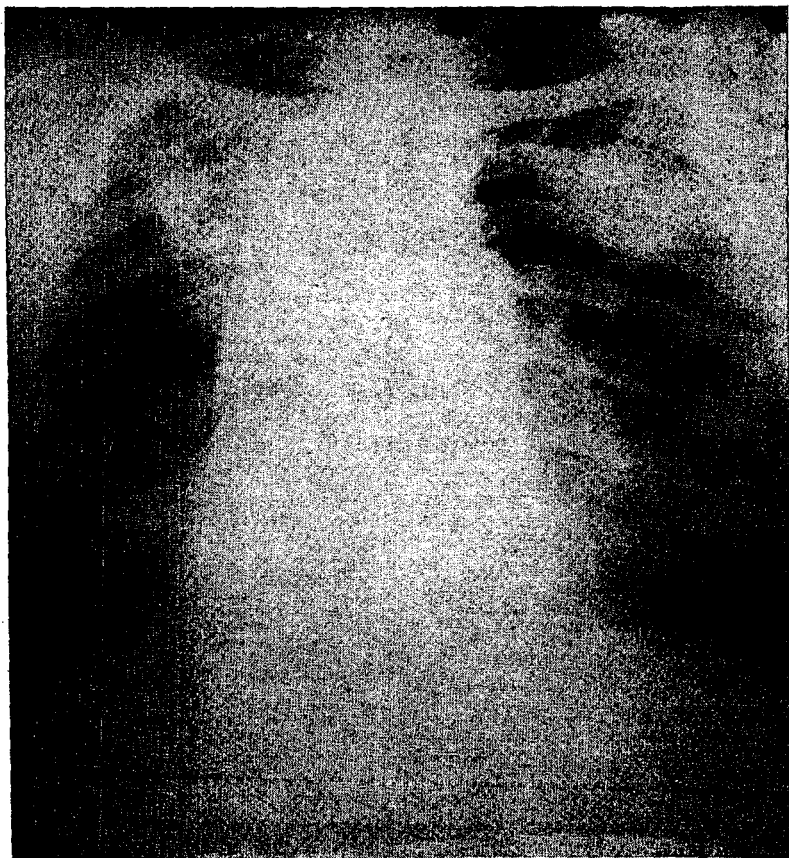


Figure 5

Advanced Silicosis with Tuberculosis in a Rock Driller—Exposure 31 Years

conglomerate type of disease, that spreads slowly from the vicinity of the original focus harboring bacilli. This lesion is productive rather than exudative in character; cavity formation, if it occurs, is a late manifestation. Aspiration and bronchogenic spread to remote portions of the lungs are limited. Silico-tuberculosis is much more commonly found in the base of the lung than simple tuberculosis. Whether the basal lesion of the silicotic is a manifestation of exogenous reinfection or whether it originates from reactivated latent foci has not been determined.

"The focus of silico-tuberculosis may be difficult to differentiate from the conglomerate focus of simple silicosis, but the former being due to an active infection is constantly, though slowly, changing. Repeated examinations by all available methods are essential for a diagnosis.

"Miliary tuberculosis occurs as a terminal event but it is particularly difficult to detect in the roentgenogram showing generalized discrete nodulation."

Standard Terminology Suggested by U. S. Public Health Service

There has in the past been considerable diversity in the interpretation of films showing the effect of dust exposure. This is of course inadvisable and for the sake of promoting greater uniformity the U. S. Public Health Service⁴⁴ has set up a tabulation of X-ray appearances with the underlying histologic changes. It is hoped that this will be accepted as the recognized standard for interpretation. The tabulation is reproduced below:

Roentgenological Appearances

Histological Appearances

HEALTHY LUNGS AND ADNEXA

- | | |
|--|--|
| 1. Healthy lungs. As defined by the NTA Committee report. | 1. Essentially the normal tissues of the vascular tree, the mediastinum, the bronchi, and trachea. |
| 2. Irregular exaggeration of the linear markings, with possibly some beading confined to the trunks. | 2. Cellular connective tissue proliferation about lymphatic trunks in the walls of vessels and bronchi. Beading may be due to various causes, as blood vessels seen end on, arteriosclerosis, minute areas of fibrosis in lymphoid tissues along the trunks. |
| 3. Increased root shadow. | 3. Cellular reaction in the tracheo-bronchial lymph nodes with extensions along afferent lymphatic trunks. |

These changes come within normal variations when not accompanied by recognized organic disease.

SIMPLE SILICOSIS

- | | |
|---|--|
| 4. Nodulation. Discrete shadows not exceeding six mm. in diameter, tending to uniformity in size, density, and bilateral distribution, with well-defined borders surrounded by apparently normal lung shadow. The outer and lower lung fields characteristically show fewer nodules. | 4. Circumscribed nodules of hyaline fibrosis located in the parenchyma of the lung. Occasionally some of these nodules may show microscopic foci of central necrosis. |
| 5. Conglomerate shadows that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by: <ul style="list-style-type: none"> a. Localized increased transparency of the lung with loss of fine detail. b. Intensification of the trunk shadow by contrast. c. Depression of the domes with possible tendency toward individualization of the costal components of the diaphragm. d. Lateral view: Increase in the preaortic and retrocardiac space with exaggerated backward bowing of the spine. Widening of the spaces between the ribs may or may not be present. | 5. The result of coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more or less hyaline fibrous tissue. The lung architecture is partially obscured. No demonstrable evidence of infection. Emphysema is a compensatory dilation of the air spaces with or without thickening of the septa. |

SILICOSIS WITH INFECTION

The characteristic appearances described under simple silicosis are modified by infection as follows:

6. Localised discrete densities and/or string-like shadows accompanying those of simple silicosis described above.
7. Mottling. Shadows varying in size with ill-defined borders and lacking uniformity in density and distribution, accompanying simple silicosis.
8. Soft nodulation. The nodular shadows described under simple silicosis, 4, have now assumed fuzzy borders and/or irregularities in distribution. This change may or may not accompany the simple mottling of 7.
9. Massive shadows of homogeneous density not of pleural origin symmetrically or asymmetrically distributed.
6. Strands of fibrous tissue, often along trunks and septa, with or without areas of calcification; indicative of "healed" infection.
7. (a) Areas of broncho-pneumonia with or without caseation, i.e., acute infection.
(b) Lobular areas of proliferative reaction with or without caseation, i.e., chronic infection.
8. Perinodular cellular reaction either exudative or proliferative in character.
9. Extensive areas of fibrosis probably due to organized pneumonia of tuberculous or nontuberculous origin superimposed upon a coexistent silicotic process. Outlines of normal structures may be partially destroyed.

Complications of Silicosis

Pulmonary

In addition to tuberculosis and pleurisy, which are the commonest and the most important complications of silicosis, certain other associated conditions are of interest. A tendency to recurrent chest colds with prolonged cough is rather characteristic. Some authorities believe that chronic bronchitis is present in all cases where the disease is well advanced. Collis and Yule³ in their statistical study found that the death rates for all non-tuberculous respiratory infections were higher among workers in siliceous dusts than in the general population.

Bronchiectasis, lung abscess and gangrene occur frequently in hard-rock miners, according to Proske and Sayers^{38a} who believe that prolonged inhalation of silica dust, contaminated with organisms from the mouth, paves the way for bacterial attack upon the bronchial mucous membranes.

Emphysema is a common complication of silicosis and tuberculo-silicosis and is often of the bullous type along the lung margins and in the apices.

Cardiac

Tuberculous pericarditis may arise sometimes by a backward lymphatic spread. Cardiac hypertrophy is not believed to be especially associated with silicosis though death with cardiac failure when an acute respiratory infection has occurred, or simply from emphysema and interstitial fibrosis, is of common occurrence.

Silicosis and Cancer

The question of whether inhalation of silica dust or the presence of silicosis predisposes to lung cancer is brought up from time to time but there is no conclusive evidence that this is the case. Occasionally the co-presence of the two conditions is striking. Dible,³⁹ for instance, reported three cases of malignant disease in 14 post mortem examinations of silicotic subjects.

On the other hand, Berblinger⁴⁰ reports that in 82 autopsied cases of lung cancer he has never found silicosis, and Teleky⁴¹ points out that were there any connection between the two conditions it would certainly have been brought out in the extensive South African experience. There, however, six cases of lung cancer were found in 1,109 autopsied white miners without silicosis, six cases in 1,023 autopsied Europeans, not working in mines and four cases in 1,083 autopsied white silicotic miners. This experience would seem to establish definitely the absence of causal relationship.

Diagnosis

In making a diagnosis of silicosis, a history of the individual's dust exposure and symptoms is of primary importance, and next to this a well taken X-ray of the chest. The physical examination, many think, is least important because of the variability and frequent absence of signs. However, it is useful in determining the presence of infection.

X-ray Technique

For the X-ray examination a rapid exposure is desirable. With 100 milliamperes an exposure of 1/10 of a second at a 66-inch target distance, varying the kilo-voltage according to chest thickness, gives very good results.

Early Silicosis

Some difficulty may be experienced in the diagnosis of early silicosis. As already stated, the increase in linear markings occurring at the beginning of a silicotic process bears no distinguishing features, and silicosis cannot be diagnosed until some true nodulation appears. Nodulation in its early stages may sometimes be simulated by fibrosis from some infectious process. In such cases the history of dust exposure, of past respiratory disease and of complaints may be decisive.

Advanced Silicosis

In its more advanced stages silicosis may very closely resemble miliary tuberculosis, but here again the story of the silicotic in relatively good health with few symptoms will contrast strikingly with that of the patient suffering from miliary tuberculosis. Fungus infections of the lungs may at times give an appearance indistinguishable from silicosis. In such cases examination of sputum by Irwin's⁴⁵ microincineration technique would demonstrate the presence or absence of silica. In medico-legal cases which have been fatal, determination of the amount of silica in lung tissue is of value.

Silico-Tuberculosis

The diagnosis of tuberculosis superimposed upon early silicosis does not usually present much difficulty because the X-ray appearances of tuberculosis are not yet obscured but the question of whether in a case of obvious tuberculosis early silicosis is present may be a more difficult one, impossible to determine except upon autopsy. The presence of tuberculosis when silicosis is in the well developed nodular stage may be suspected by the appearances already discussed. Willis⁴⁶ says "it is very helpful to remember in this connection that the pneumoconiosis rarely affects the extreme apices as tuberculosis does."

In the most advanced stage of silicosis with diffused fibrosis or massive consolidations it is very difficult in many cases to know whether tuberculosis is also present. Serial studies should then be made to show progress and a careful search for evidences of cavitation and for tubercle bacilli in the sputum.

Classification According to American Public Health Association

For the sake of clarity in diagnosis and also for the purpose of aiding in compensation settlements, various classifications of the stages of silicosis have been made from time to time. Some of these have been based primarily on X-ray findings; others on a combination of X-ray and clinical findings, and others on a roentgeno-pathological basis. They differ from each other considerably and enumeration of them all could only be confusing. The classification suggested by the Committee on Pneumoconiosis of the American Public Health Association⁴⁶ is given below:

"First stage (corresponds to anteprimary stage of South Africa):—The symptoms of uncomplicated first-stage silicosis are few and often indefinite. The man may apparently be quite well and his working capacity not noticeably impaired. Slight shortness of breath on exertion and some unproductive cough, often with recurrent colds, are the most usual symptoms. The man may have a little less ability to expand his chest than formerly, and the elasticity of the chest may be slightly impaired. The earliest specific indication of the presence of silicosis is the radiographic appearance, consisting of generalized arborization throughout both lung fields with more or less small, discrete mottling.

"This characteristic mottling is due to shadows cast by the discrete individual nodules of fibrous tissue in the lungs, and is essential to the diagnosis of silicosis; without this finding the diagnosis of silicosis is not sustained except by autopsy.

"Second stage (corresponds to primary stage of South Africa):—A definite shortness of breath on exertion is usually found, and pains in the chest are a frequent complaint. A dry morning cough is often present, sometimes with vomiting, and recurrent colds are more frequent. Even then the man's appearance may be healthy but he is dyspnoeic on exertion; he can not work as well as formerly; his chest expansion is noticeably decreased, the movement being sluggish and diminished in elasticity.

"The characteristic radiographic appearance is a generalized medium-sized mottling through both lung fields. The shadows of the individual nodules are for the most part discrete and well defined on a background of fibrous arborization, but there may be here and there larger but limited opacities due to a localized aggregation of nodules.

"Third stage (corresponds to the secondary stage of South Africa):—In the third stage the shortness of breath is marked and distressing even on slightest exertion. The cough is more frequent; the expectoration is in most cases slight but may be copious. The individual's capacity for work becomes seriously and permanently impaired; his expansion is greatly decreased even with forced inspiration; he may lose flesh; his pulse rate may be increased and his heart may become dilated.

"The radiographic appearances in the third stage are further accentuated; the mottling is more intense; the nodules are larger, of a conglomerate form so that large shadows are shown corresponding to areas of dense fibrosis.

"Physical examination of an individual may reveal changes in percussion and auscultation, mild in the first stage and increasing with the progress of the disease. These alone are not sufficient to be of great value in the diagnosis of silicosis."

Management of Silicosis

There is no cure for silicosis in the sense that the fibrosis may be made to disappear. Simple silicosis is seldom disabling and as a rule individuals suffering from it are better off at their accustomed work though every effort should be made to reduce their exposure to a minimum. When tuberculosis is superimposed the situation changes. Gardner¹ makes the following suggestions:

"No person with open tuberculosis should be permitted to work in an industry where silica dust is created. An old employee with closed silico-tuberculosis that does not incapacitate him may be allowed to do light work in departments where no dust is generated. Sanatorium treatment may be tried but it is often ineffective. A young employee with silico-tuberculosis should be given the benefit of sanatorium treatment.

"Persons over 40 with roentgenographic evidence of well-healed adult type tuberculosis can be employed in an industry with a silica hazard. Their only danger lies in the development of a massive conglomerate type of fibrosis. But it is assumed that an industry that is having pre-employment examinations is cognizant of the hazard and is making every effort to reduce the dust concentrations in its plants.

"The younger the individual with X-ray evidence of healed adult type tuberculosis, the less safely can he be exposed to silica dust.

"Roentgenographic evidence of a healed primary complex in a person over 16 years of age does not constitute grounds for disbaring him from exposure to silica."

Acute Silicosis

Under conditions of very intensive exposure to dust of an extremely high silica content, death has been reported in as short a time as one to 17 months after the beginning of exposure. Such cases have been referred to as "acute silicosis."

Kessler⁴⁷ reported six such fatal cases occurring in a plant where sand was pulverized to fine powder for use as an abrasive. The free silica content of the sand was approximately 90.24 per cent. All of the cases developed after exposure of from four to 18 months.

Chapman,⁴⁸ in 1932, reported three cases of silicosis, two of them fatal, occurring in men who worked at machines where powdered silica and alkali were mixed for the making of abrasive soaps. In one case symptoms developed after eight months of exposure, in another after 29 months. The third was incapacitated for work in a little over two years.

Kilgore⁴⁹ in the same year reported four cases of silicosis, three of them fatal, in men who worked in a polishing powder plant where quartz was pulverized on the premises. None had had an exposure over 14 months. Death had occurred 50, 21 and 14 months respectively after the beginning of exposure.

At Gauley Bridge, West Virginia, in 1929 a tunnel was constructed through rock with a silica content of 97 to 99 per cent. Under such conditions the dust hazard was severe. Many men* out of approximately 2,000 exposed, died in a period of some five years following the commencement of the work; death in these cases being attributed by some authorities to so-called "acute silicosis," while others attributed it to tuberculosis, or other respiratory disease.

Gardner⁵¹ examined at autopsy 15 such cases and found that infection had been the cause of death in all. Tuberculosis was definitely the cause of death in 11, probably the cause in two and unresolved pneumonia was the cause in two. Microscopic, but not macroscopic lesions of silicosis were present. Three characteristic changes were seen, namely: (1) Masses of small nodules in broad sheaths of fibrous tissue surrounding the pulmonary lymphatics; (2) Generalized fibrosis of the alveolar walls, an appearance usually seen only in advanced cases of silicosis; and (3) no, or slight involvement of the mediastinal lymph nodes.

The X-ray appearances of these cases were described by Sampson.⁵² He mentions two types, the first showing tuberculosis only, and the second tuberculosis with diffuse, fluffy nodulation.

Acute silicosis appears rather to be either acute infection or the acute exacerbation of previously existing infection, resulting from the effect of overwhelming doses of dust of a high quartz content.

Asbestosis⁵³

Symptoms

The disease produced by the inhalation of the silicate asbestos is characterized chiefly, but not invariably, by cough, dyspnoea, scanty expectoration, slight cyanosis and emaciation. It develops slowly, as a rule, over a period of 10 to 20 years, though fatal cases have occurred in five years time.

Physical Signs

Physical signs when present are those of a basal fibrosis with diminished breath sounds, limited chest expansion and diminished resonance. Fine dry râles and pleural friction sounds may be present. Risk of superimposed tuberculosis is less than with silicosis, though fatal termination from the fibrosis alone is more common than with silicosis.

Pathology

The pathology of the disease is definitely different from that of silicosis. The asbestos fibres are not easily ingested by dust cells and removed to lymphoid tissue, but remain in contact with the walls of the air spaces and set up a fibrosis which begins about the terminal bronchioles forming a sort of "collar" of fibrous tissue and spreads to form diffuse patches in the parenchyma. Infection, superimposed on asbestosis, as with silicosis, causes a more severe reaction.

X-Ray Appearance

The X-ray appearance in asbestosis is described as a fine pinpoint mottled "ground glass," "veiled" or "blurred" appearance, commencing at and involving principally, the bases of the lung.

* Exact figures are not obtainable. See Congressional Record 50 H. J. Res. 449, 74th Cong., 2nd Session.

Diagnosis

Diagnosis is made by the history and X-ray findings and is facilitated by finding microscopically in the sputum clumps of so-called asbestos bodies, yellowish, elongated bodies with bulbous ends. These result from the deposition on asbestos fibres of iron in the form of iron silicate.

The incidence of the disease depends upon essentially the same factors of intensity, duration of exposure, etc., as does silicosis. In this country owing to the relatively small numbers employed in work involving exposure to asbestos and to the relatively good plant hygiene it does not present a serious problem.

CHAPTER IV

INVESTIGATIONS OF SILICOSIS

Summary of Silicosis Investigations in the United States

It is impossible of course to estimate how many cases of silicosis there are in the United States. By the nature of the disease, most of them are unknown even to the subjects. But more and more information as to the risk of its development in various dusty trades is being assembled by special investigations. Most of these have been conducted by the United States Public Health Service but State Departments of Health and Labor, Universities and other agencies have done their share.

A resume of the more important studies is given below. A word of caution is necessary in comparing the findings. Standards of diagnosis for the earlier stages of the disease have varied considerably in the past among different interpreters. At one time there was a tendency to include cases showing marked increase of linear fibrosis in the early silicosis groups. As the disease has become more familiar, interpretation of early changes has become more conservative.

Asbestos

Lanza, McConnell and Fehnel⁵⁴ in 1935 reported on the examination of 126 persons employed in asbestos plants in the United States. Of the total number, four were diagnosed as having second degree asbestosis, 63 as first degree, 39 as doubtful and 20 as negative. Only one case of active tuberculosis (diagnosed by X-ray) was found. Cases of definite cardiac enlargement were frequent. Dust counts ranged from $\frac{1}{8}$ to 82 million particles per cubic foot. Particles in size up to 360 microns in greatest diameter were counted.

During the same year, a report on asbestosis in fabricating plants was published by the Department of Labor and Industry of Pennsylvania.⁵⁵

Dust concentrations in various departments ranged from 0.3 million particles per cubic foot of air to 123.3 million. Particle size was found to range from 1.35 to 2.12 microns in longitudinal diameter and from 0.45 to 0.69 microns in transverse diameter. No free silica was found. Fourteen, or 25 per cent, of 56 workers examined had clinical and roentgenological evidence of asbestosis.

In 1936, Donnelly^{55a} reported on 151 asbestos workers of whom 52 showed asbestosis and in the same year McPheeters^{55b} reported the examination of 210 asbestos mill workers of whom 25 per cent. showed questionable or definite asbestosis.

Cement

In 1928 a study of the health of cement workers was reported by the U. S. Public Health Service.⁵⁶ This study was conducted in a representative Portland cement plant and included an analysis of the causes and severity of sickness among a group of cement workers; a study of the physical condition of cement workers by means of physical examinations and X-ray films, and a study of the working environment, especially the character and amount of dust to which the workers were exposed.

The cement was composed of 62 per cent lime and 22 per cent silica together with small amounts of aluminum oxide, magnesia and sulphur trioxide. Free silica in the form of quartz was present in amounts varying from one to 6.5 per cent.

Dust counts in dusty locations ranged from 22 to 92 million particles per cubic foot. The dustier occupations of the industry were found to be associated with a high rate of disease of the upper respiratory system, skin, eyes, ears and digestive system. Of 53 individuals who were X-rayed, 15, or 28 per cent, showed evidence of lung damage from dust but of a relatively mild degree, since none of these had any symptoms of the condition. Tuberculosis was present in three of the 15 cases or five per cent of the total number.

Diatomaceous Earth

An examination of 108 men engaged in quarrying diatomaceous earth in California for use in fire brick, abrasives, concrete, etc., was reported by Legge and Rosencrantz⁵⁷ in 1932. This material contains 85 per cent free silica chiefly in the amorphous form. They found moderately advanced silicosis in 15 per cent of the men; advanced in six.

Foundries

A study of 1,614 foundry workers was made by the Special Industrial Disease Commission of Massachusetts⁵⁸ in 1933. Silicosis was found in 8.8 per cent, silicosis with tuberculosis in 2.6 per cent, and tuberculosis alone in 0.9 per cent. Dust counts in the various departments were made and ranged from one to 266 million particles per cubic foot.

In 1934 McConnell and Fehnel⁵⁹ reported an examination of 215 foundry workers in 41 foundries. Sixty-seven, or 31 per cent, were diagnosed as showing X-ray changes indicative of early silicosis. Dust counts and particle size determinations were made. The report discusses the high death rates among foundry workers for all respiratory diseases, brought out by the report of the Committee on Group Mortality which analyzed records of six large American and Canadian insurance companies.

In 1935 Warfield⁶⁰ reported the examination of 691 foundry workers of whom 129, or 18 per cent, showed silicosis.

In an investigation of 311 foundries in New York State in 1937, Greenburg, Siegal and Smith⁶¹ made X-ray examinations of 4,066 foundry workers in 80 foundries and found that of these 2.7 per cent showed silicosis.

Other studies in this industry were reported in 1937 by Kelly and Hall,^{60a} Osmond^{60b} and Sander,^{60c} in which the rates for silicosis were found to be 0.99, 5.5 and 7.0 per cent respectively.

Granite Cutting

In a statistical study of the granite industry about Barre, Vermont, published in 1922, Hoffman⁶¹ found that the mortality from pulmonary tuberculosis among granite cutters had increased from 257.7 per 100,000 in 1896 to 953.4 in 1918, while at the same time the tuberculosis mortality rate among the general population declined from 207.5 per 100,000 in 1896 to 96.4 in 1917. This excess in death rate was most marked among the men employed in cutting and especially among those who worked with pneumatic tools.

Such striking evidence of the apparent harmfulness of granite cutting induced the Public Health Service⁶² to undertake an investigation at Barre lasting from 1924 to 1926 as part of a program aimed to cover the entire field of the dusty trades. Sanitary surveys of plants were carried out, including dust determinations. Sickness and mortality records were studied and physical examinations, including X-rays, made of a number of men. The percentage of free silica in the granite of that locality was found to vary between 31.8 and 38.6. The dust counts varied from 59.2 million particles per cubic foot, for hand pneumatic tool operators, to 1.9 for office employees.

Practically all granite workers were found to have early silicosis after four years employment and well advanced silicosis in 10 years. The general incidence rate of tuberculosis, including early cases was 6.5 per cent, while the death rate from tuberculosis alone was 14.1 per 1,000, a rate which is in excess of the death rate for all causes in the general population. Among the facts noted in this careful and systematic investigation were the following: Universal occurrence of silicosis among the granite workers; a close relation between the intensity of dust exposure and the general health of the men; a sharp correlation between the length of exposure to dust and the prevalence of tuberculosis and the death rate from the disease; the death of a large proportion of workers from tuberculosis which required 20 or more years exposure to develop but was almost invariably fatal within a short time of onset, and the failure of workers to recover from their condition on going into non-dusty trades.

The conclusion was reached that the rising sickness and mortality rates from tuberculosis in this industry are due to longer use of the hand pneumatic tool.

The Special Industrial Disease Commission of Massachusetts⁵⁸ examined a group of 961 granite cutters in that State (1933) and found that 138, (14 per cent,) had silicosis and 73, (7.6 per cent,) had silico-tuberculosis. Thirteen, or 1.4 per cent, had tuberculosis only. Distribution of cases according to dust count groups is shown in the report.

In a study of 125 granite cutters from shops about New York City conducted by the Division of Industrial Hygiene and the New York Tuberculosis and Health Association,⁵⁹ 62 per cent showed silicosis. Tuberculosis definite or suspected (by X-ray) was present in 31 cases.

Granite Quarrying

Granite quarrying was investigated by the Public Health Service⁶⁴ in 1934. According to the report of this study the men were exposed to dust with a quartz content of 35.2 per cent. Seventy-five per cent of the particles were less than two microns in average diameter. Dust counts revealed very high concentrations in quarry hole drilling, 144.4 and 112.1 million particles per cubic foot respectively. Only the drillers showed pathologic lung changes. Of 36 drillers examined, half with an exposure of five to 19 years had silicosis, and four of the five who had spent more than 20 years at such work.

Grinding

The dust hazard in ax-grinding was investigated by Winslow and Greenburg⁷ in a large plant in Connecticut in 1920. No physical examinations were made, but the mortality rates among the workers were studied and found to show a high incidence of tuberculosis which was attributed to wet grinding on sandstone wheels. This was shown to be far more hazardous by actual dust count than dry grinding with an efficient exhaust. Dust counts in dry grinding shops with an exhaust system, for instance, showed an average of only one-twelfth as many particles per cubic foot as the average in the wet grinding shops.

Metal Polishing

A study of the health of workers in a *silverware* manufacturing plant was reported by the U. S. Public Health Service⁶⁵ in 1933. Dust counts in general showed less than five million particles per cubic foot. Pumice, emery, tripoli, sandstone, metal and rouge were the substances used. Of 51 X-rays taken none showed silicosis. Thirty-six were classified as having "more fibrosis than usual."

Of 80 metal polishers in various plants throughout New York City studied by the Division of Industrial Hygiene of the New York State Labor Department and the New York City Health Department,⁶⁶ two showed silicosis and 47 increased fibrosis.

Mining

Lead and Zinc—The first investigation of silicosis in mining in this country was made in 1914 and 1915 by A. J. Lanza and Edwin Higgins⁶⁷ of the U. S. Public Health Service and Bureau of Mines respectively among the lead and zinc miners of Joplin, Missouri. In that area the chief geologic formation is chert, a rock having a high percentage of free silica. Seven hundred miners were examined and of these 45 per cent were found to have silicosis, while 14 per cent had silicosis and tuberculosis and five per cent had tuberculosis. A further report of this same investigation was made by Lanza and Childs⁶⁸ containing the first detailed X-ray studies of silicosis made in this country.

Copper—Further investigations of mining conditions were made by Harrington and Lanza⁶⁹ in copper mines in Butte, Montana, in 1921. There, of 1,018 miners, 42 per cent showed definite signs of lung damage due to dust and six per cent were tuberculous.

The Bureau of Mines⁷⁰ continued its investigations with a study of 309 men in the zinc mining district at Picher, Oklahoma, in 1923. Of these 30 per cent had silicosis. Finally in 1927 a permanent clinic for the study of the disease was established by the Bureau of Mines and the Metropolitan Life Insurance Co. at Picher.

Coal—There is a prevalent opinion that coal miners do not suffer from silicosis or tuberculosis. But coal mining may produce silicosis when the associated rock has a sufficiently high silica content.

In 1935 the U. S. Public Health Service⁷¹ published a report of the examination of 2,711 active workers in the *anthracite coal industry*. Of these, 616, or 22.7 per cent, showed silicosis of a slowly developing non-disabling type. After 25 years of exposure 90 per cent of the exposed had acquired the disease. Clinical pulmonary tuberculosis was found in 15 per cent of the early cases and in 43 per cent of the late ones. The percentage of free silica in the dust breathed by these men varied from 3.4 per cent for the regular miners to 35 per cent for the rock tunnellers and muckers. Dust counts varied from less than five to more than 300 million particles per cubic foot.

Potteries

X-ray examination of 58 men engaged in making of bathroom fixtures was reported by Quaintance⁷² in 1934. They were exposed to the dust of clay containing 20 to 25 per cent free silica. Five, or 8.6 per cent, were found to have third stage silicosis after an average exposure of 16 years.

Rock Drilling, Blasting and Excavating

The apparent similarity of the risk in mining and the processes involved in excavating and tunnelling, so far as the silicosis hazard is concerned, led to an investigation of the latter processes in New York City by Smith and Fehnel,⁷³ in 1929. Analyses of specimens of rock drilled from various sites on Manhattan Island showed that the free silica content ranged from zero to 84 per cent. In the case of dry jackhammer drilling, 50 per cent of the silica particles were less than three to four microns in size, while with the water Leyner (wet) drill, 50 per cent of the particles were less than 1.8 microns in diameter.

Of the 208 drillers, blasters and excavators examined, 42 per cent showed early, and 15 per cent well-developed silicosis. Evidence of tuberculosis, including both active and inactive cases, was present in nine per cent of the total number.

Sand Blasting

The application of sand under air pressure to surfaces to be cleaned, freed from paint or etched, presents unquestionably one of the most serious silicosis hazards in industry today. Sand is always very high in free silica content, usually over 90 per cent, and it splits up on impact with the surface to which it is applied so that the dust particles become very small.

Greenburg and Winslow⁷⁴ in 1919 investigated conditions in the abrasive industry and made certain recommendations, and in 1932⁷⁵ they again reported a careful study of conditions in 28 metal working shops in four different states, using a total of 194 pieces of air blasting equipment. They found that when the sand blaster must work in the midst of his blasting he is exposed to air containing from 232 to 3,104 million particles per cubic foot, or from 23 to 310 times the amount of dust which is at present considered safe.

Even when sand blasting is conducted in closed devices, the air of the general workroom adjacent to the device contains, they found, on an average, over 20 million particles of dust per cubic foot.

Sandstone Quarrying

At Amherst, Ohio, the sandstone centre of the country, Kindel and Hayhurst⁷⁶ examined 919 quarrymen in 1926. Of these, 30 per cent showed evidence of silicosis, advanced in seven per cent, while only two per cent showed silicosis with tuberculosis. The low incidence of tuberculosis was

thought by the authors to be due possibly to an admixture of clay with the sandstone whose own free silica content varies from 94 to 97 per cent. Most hazardous was the job of grindstone turning where 77 per cent of workers were silicotic.

Slate Milling

Slate milling was studied by the U. S. Public Health Service⁷⁷ in New York State. The dust count in mining and milling operations was very high, 52 to 1,440 million particles per cubic foot. Eighty-seven per cent of 79 slate millers showed lung changes, but they were slight in all but four.

Spray Coating

The danger of silicosis resulting from the use of vitreous enamels in the manufacture of sanitary and other ware was investigated by the National Safety Council⁷⁸ and reported upon in the Final Report of its Spray Coating Committee, published in 1927. It was found that a real hazard may occur if ventilation is inadequate. Enamels examined by them which were used for spraying castings were found to contain from 21 to 37 per cent silica; those used for spraying sheet metal contained from 43 to 47 per cent silica. Dust counts at the working face of spray coating booths ranged from 400,000 particles per cubic foot where the air velocity was 212 linear feet per minute (a low count,) to the excessive count of 445,000,000 particles per cubic foot where the exhaust ventilation was so poor as to be inappreciable. X-rays were obtained of nine workers who had been employed at this work for more than three years and of these, two showed definite and a third possible, silicosis.

Talc Mining

Ninety-three per cent of 57 talc miners were found to show slight lung changes in the study by the U. S. Public Health Service⁷⁷ reported in 1933.

In another study of the effect of talc made in two Georgia talc mills by the same agency⁷⁹ reported in 1935, 66 workers were divided into three groups according to dust exposure. The first group of 33 was exposed to 300 or more million particles of dust per cubic foot; the second of 13, to an average of 135 million particles and the third of 20, to an average of 17. Sixteen, or approximately half, of the mill workers in the first group showed nodular fibrosis of the lungs, equally divided between early and advanced stages. In the second group six of the 13 showed early changes. There were no changes in the third group.

The type of the talc in the region studied was hydrous magnesium silicate, $H_2Mg_3(SiO_3)_4$.

Trap Rock Quarrying

The results of an X-ray examination of 607 trap rock quarry workers were reported by Goldwater.⁸⁰ This rock has a low percentage of free silica, usually less than five. Of the entire group, two showed early and five more advanced silicosis, one complicated by tuberculosis.

CHAPTER V

PREVENTION OF SILICOSIS

The first step to be taken in the prevention of silicosis in any plant is an investigation of the existence and severity of the hazard. In other words, the free silica content of the dust in question, if not known, must be determined by suitable analysis, and its amount or intensity measured by dust counts.

Analysis of Dust for Free Silica

Determining the presence of free silica in any material is a matter for an experienced investigator trained in chemistry, and if possible in petrographic analysis. Chemical methods alone can yield directly only the amount of total, or free and combined silica in a sample. From this figure the free silica must be calculated indirectly by ascertaining the amount in combination with bases known to be present and deducting this from the total silica. In dust containing a variety of minerals, it may be difficult to calculate the amount of uncombined silica correctly.

Drinker and Hatch¹⁸ describe two methods for the determination of free silica and reference is made to their text book for details. The methods are (1) the combined chemical and petrographic method according to Knopf, and (2) the immersion method according to Ross and Sehl. Their studies show that in case of mixed dusts *it is necessary to separate the sample into size fractions and determine the composition of each fraction* in order to determine the true hygienic significance of the dust. For instance, the percentage by weight of free silica in the total sample of a foundry dust examined was 58.5 per cent, but the amount of free silica with a particle size less than 10 microns was only 5.7 per cent by weight of the total sample.

Dust Sampling Methods

Having determined the percentage of free silica in the dust in question and found it to be such as to present a possible silicosis hazard the next step is to determine the intensity of exposure by actually counting the number of particles in a given sample of air.

There are a number of methods of collecting dust samples, depending upon six general principals of operation, namely: Settlement, filtration, washing, impingement, electric precipitation and thermal precipitation. Those best known are the Kotze Konimeter, which has been employed in routine dust sampling in South Africa for more than 20 years; the Owens Jet Dust Counter which was long popular in Great Britain, and the Greenburg-Smith Impinger, the official instrument of the U. S. Public Health Service. All of these employ the principle of impingement.

Konimeter

The Konimeter is a small instrument which can be held in the hand and has the advantages of simplicity and ease of operation. Air is drawn in through a nozzle by means of a spring-actuated pump and impinges upon a collecting plate. Samples of 2.5 and 5 cc. of air (and multiples of these) may be collected. The glass plate upon which the air impinges is covered with a thin film of petroleum or glycerin jelly which retains the dust. Thirty samples can be collected on a single disc. Immediate examination of the spots under the microscope follows the collection of the samples.

The disadvantages of the Konimeter are that the dust-collecting efficiency is low, and it is selective in respect to size of particles.

Owen Jet Dust Counter

The Owen Jet Dust Counter is somewhat similar to the Konimeter but it has a capacity of 50 cc. and the impinging surface is an ordinary cover slip. It has essentially the same advantages and disadvantages of the former.

Greenburg-Smith Impinger

With the Greenburg-Smith Impinger apparatus a known amount of air, usually 10 to 30 cubic feet, is drawn through a tube into a flask of dis-

tilled water. As the air leaves the tube it strikes a glass impinging plate submerged beneath the surface of the water. The dust particles in the air are thus caught in the water, diluted and placed in a counting chamber for microscopical examination and counting.

Electric Precipitator

This is an instrument which, though less used than the other three in the past, has a high degree of efficiency and is used by the U. S. Bureau of Mines in testing respirators. Dust laden air is passed between two surfaces carrying a high electric potential and under the force of the electric field the particles are driven in a direction normal to the air motion and so precipitated upon the collecting surface. Samples cannot be examined by direct microscopic count but must be washed out into water or other liquid first.

Thermal Precipitator

This instrument is popular in Great Britain at the present time but is not used to any extent in this country. It depends upon the principle that a dust free area will occur around a hot rod.

Dusty air is drawn through a slot across which is placed an electrically heated wire. The walls of the slot are formed by cover slips kept cool by a backing of brass blocks which act as heat conductors. The dust is deposited on the cover glasses which can be removed for counting.

For a complete description of the above methods, and others, the reader is referred to the chapter on "Dust Concentration" in *Industrial Dust* by Drinker and Hatch.¹⁸

Particle Size Distribution

In addition to counting the number of particles in a given sample, an estimate of particle size distribution—that is to say the percentage of particles of various sizes under 10 microns—is valuable. With the Owens apparatus it is possible to measure particles as small as 0.5 microns in diameter while even smaller particles can be distinguished and their presence recorded. Particles of one micron in size are shown by the Greenburg-Smith Impinger method and an experienced observer can detect particles as small as 0.7 microns. In a study of 26 samples of various industrial dusts Bloomfield found that most of the particles—69 per cent on an average—were between one and three microns in average diameter. Twenty-one per cent were less than one micron and two per cent were less than 0.5 microns.

Hatch and Pool²¹ advocate the use of dark field illumination with a special counting cell to obtain greater magnification. With the dark-field microscope, particles down to slightly more than 0.1 micron in size can be enumerated.

Preventive Measures

When an injurious amount of an injurious dust has been demonstrated by dust analysis and dust counts, the question of how to correct the situation next arises.

The prevention of silicosis involves several different angles of attack depending upon the particular problem under consideration, and usually several methods in combination are necessary for the best results. The methods of attack all follow certain familiar principles. They may be divided logically into three groups: Those having to do with the process; those having to do with the work place, and those having to do with the worker himself.

Related to Process

The first group of preventive measures, those related to the process, comprises the following:

1. Substitution of non-silicosis-producing material.
2. Enclosure and segregation of dusty processes.
3. Local exhaust ventilation for the removal of dust at point of origin.
4. Suppression of dust by water.

Related to Work Place

The second group of preventive measures, those related to the work place, includes:

5. General artificial ventilation.
6. Plant cleanliness.

Related to Worker

The third group, relating to the worker, comprises:

7. Direct protection of the worker.
8. Alternation of work.
9. Medical supervision.
10. Education.

Substitution

This solution of the problem of prevention is at once the simplest, where practicable, and the most satisfactory. It has been applied successfully in several important industries.

In abrasive blasting the use of a metal abrasive instead of sand reduces the free silica content of the dust to less than five per cent from 42 per cent or above. Metal abrasive, steel grit, lasts longer than sand and is consequently cheaper. It can be used with the same equipment. Aluminum oxide also makes a satisfactory substitute for sand, especially in monument work, and has the advantage of greater cutting power.

The widespread use of artificial stones of silicon carbide or aluminum oxide instead of natural stones in metal grinding is another example of substitution with very beneficial results so far as silicosis is concerned. Studies by Clark⁸³ have shown no silicosis hazard in exposure to the dust of aluminum oxide, and no increased evidence of tuberculosis.

The silicosis hazard which exists in foundries, is due to sand blasting of castings and also the use of "parting compounds" for dusting molds which are often very high in free silica content. In addition to substitutes for sand in blasting, non-siliceous parting compounds are available and are quite generally being substituted.

The pottery industry, especially abroad, has always been associated with high silicosis rates due to the use of flint in bedding the ware for firing. Some plants have reduced this hazard markedly by the substitution of clay with its low free silica content for flint which is pure quartz.

These are a few outstanding examples of silicosis prevention by substitution. Other applications of the principle may be anticipated in the future. The first thing for the preventive minded employer to ask himself is: "Do I need to use this dangerous material?"

Isolation of Dusty Processes

Next to removing the silicosis hazard entirely by giving up the use of siliceous material, possibly the most effective safeguard when practicable, is isolation of the dusty process either by mechanical enclosure or geographical segregation.

Some processes, notably certain types of sandblasting, lend themselves admirably to enclosure by barrels, tables or cabinets which can be constructed so that very little dust escapes into the workroom. Bloomfield and Greenburg⁸⁴ found in a study of enclosed sandblasting operations, that with proper apparatus properly maintained the average amount of dust in rooms at large was less than five million particles per cubic foot. Strict care in upkeep is necessary, however, for under less than ideal conditions the average room atmosphere where this type of work was carried on contained over 20 million particles of dust per cubic foot, a dangerous concentration.

Aside from enclosure in mechanical systems, situations occur where dusty processes can be isolated in such a way that a minimum number of workers will be exposed. Measures for the control of silicosis in metal grinding in Canada provide for the "racing" of grindstones in separate rooms. Surfacing machines in stone yards could be similarly isolated. Instead of this, they are often operated in the midst of the yard where the dust created affects everybody present.

Isolation of dusty processes by *time*, as well as geographically, is another possibility under some circumstances. An example is the shake-out process in foundry work which is often done at night when only the shake-out crew itself is exposed.

Local Exhaust Ventilation

For the many dusty processes where silica is necessarily present and isolation is not practicable, the best preventive measure is removal of dust



Figure 6

Rock Drilling—No Dust Control. Serious Silicosis Hazard

at point of origin by exhaust ventilation. There is almost no limit to the possible application of this principle. It is widely used in such operations as grinding, polishing, mixing, filling, etc., and new applications are continually being developed. One of the most important and recent of these is a method of exhaust for the removal of the dust in rock drilling, long a very serious source of silicosis in mines and tunnel construction. Another hazardous trade where control is dependent upon properly applied local exhaust ventilation is stone cutting.



Figure 7

Rock Drilling—Dust Removed. Silicosis Hazard Under Control

There is no need here to discuss details of such ventilation since each situation presents its own particular problems in the engineering field. The crucial test of whether or not a system is adequate, lies in its ability to maintain the dust in the air at a low level and its efficacy should always, therefore, be tested by dust counts.

Water

Suppression of dust by the use of water as a spray or stream applied at the point of origin of the dust has a place in the control of silicosis but its value should not be overestimated. The finest particles are not laid by water but actually held in suspension in the air by droplets of moisture. Dry drilling under adequate exhaust will produce lower dust counts than wet drilling. Winslow and Greenburg,⁷ in their investigation of an ax factory found much more dust present under conditions of wet than dry grinding when the latter was done with proper exhaust. With wet methods, the dampened dust itself must be removed or it will dry out and constitute a hazard all over again.

Under some circumstances, wet methods especially in combination with general ventilation are valuable. Reduction of dust and consequently the silicosis rate in the South African mines has been accomplished through these means. In operations, such as stone crushing and grinding, dust may be reduced by the use of water when other measures are impracticable.

General Artificial Ventilation

Changing the air of the workplace frequently by artificial ventilation, bringing in fresh air and removing the stale is a valuable adjunct to other methods of control. Its effect is actually to dilute the dust present. In the mines already mentioned the use of wet drills is combined with general artificial ventilation by fans which ensure a circulation of about 60 cubic feet per person, per minute.

Plant Cleanliness

One of the most important parts in dust control is played by good housekeeping, that is to say by measures to promote plant cleanliness. In well kept foundries it is estimated that such measures account for 75 per cent of dust control. If a plant is not kept clean, dust accumulating from day to day, settling on floors and other parts of the workrooms, adds materially to the general hazard by being stirred up and re-circulated. When circumstances are appropriate, arrangements for vacuum cleaning or hosing down workrooms at intervals, preferably outside working hours, should be included as a matter of course in all silicosis preventive programs. Special arrangements for the care of dusty material come under this heading. A case in point is the use of a grating in the floor of a sandblasting room to allow the sand to fall through to a container instead of accumulating on the floor. Under this item of good housekeeping, too, should be included the careful upkeep of all ventilating devices and other protective apparatus. If this is not done even the best equipment will deteriorate rapidly.

Masks and Helmets

Direct protection of the worker by the use of masks or helmets is impracticable in many situations owing to the difficulty of a man's carrying on heavy work when so encumbered; so that in general, control should be sought by other means, but there are situations when this form of protection is satisfactory and even necessary. In abrasive blasting, for instance, when the operator must be in the same room with the object being cleaned, a positive pressure helmet, that is to say a helmet in which pure air is provided from an outside source, is absolutely essential in addition to room ventilation. Various types of helmets of satisfactory design are available. In studies made by Bloomfield and Greenburg²⁴ it was found that with a properly made helmet an air flow into the helmet of six cubic feet per minute was sufficient to supply clean air (dust count under three million particles per cubic foot) under all conditions encountered. It is of course essential that the helmet be inspected frequently for defects and

kept in sound condition. Incidentally the same studies revealed the total inadequacy of non-positive-pressure helmets in abrasive blasting. Where no outside air was supplied the dust counts under these helmets averaged 581 million particles per cubic foot. In such circumstances the helmet gives a totally false sense of security.

Masks represent a cheap and simple type of direct protection which has much in its favor under appropriate conditions. They are eminently adapted to a situation where the dust exposure is moderately severe but of short duration. Masks of high efficiency against silica-containing dust have been developed which are at the same time reasonably comfortable and offer relatively little resistance to breathing. If such masks are properly maintained, there can be no objection to them and they might under such circumstances, take the place of expensive ventilating equipment. But unfortunately the human element, ignorance or indifference on the part of the worker and laxity on the part of the employer, combine to destroy their effectiveness. It is the common carelessness found in their use, together with the sense of false security when carelessness exists, which renders them unsatisfactory as a rule in actual practice.

Medical Supervision

In enacting its so-called "silicosis legislation" in 1936, the New York Legislature said:

"It is hereby declared to be the policy of the Legislature of this State, in enacting this Article, to prohibit through every lawful means available, any requirement as a pre-requisite to employment which compels an applicant for employment in any occupation coming within the purview of this Article to undergo a medical examination."

However desirable pre-employment physical or medical examinations in the dusty industries may be from a medical point of view, economic considerations preclude them in New York State at the present time. As a representative of the workers expressed it:

"We want our jobs whether we have silicosis or not."

However, the young worker entering a dusty industry for the first time will be doing himself a distinct service if he subjects himself to a medical examination to determine that he is not peculiarly susceptible to dust irritation. Such a worker should repeat these examinations at regular intervals to keep a constant check-up on his condition.

Such a procedure will absolutely protect the individual worker from silicosis if he heeds the information disclosed by these examinations. First, because it is known that people who have had previous lung disease or who have any obstruction to breathing will get the disease more easily than others and hence should not be employed for this work for their own good. Secondly, it is necessary to know whether measures in use are in fact effective, and this can only be ascertained by periodic check-up examinations to discover whether silicosis is developing. Thirdly, if early pre-silicotic changes in the lungs are found by X-ray examination following dust exposure, silicosis may be avoided by prompt action in changing to another type of work.

The interval between examinations should depend upon the intensity of exposure. Where it is moderate, annual check-ups will probably be sufficient; where very severe, re-examinations had best be made at least every six months. The initial examination should include a complete physical survey with an X-ray of the chest. Subsequent check-ups may be limited to chest X-ray examinations only.

Education

Education of the worker in the nature of the risk he is running, the part he can play in preventing the disease by intelligent use of preventive equipment and the value to himself of physical examinations should be a part of every preventive program. If more attention were paid to education the advantage of physical examinations as a device to keep men healthy and preserve their working capacity would be apparent.

Preventive measures have been discussed separately but in practice, no one alone, except of course the first—substitution of a harmless substance—is enough, and the more that are used in combination the more complete will protection be. A satisfactory and comprehensive program for the prevention of silicosis in the majority of dusty industries will include physical supervision and education, alternation of men at dusty and non-dusty work, segregation of dusty processes, local exhaust ventilation, or masks or helmets, or both and finally plant cleanliness and an invincible determination on the part of those responsible to keep down dust. The efficiency of measures to remove or control dust can be demonstrated quite simply by dust counts and these should always be made at intervals in conjunction with a preventive program.

New York State Code for the Control of Dust in Rock Drilling

Action on the part of State Departments to formulate rules for controlling dust in specific industries is a highly constructive approach to the problem of silicosis prevention.

In New York State the following code relating to the control of silica dust in rock drilling became effective May 1, 1937.

A committee is also at work on the formation of rules for the control of dust in stone cutting and finishing.

BULLETIN NO. 33: Rules Relating to the Control of Silica Dust in Rock Drilling.

These rules shall apply to all rock drilling operations in silica-bearing rock.

"Rock Drilling" shall mean drilling, cutting, chipping, channeling, broaching or crushing to excavate rock, by means of machinery or hand hammers.

"Silica-bearing rock" means any rock formation, natural or synthetic, containing, as a component part, free silicon dioxide. **"Silica-bearing rock"** shall be divided into two classes:

Class I.—Any rock formation of substantially uniform geological character having free silicon dioxide as a component part which is known from geological studies or otherwise can be shown by the applicant to the satisfaction of the Industrial Commissioner, to run uniformly less than ten (10) per cent by weight, of the rock formation.

Class II.—All rock formations having free silicon dioxide as a component part, ten (10) per cent or more by weight, and all other formations, natural and synthetic, having a variable and unpredictable content of free silicon dioxide.

"Injurious silica dust concentration" shall mean dust produced from drilling silica-bearing rock which is in excess of the following values as determined by an approved dust count method:

Class I.—One hundred million (100,000,000) particles per cubic foot of air.

Class II.—Ten million (10,000,000) particles per cubic foot of air.

"Approved" means approved by the Industrial Board.*

Dust Control: All rock drilling operations in silica-bearing rock shall be executed so that there will be no dissemination of injurious silica dust concentrations into the atmosphere.

Dust Control Methods: Dust control methods shall be by any of the following:

Suction or exhaust methods.

Wet method.

Or other methods approved by the Industrial Board.*

Approval: Only approved dust control equipment and methods shall be used. To obtain approval, dust control equipment shall be substantially constructed and installed and shall successfully meet the tests prescribed by the Industrial Commissioner.

The Industrial Commissioner shall establish standards for testing of dust control equipment and methods for the purpose of securing approval of such equipment by the Industrial Board.*

Notification of Starting Work: Before starting any rock drilling operations to which these rules apply, notice shall be filed with the Industrial Commissioner, advising as to the location and the date of starting work.

Inspection by Industrial Commissioner of Dust Control Equipment During Operation: All approved dust control equipment and methods shall be subject to inspection and test by the Industrial Commissioner during operation on any rock drilling work to which these rules apply; and tests to determine dust concentrations may be made by the Industrial Commissioner.

The employer engaged in rock drilling shall make frequent inspections and tests to determine whether the approved dust control methods and equipment are being operated and maintained to meet the approval of the Industrial Board;* and shall make written record of emergencies or difficulties resulting in insufficiency of operation.

* By act of the 1937 Legislature, approval is now a power of the newly created Board of Standards and Appeals.

CHAPTER VI COMPENSATION FOR SILICOSIS

Compensation for Silicosis in New York State.

Unquestionably the single most potent factor in silicosis prevention is the making of silicosis a compensable disease. Efforts to do this have been made in New York State from time to time since 1925, but it was only in 1936 that they were finally successful.

Sections of the Law dealing with compensation for silicosis in this State are given below:

CHAPTER 887, LAWS OF 1936

AN ACT to amend the Workmen's Compensation Law, in relation to occupational diseases and in relation to special provisions for compensation for certain injuries to the respiratory tract resulting from the inhalation of harmful dust, and to amend the Labor Law, in relation to control of harmful dust in public works.

Became a law June 6, 1936, with the approval of Governor Herbert H. Lehman.

The People of the State of New York, represented in Senate and Assembly, do enact as follows:

Section 1. Paragraph 28 of Subdivision 2 of Section 3 of Chapter 816 of the Laws of 1913, entitled "An act in relation to assuring compensation for injuries or death of certain employees in the course of their employment and repealing certain sections of the Labor Law relating thereto, constituting Chapter 87 of the Consolidated Laws," as re-enacted by Chapter 41 of the Laws of 1914, such paragraph having been added by Chapter 254 of the Laws of 1935, is hereby amended to read as follows:

28. Any and all occupational diseases

28. Any and all employments enumerated in Subdivision 1 of Section 3 of this Chapter.

Nothing in Paragraph 28 of this Subdivision shall be construed to apply to any case of occupational disease in which the last injurious exposure to the hazards of the disease occurred prior to September first, 1935; nor to any disability or death due to any disease described in Article 4-a of this Chapter.

2. Such Chapter is further amended by the insertion therein of a new Article, to follow Article 4, to be Article 4-a, to read as follows:

ARTICLE 4-a

Silicosis, and Other Dust Diseases

Section 65. Prevention of silicosis and other dust diseases.

66. Compensation payable for disability or death.

67. Liability of employer.

68. Medical treatment and care.

69. Workers, when not entitled.

70. Special medical examiners.

71. Expert consultants.

72. Alternate remedy.

65. Prevention of silicosis and other dust diseases. 1. It is hereby declared to be the policy of the Legislature of this State, in enacting this Article, to prohibit through every lawful means available, any requirement as a pre-requisite to employment which compels an applicant for employment in any occupation coming within the purview of this Article to undergo a medical examination.

2. The Industrial Commissioner and the Industrial Board are hereby required to add to the Industrial Code, as provided in Sections 28 and 29 of the Labor Law, effective rules and regulations governing the installa-

tion, maintenance and effective operation in all industries and operations wherein silica dust or other harmful dust hazard is present, of approved devices designed to eliminate such harmful dusts and to promulgate such other regulations as will effectively control the incidence of silicosis and similar diseases.

66. **Compensation payable for disability or death.** Compensation shall not be payable for partial disability due to silicosis or other dust disease. In the event of temporary or permanent total disability or death from silicosis or other dust disease, notwithstanding any other provision of this Chapter, compensation shall be payable under this Article to employees in the employments enumerated in Section 3 of this Chapter or to their dependents in the following manner and amounts: If disablement or death occur during the first calendar month in which this Act becomes effective not exceeding the sum of \$500; if disablement or death occur during the second calendar month after which this Act becomes effective not exceeding the sum of \$550; thereafter the total compensation and benefits payable for disability and death shall increase at the rate of \$50 each calendar month. The aggregate amount payable shall be determined by the total amount payable in the month in which disablement or death occurs. In no event shall such compensation exceed an aggregate total of \$3,000. The requirement as to payments into the special funds provided for in Subdivisions 8 and 9 of Section 15 for each case of injury causing death in which there are no persons entitled to compensation shall not apply to any claim arising under this article.

Compensation payable hereunder shall be paid from the eighth day following total disablement at the rate of 66 and two-thirds per centum of the average weekly wage to be computed under Section 14 of this Chapter; but in no case shall compensation exceed \$25 per week nor in the event of total disability be less than eight dollars per week; provided, however, that in the event of death from such disease his dependents shall receive, in the manner provided by Sections 16 and 17 of this Chapter, any balance remaining between the amounts paid for disability and the total compensation payable under this article.

Notwithstanding the provisions of Section 28 of this Chapter, all claims for compensation resulting from inhalation of harmful dust, where the last exposure occurred between the effective date of this Act and September 1, 1935, shall be barred unless filed within 180 days from the day on which this Act takes effect.

67. **Liability of employer.** An employer shall be liable for the payments prescribed by this Article for silicosis or other dust disease when disability of an employee resulting in loss of earnings shall be due to an employment in a hazardous occupation in which he was employed, and such disability results within one year after the last injurious exposure in such employment; or, in case of death resulting from such exposure, if such death occurs within five years following continuous disability from such disease. The provisions of Section 44 of this Chapter shall not apply to claims arising under this Article.

The employer in whose employment the employee was last injuriously exposed in a hazardous occupation and the insurance carrier, if any, which was on the risk at the time of the last injurious exposure in such employment, shall be liable for any payments required by this Article; the notice of injury and claim shall be made to such employer.

68. **Medical treatment and care.** Notwithstanding any other provisions of this Chapter the medical treatment herein provided for shall be limited in the case of an employee disabled by an occupational disease due to or resulting from the inhalation of harmful dust to a period of 90 days from the date of such disablement, but the requirement for such medical treatment may be extended for an additional period not to exceed 90 days upon the order of the Industrial Board.

69. **Workers, when not entitled.** If an employee, at the time of his employment, falsely represents in writing that he has not previously been

disabled from the disease which is the cause of disability or death or has not received compensation or benefits under this Article, no compensation shall be payable.

70. Special Medical Examiners. The Industrial Commissioner shall divide the State into five districts and in each district may appoint two or more Special Medical Examiners who shall be licensed physicians in good professional standing, each of whom shall have had, at the time of his appointment, and immediately prior thereto at least five years of practice in the diagnosis, care and treatment of pulmonary diseases. Such Examiners shall be employed on a per diem basis as the exigencies of the work may require. Fees of Examiners shall be fixed by the Industrial Commissioner within the limits of the appropriation therefor. Each position of Special Medical Examiner provided herein shall be in the exempt class of Civil Service.

Whenever a claim is made under this Article and an examination of the claimant by an impartial physician is desired by any party in interest, the Industrial Commissioner shall order such Medical Examiners to make the necessary medical and X-ray examinations of the claimant in an effort to obtain the medical facts in an impartial manner.

For the purposes of adjudication under this Chapter, the Industrial Board* shall adopt rules of practice and procedure and shall prescribe methods and standards under which physical examinations, X-rays and other studies shall be conducted.

71. Expert Consultants. The Industrial Commissioner shall appoint as Expert Consultants on dust diseases three licensed physicians in good professional standing, each of whom shall have had, at the time of his appointment, and immediately prior thereto, at least 10 years of practice in the diagnosis, care and treatment of diseases of the pulmonary tract, along with the interpretation of X-ray films thereof. They shall be paid at a salary to be fixed by the Industrial Commissioner not to exceed \$7,500 per year. Each such position of Consultant shall be in the exempt class of civil service.

The Industrial Commissioner or the Industrial Board* shall on their own volition or on the application of either an employee, an employer, or an insurance carrier, direct such Expert Consultants to make examinations of claimants, to review the findings of Special Medical Examiners, to read and review the files of compensation cases when necessary, and to inform the Industrial Commissioner and the Industrial Board* of their opinion as to their findings in such cases.

72. Alternative remedy. The liability of an employer prescribed by this Article shall be exclusive and in place of any other liability whatsoever, at common law or otherwise, to such employee, his personal representatives, husband, parents, dependents or next of kin, or anyone otherwise entitled to recover damages, at common law, or otherwise on account of any injury, disability, or death, caused by the inhalation of harmful dust, except that if an employer fail to secure the payment of compensation for his injured employees and their dependents as provided in Section 50 of this Chapter, an injured employee, or his legal representative in case death results from the injury or disease, may, at his option, elect to claim compensation under this Chapter, or to maintain an action in the courts for damages on account of such injury or disease; and in such an action it shall not be necessary to plead or prove freedom from contributory negligence nor may the defendant plead as a defense that the injury or disease was caused by the negligence of a fellow servant or that the employee assumed the risk of his employment, nor that the injury or disease was due to the contributory negligence of the employee.

3. Chapter 50 of the Laws of 1921, entitled "An act in relation to labor, constituting Chapter 31 of the Consolidated Laws," is hereby amended by inserting therein a new Section, to be Section 222-a, to read as follows:

222a. Prevention of dust hazard in public works. In the construction of public works by the State or a public benefit corporation or a municipal

* By act of the 1937 Legislature this power is now a function of the Board of Standards and Appeals.

corporation or a commission appointed pursuant to law wherein a harmful dust hazard is created for which appliances or methods for the elimination of harmful dust have been approved by the Industrial Board, a provision shall be inserted in each contract for the construction of such work requiring the installation, maintenance and effective operation of such appliances and methods, and a further provision shall be inserted in such contract that if this Section is not complied with, the contract shall be void. In the construction of public works performed directly by the State or a public benefit corporation or a municipal corporation or a commission appointed pursuant to law wherein a harmful dust hazard is created for which appliances or methods for the elimination of silica dust or other harmful dust have been approved by the Industrial Board, the department, board or officer in the State, public benefit corporation, or municipal corporation or commission or board appointed pursuant to law, having jurisdiction over the construction of such work shall provide for the effective use of such approved appliances or methods in connection therewith. A violation of this Section shall constitute a misdemeanor and shall be punishable by a fine of not more than \$500 or by imprisonment for not more than one year or by both fine and imprisonment.

4. This Act shall take effect immediately.*

Compensation for Silicosis in Other States

The rapidly developing interest in and knowledge of conditions affecting the health of workers has resulted in quite a marked recent increase in State laws providing for occupational disease. As is well known such laws fall into two groups (1) those covering all occupational diseases in a general or "blanket" law and (2) those listing special diseases for which compensation will be allowed.

Silicosis at the date of writing† is covered by "blanket" laws in the following states:

- California.
- Connecticut.
- District of Columbia.
- Illinois.
- Indiana.
- Massachusetts—by court definition of injury.
- Missouri—provisional upon employer's wish.
- Nebraska—limited to a few industries.
- North Dakota—if it can be considered to result from "accident."
- Wisconsin.

Silicosis is included in schedule laws in the following states:

- Kentucky.
- North Carolina.
- Ohio.
- Michigan.
- West Virginia.
- Washington.

* June 6, 1937.

† June, 1937.

BIBLIOGRAPHY

1. Gardner, L. U.: Pathology of the pneumoconioses. N. Y. State Jour. Med., 36, 1377, 1936.
2. Lanza, A. J. and Vane, R. J.: The prevalence of silicosis in the general population and its effects upon the incidence of tuberculosis. Am. Rev. Tuberc., 29, 8, 1934.
3. Britten, R. H.: Occupational mortality among males in England and Wales, 1921-1923. A summary of the report of the registrar general. Pub. Health Repts. Repr., 1233, 1928.
4. Irvine, L. G., Simson, F. W. and Strachan, A. S.: The clinical pathology of silicosis. Silicosis. International Labor Office. Geneva, 1930.
5. Thompson, L. R. and Britten, R. H.: The silica dust hazard in the granite cutting industry. Jour. Ind. Hyg., 12, 123, 1930.
6. Dreessen, W. C. and Jones, R. R.: Anthracosilicosis. Jour. Am. Med. Assoc., 107, 1179, 1936.
7. Winslow, C. E. A. and Greenburg, L.: A study of the dust hazard in the wet and dry grinding shops of an ax factory. Pub. Health Repts., 35, 2393, 1920.
8. Collis, E. L. and Yule, G. U.: The mortality experience of an occupational group exposed to silica dust, compared with that of the general population and an occupational group exposed to dust not containing silica. Jour. Ind. Hyg., 15, 395, 1933.
9. Gardner, L. U.: Silicosis and related conditions. Jour. Ind. Hyg., 19, 111, 1937.
10. Audley, J. A.: Silica and the silicates. Bailliere, Tindall and Cox, London, 1921.
11. Mellor, J. W.: Modern inorganic chemistry. Longmans, Green & Co., New York, 1922.
12. Gregory, T. C.: The condensed chemical dictionary. Chemical Catalog Co., New York, 1930.
13. Jones, W. R.: Silicotic lungs, the minerals they contain. Jour. Hyg., 33, 307, 1933.
14. Gardner, L. U.: The experimental production of silicosis. Pub. Health Repts., 50, 695, 1935.
15. Ladoo, R. B.: Nonmetallic minerals. Occurrence, Preparation, Utilization. McGraw-Hill Book Co. New York, 1925.
16. Silicosis. Records of the International Conference held at Johannesburg, August 1930. International Labor Office. Geneva, 1930.
17. Middleton, E. L.: Industrial pulmonary disease due to inhalation of dust. Lancet, 2, 1, 59, 1936.
18. Drinker, P. and Hatch, T.: Industrial dust. McGraw-Hill Book Co. Inc. New York and London, 1936.
- 18a. National Silicosis Conference. Summary reports submitted to the Secretary of Labor by Conference Committees, February 3, 1937. Bulletin 13. United States Dep't. of Labor, Division of Labor Standards.
19. Gye, W. E. and Purdy, J. S.: The poisonous properties of colloidal silica; the effects of the parenteral administration of large doses. Brit. Jour. Exp. Path., 3, 75, 1922.
20. Mills, R. G.: The effect of prolonged exposure of the siliceous spicules of a fresh water sponge (*spongilla fragilis*) to the action of animal tissues. Am. Jour. Hyg., 13, 224, 1931.

21. Belt, T. H.: The pathology of silicosis of the lung. *Can. Pub. Health Jour.*, 20, 495, 1929.
22. Heffernan, P. and Green, A. T.: The method of action of silica dust in the lung. *Jour. Ind. Hyg.*, 10, 272, 1928.
23. Cummins, S. L. and Weatherall, C.: Effects of colloidal silica upon the growth of tubercle bacilli in blood. *Brit. Jour. Exper. Path.*, 12, 245, 1931.
24. Gye, W. E. and Kettle, E. H.: Silicosis and miners' pthisis. *Brit. Jour. Exper. Path.*, 3, 241, 1922.
25. Gardner, L. V.: Will the inhalation of siliceous dusts activate a partially healed focus of tuberculous infection? *Pub. Health Repts.*, 45, 282, 1930.
26. Dowd, G.: A bacteriological study of attenuated (R.) tubercle bacilli recovered from silicotic and normal guinea pigs. *Am. Rev. Tuberc.*, 32, 62, 1935.
27. King, E. J. and Dolan, M.: Silicosis and metabolism of silica. *Can. Med. Assoc. Jour.*, 31, 21, 1934.
28. Bloomfield, J. J. and Goldman, F. H.: The urinary excretion of silica by persons exposed to silica dust. *Pub. Health Repts.*, 50, 421, 1935.
29. Goldwater, L. J.: The urinary excretion of silica in non-silicotic humans. *Jour. Ind. Hyg.*, 18, 163, 1936.
30. Sweany, H. C., Porsche, J. D. and Douglass, J. R.: Chemical and pathological study of pneumoconiosis with special emphasis on silico-tuberculosis. *Arch. Path.*, 22, 593, 1936.
31. Miller, J. W. and Sayers, R. R.: The physiological response of the peritoneal tissue to dusts introduced as foreign bodies. *Pub. Health Repts.*, 49, 80, 1934.
32. McCord, C. P., Fleming, R. L., Ainslee, H. and Johnston, J.: The measurement of the harmfulness of dusts for humans through the agency of animal reactions. *Surg. Gyn. and Obstet.*, 63, 129, 1936.
33. Stüber, K.: The cellular response of lymph nodes to various dust suspensions introduced into lymphatics. *Jour. Ind. Hyg.*, 16, 282, 1934.
34. Sutherland-Strachan, A. and Simson, F. W.: A preliminary study of the pathology of silicosis as seen on the Witwatersrand. *Silicosis, International Labor Office. Geneva, 1930.*
35. Willis, H. S.: Pneumoconiosis and tuberculosis. *Medicine*, 9, 413, 1930.
36. McNally, W. D.: Silicon dioxide content of lungs in health and disease. *Jour. Am. Med. Assn.*, 101, 584, 1933.
37. Pancoast, H. K. and Pendergrass, E. P.: A review of our present knowledge of pneumoconiosis based on roentgenological studies with notes on the pathology of the condition. *Amer. Jour. Roentgenol.*, 14, 381, 1925.
38. Lehmann, G.: The significance of the filtering action of the nose on the incidence of pneumoconiosis. *Med. Wehnschr.*, 80, 1166, 1933.
- 38a. Proske, H. O. and Sayers, R. R.: Pulmonary infection in pneumoconiosis. A bacteriologic and experimental study. *Pub. Health Repts.*, 49, 839, 1934.
39. Dible, J. H.: Silicosis and malignant disease. *Lancet*, 982, 1934.
40. Berblinger, W.: The increase of lung cancer and diseases of the lung caused by dust. *Abst. Med. Klin.*, 27, 1337, 1931, *Am. Jour. Can.*, 16, 839, 1932.
41. Teleky, L.: Occupational cancer of the lung. *Jour. Ind. Hyg. and Tox.*, 19, 73, 1937.

42. Pancoast, H. K. and Pendergrass, E. P.: A review of pneumoconiosis: further roentgenological and pathological studies. *Am. Jour. Roentgenol.*, 26, 574, 1931.
43. Gardner, L. U.: The diagnosis of silicosis with special reference to roentgenological manifestations. *Ann. Int. Med.*, 10, 166, 1936.
44. Pancoast, H. K., Pendergrass, E. P., Riddell, A. R., Lanza, A. J., McConnell, W. J., Sayers, R. R., Sampson, H. L. and Gardner, L. U.: Roentgenological appearances in silicosis and the underlying pathological lesions. Report by Committee Composed of doctors. *Pub. Health Repts.*, 50, 989, 1935.
45. Irwin, D. A.: Histological demonstration of siliceous material by micro-incineration. *Canad. Med. Assoc. Jour.*, 31, 135, 1934.
46. Report of the Committee on Pneumoconiosis of the Am. Pub. Health Assoc. *Am. Pub. Health Assoc. Year Book*, 1932-33.
47. Kessler, H. H.: Silicosis in the abrasive powder industry. *Am. Jour. Pub. Health*, 21, 1930, 1931.
48. Chapman, E. W.: Acute silicosis. *Jour. Am. Med. Assoc.*, 98, 1439, 1932.
49. Kilgore, E. S.: Pneumoconiosis, an unusually acute form. *Jour. Am. Med. Assoc.*, 99, 1414, 1932.
50. An investigation relating to health conditions of workers employed in the construction and maintenance of public utilities. Hearings before a subcommittee of the committee on labor. House of Representatives 74th Congress. Second session on H. J. Res. 449, 1936.
51. Gardner, L. U.: Pathology of acute silicosis. *Am. Jour. Pub. Health*, 23, 1240, 1933.
52. Sampson, H. L.: The roentgenograms in so-called acute silicosis. *Am. Jour. Pub. Health*, 23, 1237, 1933.
53. Ellman, P.: Pulmonary asbestosis, its clinical, radiological and pathological features and associated risk of tuberculous infection. *Jour. Ind. Hyg.*, 15, 165, 1933.
54. Lanza, A. J., McConnell, A. J. and Fehnel, J. W.: Effects of the Inhalation of asbestos dust on lungs of asbestos workers. *Pub. Health Repts.*, 50, 1, 1935.
55. Commonwealth of Pennsylvania, Asbestosis. *Spec. Bull.*, 42, 1935.
- 55a. Donnelly, J.: Pulmonary asbestosis, incidence and progress. *Jour. Ind. Hyg.*, 1936.
- 55b. McPheeters, S. B.: A survey of a group of employees exposed to asbestos dust. *Jour. Ind. Hyg.*, 1936.
56. Thompson, L. R., Brundage, D. K., Russell, A. E. and Bloomfield, J. J.: The health of workers in dusty trades. I. Health of workers in a Portland cement plant. *Pub. Health Serv. Bull.*, 176, 1928.
57. Legge, R. T. and Rosencrantz, E.: Observations and studies on silicosis by diatomaceous silica. *Am. Jour. Pub. Health*, 22, 1055, 1932.
58. Commonwealth of Massachusetts, Report to the General Court of the Special Industrial Disease Commission. House No. 1350, 1934.
59. McConnell, W. J. and Fehnel, J. W.: Health hazards in the foundry industry. *Jour. Ind. Hyg.*, 16, 227, 1934.
60. Warfield, L. M.: Results of the chest examinations of 2,500 in a heavy industry plant. *Ind. Med.*, 4, 302, 1935.
- 60a. Kelly, J. F. and Hall, R. C.: Silicosis in modern foundries. *N. Y. State Jour. Med.*, 37, 1, 1937.

- 60b. Osmond, L. H.: Dust hazard among foundrymen. *Am. Jour. Roentgenol.*, 38, 122, 1937.
- 60c. Sander, O. A.: The lung findings in foundry workers. A four year survey. Paper read before Ind. Hyg. Section. A. P. H. A. at 66th Annual Meeting, N. Y. C., 1937.
61. Hoffman, F. L.: The problem of dust phthisis in the granite-stone industry. *U. S. Bur. Labor Stat.*, Bull. 293, 1922.
62. Russell, A. E., Britten, R. H., Thompson, L. R. and Bloomfield, J. J.: The health of workers in dusty trades. II. The exposure to siliceous dust (granite industry). *Pub. Health Serv. Bull.*, 187, 1929.
63. Smith, A. R.: A study of granite cutting and granite cutters in the vicinity of New York City. *Am. Jour. Pub. Health*, 24, 821, 1934.
64. Bloomfield, J. J. and Dreessen, W. C.: Silicosis among granite quarriers. *Pub. Health Repts.*, 49, 679, 1934.
65. The health of workers in dusty trades. Exposure to the dusts of a silverware manufacturing plant. *Pub. Health Serv. Bull.*, 28, 1933.
66. Smith, A. R.: Silicosis among metal polishers. *Ind. Bull.*, 16, 6, 1937.
67. Higgins, E. and Lanza, A. J.: Pulmonary disease among miners in the Joplin District, Missouri, and its relation to rock dust in the mines. *U. S. Bur. Mines Tech. Paper* 10, 1915.
68. Childs, S. B. and Lanza, A. J.: Miners' consumption, a study of 433 cases of the disease among zinc miners in southwestern Missouri, with a chapter on roentgen ray findings in miners' consumption. *Pub. Health Serv. Bull.*, 85, 1919.
69. Harrington, D. and Lanza, A. J.: Miners' consumption in the mines of Butte (Montana). *U. S. Bur. Mines Tech. Paper* 260, 1921.
70. Sayers, R. R., Meriwether, F. V., Lanza, A. J. and Adams, W. W.: Silicosis and tuberculosis among miners of the tri-state district of Oklahoma, Kansas and Missouri. *U. S. Bur. Mines, Tech. Paper* 545, 1933.
71. Sayers, R. R., Bloomfield, J. J., Dallavalle, J. M. et al.: Anthracosilicosis among hard coal miners. *Pub. Health Serv. Bull.* 221, 1935.
72. Quaintance, P. A.: Silicosis, a study of 106 pottery workers. *Am. Jour. Pub. Health*, 24, 1244, 1934.
73. Smith, A. R. and Fehnel, J. W.: Silicosis among rock drillers, blasters and excavators in New York City based on a study of 208 examinations. *Jour. Ind. Hyg.*, 11, 37, 1929.
74. Winslow, C. E. A., Greenburg, L. and Greenberg D.: The dust hazard in the abrasive industry. Incidence of tuberculosis among workers exposed to mineral and metallic dusts. *Pub. Health Repts.*, 34, 1171, 1919.
75. Greenburg, L. and Winslow, C. E. A.: The dust hazard in air-pressure abrasive blasting (sand blasting). *Arch. f. Gewerbepath. u. Gewerbehyg.* 3, 577, 1932.
76. Hayhurst, E. R. and Kindel, D. J.: Stereoscopic X-ray examination of sandstone quarry workers. *Am. Jour. Pub. Health*, 17, 818, 1927.
77. Dreessen, W. C.: Effects of certain silicate dusts on the lungs (talc and slate). *Jour. Ind. Hyg.*, 15, 66, 1933.
78. National Safety Council. Final report of the spray coating committee, 1927.
79. Dreessen, W. C. and Dallavalle, J. M.: The effects of exposure to dust in two Georgia talc mills and mines. *Pub. Health Repts.*, 50, 131, 1935.

80. Goldwater, L. J.: Incidence of silicosis in trap rock quarry workers as determined by X-ray. Jour. Ind. Hyg., 18, 550, 1936.
81. Hatch, T. and Pool, C. L.: Quantitation of impinger dust samples by dark field microscopy. Jour. Ind. Hyg., 16, 177, 1934.
82. Proceedings of Conference Concerning effects of Dust upon the Respiratory System. Industrial Commission of Wisconsin. Chicago, 1933.
83. Clark, W. I.: The dust hazard in the abrasive industry. Jour. Ind. Hyg., 11, 92, 1929.
84. Bloomfield, J. J. and Greenburg, L.: Sand and metallic abrasive blasting as an industrial health hazard. Jour. Ind. Hyg., 15, 184, 1933.
85. Greenburg, L., Siegal, W. and Smith, A. R.: Silicosis in the foundry industry. New York State Department of Labor, Spec. Bull. 197, 1938.

INDEX

A

	PAGE
Anthracosis.....	9
Asbestos.....	13
bodies.....	35
industry, studies.....	36
Asbestosis.....	9, 34
diagnosis.....	34
liability to tuberculosis in.....	34
pathology.....	34
physical signs.....	34
symptoms.....	34
X-ray appearances.....	34
Anthraco-silicosis.....	17

B

Blasting—see rock-drilling.....	39
Buhrstone.....	12, 14

C

Cadmium.....	19
Cement industry.....	16
dust counts in.....	36
respiratory disease in.....	36
study of U. S. P. H. S.....	36
tuberculosis in.....	36
Chalcosis.....	9
Chert.....	13, 14, 19
Chrysolite.....	13
Clay.....	13
Coal.....	10, 39
Coal mining industry.....	39
silicosis in.....	39
study U. S. P. H. S.....	39
tuberculosis in.....	39
Copper mining industry.....	38
study in.....	38
Cornstarch.....	19
Cristobalite.....	12

D

Diamond.....	18
Diatomaceous earth.....	13, 14, 37
analysis for free silica.....	37
Dust, counting.....	41
particle size.....	42
Dust cells.....	19, 20
Dustiness, degree of.....	16
Dustiness, safe standards.....	17

E

Education, in silicosis prevention....	46
Electric precipitation.....	42
Emery.....	18
Excavating—see rock-drilling.....	39

F

	PAGE
Feldspar.....	13
Ferric oxide.....	19
Fibrosis, in silicosis.....	20, 28
Flint.....	12, 13, 14, 15
Foundry industry.....	15
respiratory disease in.....	37
silicosis in.....	37
studies.....	37
tuberculosis in.....	37
Fresh water sponge, experiments with	18

G

Ganister.....	13, 14
Gold mining industry, silicosis in....	15
Granite industry.....	10, 13, 15, 17, 37
dust counts.....	37
silicosis in.....	37
studies.....	37
tuberculosis in.....	26, 37
Grave diggers, silicosis in.....	15
Grinding.....	10
dust hazard.....	38

H

Helmets.....	46
Housekeeping, in silicosis prevention.	46
Hydrous magnesium silicate.....	13

I

Impinger.....	41
Iron.....	18

K

Konimeter.....	41
----------------	----

L

Leather dressers, silicosis in.....	15
Lead and zinc mining industry....	38, 39
silicosis in.....	38
studies.....	38
tuberculosis in.....	38

M

Macrophages.....	19
Manganese.....	19
Masks.....	46
Medical supervision, in silicosis pre- vention.....	46

M	PAGE
Metallurgists, silicosis in.....	15
Metal polishing.....	14, 38
dust counts.....	38
silicosis in.....	38
studies.....	38
Mica.....	13, 15
Milling.....	15
Mining.....	38
coal.....	10, 39
copper.....	38
gold.....	15
lead and zinc.....	38, 39

N	
National Silicosis Conference.....	17
Negroes, susceptibility to silicosis....	17

O	
Owen jet dust counter.....	41

P	
Pegmatite.....	13
Phagocytes.....	19
Pneumoconiosis.....	9
Pottery industry.....	15, 39
composition dust.....	39
silicosis in.....	39
study in.....	29
Pulmonary lymphatic system.....	19

Q	
Quarrying.....	15, 38
Quartz.....	12, 14, 15, 16
Quartzite.....	13, 14

R	
Respiratory infection, predisposing to silicosis.....	17
Rock crystal.....	12
Rock drilling.....	15, 39
code.....	48
composition dust.....	39
silicosis in.....	39
study of.....	39
tuberculosis in.....	39
Rotten stone.....	13

S	
Sand.....	13, 14, 15
Sand blasting.....	13, 15, 39
dust counts.....	39
studies.....	39
Sandstone quarrying.....	15, 39
silicosis in.....	39
study.....	39
tuberculosis in.....	39
Semi-precious stones.....	12, 14
Sericite.....	13
Siderosis.....	9

S	PAGE
Silica.....	9
amorphous.....	12, 13, 14, 16
chemical properties.....	12
colloidal.....	18
combined.....	12
crystalline.....	12, 16
free.....	12, 16
industries with exposure to.....	15
non-crystalline, see amorphous.	
number exposed, N. Y. State.....	9
number exposed U. S.....	9
operations with exposure to.....	13, 14, 15
peritoneal reaction to.....	19
solubility.....	12, 18
toxic properties.....	11, 18
uses.....	13, 14, 15
urinary secretion.....	18
vitreous.....	12
Silica gel.....	18
Silica in.....	
abrasives.....	14, 15
acid towers.....	14
blood.....	19
building stone.....	14
ceramics.....	14
chemical apparatus.....	14
decorative materials.....	14
ferrosilicon.....	14
fertilizers.....	14
filler.....	14
flux.....	14
foundry mold wash.....	14
gems.....	14
grindstones.....	14
glass.....	14, 15
insecticides.....	14
insulation.....	14
lung tissue.....	25
lymph node reaction.....	19
metal buffing.....	14
monuments.....	14
paint.....	14
paving stones.....	14
refractories.....	14, 15
sand blasting.....	14
sand paper.....	14
silicon alloys.....	14
sodium silicate.....	14
sputum.....	19
structural materials.....	14
surfacing mixtures.....	14
tooth powders and pastes.....	14
tube mills.....	14
Silica sol.....	18
Silicates.....	13, 16
Silicatosi.....	13
Silicon dioxide—see silica	
Silicosis.....	9
acute.....	32
and cancer.....	31

S	PAGE	S	PAGE
and tuberculosis.....	9, 22, 26	Sillimanite.....	13
arborization in.....	27	Slate.....	13, 15
bronchiolitis in.....	20	Slate milling.....	40
compensation.....	50	dust counts in.....	40
New York State.....	50	study in.....	40
other states.....	63	Spray coating.....	40
complications.....	31	dust counts.....	40
course.....	26	silica in enamels.....	40
definition.....	9	silicosis in.....	40
diagnosis.....	31	study in.....	40
early.....	32	Stone works.....	15
factors in development.....	16		
first stage.....	32		
infection in.....	30	T	
length of exposure.....	16	Talc.....	13
linear markings in.....	26	Talc mining industry.....	40
management.....	32	dust counts.....	40
physical signs.....	26	studies in.....	40
pleural plaques, in.....	22	Thermal Precipitator.....	42
post mortem appearance in.....	24	Trap rock quarrying.....	40
predisposing factors.....	25	composition rock.....	40
prevention.....	41, 42	silicosis, in.....	40
pseudo tubercle in.....	21	study in.....	40
rate of development.....	25	Tremolite.....	13
respiratory infection in.....	25, 31	Tridymite.....	12
second stage.....	32	Tripoli.....	13, 14
simple.....	30	Tuberculosis, mortality rates.....	9, 10
standard terminology.....	30	Tuberculosis and silicosis—see silicosis	
susceptibility to.....	17	Tunnel construction.....	15
symptoms.....	25		
third stage.....	32	V	
tracheo-bronchial lymph nodes in.....	20	Ventilation, in silicosis prevention.....	44, 46
X-ray appearances.....	26, 30	Vitreous enamelling.....	15
X-ray technique for.....	32		
Silico-tuberculosis.....	23, 29, 32	W	
diagnosis.....	32	Water, use of in silicosis prevention.....	46
symptoms.....	26		
X-ray appearances.....	29		