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Dakota, Colorado, and Wisconsin) indicates that the severity of dental caries is, in general, lower in mottled enamel areas as compared with normal areas in the same State.

3. Inasmuch as it appears that the mineral composition of the drinking water may have an important bearing on the incidence of dental caries in a community, the possibility of partially controlling dental caries through the domestic water supply warrants thorough epidemiological-chemical study.

ACKNOWLEDGMENT

The outline of the artesian basin in eastern South Dakota shown in figure 1 was taken from plate LXIX, by N. H. Darton, in part 2 of the Seventeenth Annual Report of the United States Geological Survey, 1895–96.

The writer desires to express his indebtedness to Senior Statistician Wm. M. Gafafer and Senior Chemist E. Elvove, National Institute of Health, for many helpful suggestions and criticisms in the preparation of this paper, to the Wisconsin State Board of Health for supplying information on the fluoride content of the water supplies of the seven Wisconsin cities with high dental caries attack rates, and to Principal Statistician Selwyn D. Collins, National Institute of Health, for a review of the paper.

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SILICOSIS AND SIMILAR DUST DISEASES

Medical Aspects and Control

By R. R. Sayers, *Senior Surgeon*, and R. R. Jones, *Passed Assistant Surgeon*, *United States Public Health Service*

The pneumoconioses represent a class or type of diseases of the lung which develop slowly as a result of occupational exposure. The term "pneumoconiosis" may be applied to any pathological condition of the lung produced by the inhalation of dust. It is common further to classify pneumoconiosis according to the chief constituents of the dust producing the condition; for example, silicosis produced by silica, asbestosis produced by asbestos, siderosis produced by iron, anthracosis produced by carbon particles, etc. Of these, silicosis and asbestosis are by far the most important from the viewpoint of number of workers exposed and the disability associated with their development.

Since silicosis is the most important, and control measures effective in preventing silicosis are applicable in the prevention of other forms of pneumoconiosis, this discussion will be limited chiefly to reporting a summary of our knowledge concerning the cause and prevention of silicosis.

The Committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association (*1*), defined silicosis as follows:

> 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.

This definition of the disease seems to be satisfactory from a medical point of view; however, in a law recently passed in West Virginia, for the purpose of the act silicosis was defined as an insidious fibrotic disease of the lung or lungs, due to prolonged inhalation and accumulation, sustained in the course of and resulting from employment, of minute particles of dust containing silicon dioxide (SiO_2) over such a period of time and in such amounts as result in the substitution of fibrous tissue for normal lung tissues; and the term silicosis shall also include silicosis accompanied by tuberculosis of the lungs evidenced by the presence of tubercle bacillus in the sputum.

While the foregoing definitions of silicosis were obtained from sources that might be expected to have somewhat different views, all have one thing in common—that the cause is silica or quartz.

Although other dusts, when inhaled in sufficient concentrations over a long period of time, have been shown to be capable of producing a definite pulmonary fibrosis, nevertheless the

acterized by nodular fibrosis has to date been shown clinically and experimentally to be associated only with the inhalation of dusts containing silica.

According to Dorland's Medical Dictionary, "Pneumoconiosis is a lung disease due to the inhalation of minute particles of dust. It is attended by fibroid induration." Pneumoconiosis is a general term, and the lung disease caused by any one dust usually takes the name of that dust, as anthracosis, silicosis, chalicosis, siderosis, and asbestosis.

SILICA IN NATURE

Silica is the most abundant constituent of the minerals and rocks that make up the crust of the earth. It occurs in two forms, free and combined. The free silicas as a group are definite compounds in the form of SiO_2. The combined forms are silicates. Of free silicas which occur in nature, that known as quartz is by far the most common. Quartz is hard mineral and chemically resistant to reagents. It is an abundant constituent of granite, schist, and other rocks, and the chief component of sandstone and quartzite. Many ores are deposited in veins that consist nearly wholly of quartz. Probably the next most common form in which free silica exists in nature is the amorphous hydrated form known as opal (SiO_2–H_2O). Opal is a silica of colloidal origin and occurs abundantly in the diatomaceous earths. It is less resistant to reagents than quartz. Another type of free silica frequently found is flint; and with flint is found chalcedony, a waxy, translucent form of silica interpreted as consisting of fibers of quartz with a small amount of interstitial opal. Other forms of free silica occurring less abundantly in nature are tridymite, cristobalite, and siliceous glass, or vitreous silica.

OCCUPATIONAL EXPOSURE TO SILICA

Owing to the fact that the earth's crust contains so great an amount of silica, it is obvious that those occupations concerned with the driving of tunnels, development of highways, and mining are frequently associated with a silicosis hazard. A second class of occupations exposing the workers to this hazard are those connected with industries that have to do with the processing and industrial use of mineral products, such as the smelting and refining of ores, the use of sand and gravel for structural purposes, the carving of stone, particularly granite, the manufacture and use of certain abrasives, and the processing of the various forms of free silica. According to Knopf (*2*), the most common forms of free silica used industrially are massive crystalline quartz, quartzite, sandstone, flint, tripoli, diatomaceous earths, and silica sand. Table 1, from Ladoo (*3*), illustrates the great variety of uses to which silica is put in industry and shows the kind of silica adapted to each purpose.

TABLE 1.—*Uses of silica*

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.
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, hubstones, pulpstones, oilstones, etc.	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 pastes	Various forms of pure silica finely ground.
Wood polishing and finishing	All forms of silica ground to medium fineness.
Refractory uses: In making silica firebrick and other refractories	Fairly pure quartzite known as ganister; not less than 97 percent SiO_2 nor more than 0.40 percent alkalies, tightly interlocking grains desired.
Metallurgical uses:	
In making silicon, ferrosilicon, 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 carborundum	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	
In insecticides	
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	
Ceramic uses: In the pottery industry as an ingredient of bodies and glazes	Flint, tripoli, and chert, and other amorphous silica preferred; also all other forms of very pure silica, all finely ground.
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.	Do.
Structural materials: Sand-lime brick	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.

In a recent survey (*4*) carried on in a large manufacturing center, it was found that about 9 percent of the industrial workers were employed in occupations where the silica hazard required consideration. According to the census for 1930, there were gainfully employed in the manufacturing and mechanical industries in this country approximately 14 million persons. If the above mentioned survey can be accepted as representative of the occupational distribution of these workers, it appears that there are nearly 1,200,000

turing and mechanical industries alone. Lanza and Vane (*5*), in their discussion concerning the prevalence and effect of silicosis, state: "Our very rough, but obviously conservative, estimate of the number of workers exposed to silica dust to a harmful degree in the United States is, therefore, upwards of 500,000."

FACTORS INFLUENCING THE ACTION OF SILICA DUST PARTICLES AS THE EXCITING CAUSE OF SILICOSIS

Although it has been accepted that silica is the exciting cause of silicosis, there are certain factors which must be considered as influencing its action.

Tissue reaction to dust.—Early workers were inclined to consider that the injury caused by the dust particle was due to mechanical irritation produced by its hard and cutting edges. Gardner (*6*) has shown experimentally that the inhalation of finely divided carborundum dust having sharp edges, of a greater hardness than silica particles, does not produce the nodular reaction characteristic of silicosis. Collis (*7*) was one of the early workers to draw our attention to the chemical action of dust. Gye and Kettle (*8*) have shown that silica in solution or noncrystalline form exerts a toxic action upon the tissues which leads to the proliferation of fibroblastic cells. Lately, Miller and Sayers (*9*) have reported results of experimental studies which illustrate clearly the reaction of peritoneal tissues to certain dusts. Only the silica-containing dusts have uniformly produced a proliferative reaction. Other dusts have been either completely absorbed, leaving no scar tissue, or have remained unaltered in the form in which they were injected. These latter reactions are classed as "absorptive" or "inert."

Size of dust particles.—Since dust, to exert its harmful action, must enter the finer divisions of the lung, the particle size of the atmospheric dust bears a definite relationship to the injurious effect produced. The silica must be present in the air in particles small enough to enter the finer air spaces and of such dimensions that the phagocytic cells may engulf them. The natural defenses of the respiratory tract probably prevent many particles larger than 10 microns from ever reaching the finer divisions of the lung, and such as do are likely to be expelled with the bronchial secretions. The soluble silica plays a definite part in the production of the disease, and the size of the particle also affects the rate of solution, due to the fact that the smaller the particles the greater the total surface area exposed to the action of solvents.

Table 2 shows the size distribution of various industrial dusts as compared with the dust particles observed in the outdoor air in the general atmosphere. Apparently about 70 percent of the particles found in industrial dusts generally are between 0.5 and 3 microns in

TABLE 2.—*Size-frequency distribution of various industrial dusts as compared to outdoor dust* [1]

[Average (requency in percent]

Kind of dust	Number of samples	Median	Size group in microns											
			0–0.49	0.5–0.99	1–1.49	1.5–1.99	2–2.49	2.5–2.99	3–3.49	3.5–3.99	4–4.49	4.5–4.99	5–5.49	5.5–5.99
Outdoor dust	170	0.5	66.0	21.0	8.3	0.6								
Anthracite mining	9	1.4	19.7	18.0	24.7	20.3	12.6	6.2	2.8	1.6	1.1	0.2	0.2	0.3
Granite cutting	4	1.4	18.0	18.6	22.6	24.6	10.4	4.6	2.1	.6	.6	.3	1.0	
Open-pit mining:														
Crusher house	1	1.4	18.0	20.0	22.0	22.0	10.5	2.6	2.0					
Screen house	1	1.8	21.0	23.0	23.0	16.0	10.0	4.6	2.6	.6				
Disk crusher	1	.9	66.0	21.0	6.0	4.0	3.0	1.0	1.0					
Foundry parting compound	1	.6	72.0	17.0	5.2	2.8	1.6	1.0	1.6	2.0	.6			
General foundry air	1	1.2	56.0	22.0	9.0	5.0	3.0	1.0	1.0	2.0	1.0			
Granite milling	1	1.6	18.0	20.0	21.0	14.0	10.0	6.0	4.0	2.0	2.0	1.0	1.0	1.0
Marble cutting	1	1.0	12.0	20.0	21.0	21.0	11.0	6.0	3.0	1.0	1.0	2.0	2.0	1.0
Limestone	1	2.4	18.0	19.0	21.0	13.0	11.0	6.0	6.5	4.5	5.0	3.8	2.5	11.5
Limestone dust	1	2.2	8.0	10.0	20.5	14.0	11.5	6.0	6.6	5.0	5.5	4.0	7.0	10.0
Phosphate dust	1	1.5	12.0	22.0	23.5	21.0	6.0	6.0	1.6					
Anthracite-coal mining:														
Breaker air	2	1.0	71.0	21.0	25.0	4.0	3.0	2.0	2.0					
Mine air	1	.8	71.0	20.0	17.0	7.0	4.0	2.0	1.0					
Coal drilling	1	1.0	66.0	22.0	24.0	7.0	1.6		.5					
Coal loading	1	1.0	71.0	22.0	24.0	6.0	1.6	.7	.2					
Rock drilling	1	.6	66.0	23.0	23.0	12.5	5.5	1.6	.6	1.6				

[1] From Public Health Bulletin No. 217, *Determination and Control of Industrial Dusts*, by J. J. Bloomfield and J. M. Dalla Valle. 1935, p. 42.

diameter. There are no doubt many times as many particles too small to count by the method used, but experimentally it has been shown that a great percentage of such submicroscopic particles are not retained in the lungs but pass out with the expired air. Sayers (*10*) has shown that less than 15 percent is retained when the finer particulate matter, such as lead in the form of fumes, is inhaled. The greater majority of particles found upon microscopic examination of the lung also fall within the limits of from 1 to 3 microns.

Another reason for considering the size of the particles as affecting the harmfulness of the dust, is that it is the larger ones that settle out rapidly while the rate of falling for the smaller particles is very slow. Figure 1 illustrates graphically this difference; those under 1 micron

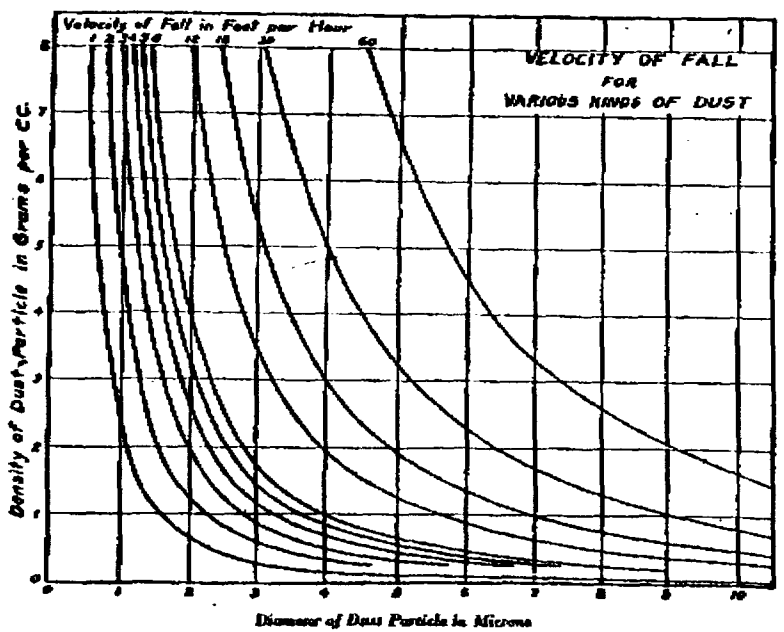

FIGURE 1.—Rapidity of settling in relation to size and density of dust particles.

fall at a rate of from 1 to 3 feet per hour, varying with the specific gravity, while a particle 5 microns in diameter of 7 specific gravity falls about 60 feet per hour. Particles of more than 10 microns would settle out in a relatively short time. The fact that the finer particles remain suspended in the atmosphere for long periods greatly increases their chance of being inhaled.

Thus we may say, from the viewpoint of etiology, that the harmfulness of a given dust containing free silica is directly influenced by the number of particles of free silica it contains less than 10 microns in diameter, and probably the greatest harm is produced by those between 1 and 3 microns.

Concentration of dust in the atmosphere.—The relationship of dust concentration and duration of exposure are closely associated in their etiological significance. The rate at which silicosis will develop, excluding certain factors considered as predisposing, depends upon the dosage of free silica. This dosage is obviously dependent upon the amount of silica in the air inhaled and the duration of exposure.

In 1902, a committee, of which Dr. J. S. Haldane was a member, reported (*11*) upon an investigation made to determine the cause of excessive mortality rates from tuberculosis among the Cornish tin miners. This group decided that it was evident that the inhalation of stone dust by these men in the performance of their tasks as miners was the cause of permanent damage produced upon the lungs. Furthermore, they noted that the condition developed gradually in the case of the ordinary miner, but rapidly in the case of the machine workers who were exposed to greater amounts of dust. Dr. Watkins-Pitchford (*12*) and Dr. Mavrogordato (*13*) and other South African workers, in their discussions relating to the etiology of silicosis, emphasize the relationship of the concentration of dust and duration of exposure to the degree of lung changes produced. Mavrogordato has suggested the advisability of intermittent employment as an aid in lessening the amount of changes produced; but aside from the fact that it does delay the reaction, it has not been shown that it could safely be relied on to prevent silicosis where exposure is to sufficient concentrations. Workers in this country have also shown the relationship of dosage to the severity of the reaction. Reports of the Picher Clinic (*14*), of the Tri-State District of Oklahoma, Kansas, and Missouri lead and zinc mines, which is a cooperative undertaking, by agents of the operators, the Metropolitan Life Insurance Co., the Bureau of Mines, and the Public Health Service have shown clearly the effects of various periods of exposure to silica as met with in the mining processes carried out in the district. Similar studies upon the health of granite workers (*15*) (*16*) likewise stress the duration of exposure necessary to produce definite degrees of silicosis.

Role played by other dusts present with silica.—Most authorities agree that the presence of other inorganic dusts in the silica-containing atmosphere may tend to influence the rate of progressive reaction resulting from inhaled silica. Some of them tend to alter somewhat the radiographic appearance so that the nodular shadows are less discrete.

Some have thought that the relative absence of silicosis in the cement industry was due to the calcium present. However, investigations (*17*) have led to the belief that the absence of evidence of extensive pulmonary fibrosis among employees in the cement industry is due to the fact that there is insufficient total exposure to free silica (considering the percent of free silica in the dust, concentration of dust, and duration of exposure)

No complete reports of pulmonary fibrosis resulting from the inhalation of organic dusts have appeared in the literature. Always when such fibrosis is associated with the breathing of air containing organic dust, either inorganic dust has been shown to be present or at least its absence has not been definitely proved.

In regard to the presence of other irritating dusts, Chapman (*18*), Kessler (*19*), MacDonald (*20*), Kilgore (*21*), and others have reported cases of so-called acute silicosis resulting from the inhalation of air containing high concentrations of silica along with strong alkali in a fine powdered form. Some have suggested that the rapid action of the silica in the production of pulmonary fibrosis is aided by the presence of these alkalis, because silica is more soluble in alkaline solutions. Kettle (*22*) failed to demonstrate such actions experimentally and states that proof is still lacking that any action of the kind may occur. Pathological reports furnish evidence of a definite fibrosis; but the entire picture differs remarkably from that of the typical silicotic, such as has been furnished by Gardner (*23*) in his reports upon the examination of tissues from clinical cases of silicosis as well as his pathological reports made from tissues obtained in his experimental studies.

Sayers, Meriwether, and Lanza (*14*) reported that of the employees in the lead and zinc mines, those who gave a history of previous coal-mining experience developed a definite silicosis in a shorter time than employees of the same age without this experience. This cannot be taken to mean, however, that the inhalation of coal dust hastened the action of silica, for it is doubtful whether any of these coal miners had worked where they were not exposed to some free silica, so that the total dosage of silica was not the same in the two groups.

OCCUPATIONAL HISTORY

An important factor in determining etiological significance of total dust exposure is a complete, carefully taken occupational history.

TABLE 3.—*Occupational record*

Dated: August 3, 1933. Office: Industrial Hygiene.
Name: Kosma Michael. Present age: 49.
Age began work: 15. Number of years worked: 34.

Specific occupation	Specific industry	Number of years in—	
		Hard coal	Non-dusty
Present: Section foreman	Anthracite coal	5	
Preceding present:			
1. Contract miner (chamber)	do	15	
2. Miners' laborer (chamber)	do	3	
3. Mule driver (dry mine)	do	2	
4. Patcher (dry mine)	do	2	
5. Slate picker (dry breaker)	do	2	
6. Farm laborer (Pa.)	Agriculture		1

Remarks: Estimates total time lifa during working life—3 years. By recording the complete data, the total dust exposure may be determined and summarized [illegible]

Table 3a shows the activities and associated dust exposures of chamber miners and laborers. It will be seen that each activity engaged in by those coal miners was studied separately. Experience has shown that the various occupations comprising the process of any dusty occupation are usually associated with dissimilar dust exposure.

TABLE 3a.—*Dust exposure of contract miners and helpers (laborers—Chamber mining*

Activity	Number of samples	Number of hours in activity	Average dust count, millions of particles per cubic foot of air	Millions of particle-hours per cubic foot
Jack-hammer drilling	23	1	575	575
Hand loading	22	2	1,138	2,276
After firing	7	½	834	208
Taping and wiring	2	½	80	20
Setting up props, and in main airways	8	2½	15	41
Total	62	6½		3,121

3,121 million particle-hours per cubic foot equals 480 million particles per cubic foot 6½ hours.

It is apparent from this analysis that the hand loading of coal contributes about 73 percent of the total dust exposure of chamber miners and laborers, although this occupation comprises less than one-third of the total working hours. Jack-hammer drilling, which occupied but 1 hour of the 6½-hour miners' day, amounts to 18 percent of the total exposure, while the practice of entering a room too soon after blasting unduly exposes miners to a large amount of dust, this quantity being about 7 percent of the total dust exposure. Table 4 shows the method of analysis used to determine an estimate of the individual's total dust exposure.

TABLE 4.—*Estimation of the individual's total dust exposure*

Occupation	Number of years	Dust concentration in millions of particles per cubic foot (average)	Millions of particle-years per cubic foot
Slate pickers (dry mine)	2	380	760
Patchers (dry mine)	2	76	152
Mule drivers (dry mine)	3	71	213
Miner's laborer (chamber)	3	480	1,440
Contract miner (chamber)	15	480	7,200
Section foreman	5	7	35
Total	30		9,700

$\frac{9,700 \text{ millions of particle years per cubic ft.}}{30} = 325$ millions of particle years per cubic foot.

More detailed application of our knowledge of the true cause of silicosis is illustrated in table 5. In this study of the actual duties required by the job, it is revealed that the greatest hazard, so far as [illegible] operations are concerned, is that in connection with the use

of a parting compound. It is generally agreed that the most important silica exposure of molders is due to other silica-generating processes often conducted in the vicinity of molding operations. It is obvious that the risk associated with molding operations can be materially lessened either by using a parting compound containing less silica or in properly safeguarding the user by adequate dust control during this operation. With such occupational histories available, and the knowledge of the percentage of total dust exposure revealed by detailed occupational analysis, it was found possible in a recent study to prophesy in 9 cases out of 10 approximately the conditions that would be found upon physical examination in cases where the total changes were due chiefly to dust inhalation.

TABLE 5.—*Dust exposure of molders*

Activity	Time of exposure in minutes (a)	Average dust exposure in millions of particles per cubic foot (b)	Millions of particle-minutes (a×b)
Use of parting compound	54	63.8	3,445
Remaining task in molding	412	4.4	1,813
Pouring	59	3.1	183
Dumping molds (shake out)	15	22.5	338
Total	540		6,059

$\frac{6,059 \text{ million particle-minutes}}{540} = 11$ million particles per cubic foot.

PREDISPOSING CAUSES

Geographical distribution.—Eight countries were represented at the International Conference held in Johannesburg in 1930. A recent bibliography on pneumoconiosis lists references to the literature of 26 countries.

Since the literature of practically all the principal nations of the world contains articles on this subject, it is apparent that no nationality is exempt, and that all races are susceptible is shown by the wide distribution of silicosis.

Age.—Although the data show incidence is higher among the younger miners in districts where the percentage of free silica is high, and among older miners where the percentage of silica is low, age in itself probably is no great factor.

Previous exposure.—Previous occupations of the men are reported to have a definite influence in predisposing to silicosis, if they have been exposed to dust or to other respiratory irritants.

Individual susceptibility.—The factor of individual susceptibility is often mentioned. Generally speaking, if there be any difference in individual susceptibility, it can usually be considered an acquired and not a congenital condition. Ickert (*24*) quotes Bohme and Lucanus and Schulte-Tigge as stating that it is essential that the individual possess excellent functioning nasal passageways, in order that the self-cleansing mechanism may work efficiently. He calls attention to the fact that Irvine, Simpson, and Strachan report the "classical" type of silicosis to be more common among the robust type of individuals with less respiratory reserve. Ickert found some slight variation in the susceptibility of persons according to their type of body build. As a whole, the group classed as slender individuals developed simple silicosis somewhat slower than the stoutly built persons; but the incidence of advanced silicosis was greater in the former class, being nearly double that developing in the sturdy workers.

Lehmann's (*25*) experiments to determine the functional efficiency of the upper respiratory tract in the removal of dust, suggests that abnormalities of the nasal passageways probably play some part in the rapidity with which silicosis may develop.

It is suggested by some that chronic bronchial asthma may be considered a predisposing factor affecting individual susceptibility. The spasmodic attacks, if frequent, lead to a reduction in the individual's vital capacity. Aside from the pulmonary fibrosis, other pathological manifestations of silicosis such as bronchiectasis, emphysema, and right heart hypertrophy and dilatation, may be aggravated by this chronic condition.

Role of infection.—Since respiratory infection has been shown to be the greatest complicating factor in the development of silicosis, certainly the history of present and past respiratory infections will have to be given consideration in the statistical analysis of records upon which such conclusions are based.

Infections developing along the respiratory tract may be of importance. Sinus infections may act by decreasing the efficiency of the upper respiratory tract in the removal of dust from the air passing to the lungs, and also they may be the source of infections that spread to the lungs, and also they may be the source of infections that spread to the lower respiratory tract. Acute pneumonic conditions as well as the more chronic lung changes such as chronic bronchitis, bronchiectasis and bronchiolectasis, emphysema, and pleurisy, all tend to decrease the ability of the lung to rid itself of foreign materials, through lessened lymphatic drainage and decreased power to force the bronchial secretions and foreign matter from the lungs.

Clinical findings and diagnosis.—The subjective and objective signs of silicosis vary according to the rate of development and degree of pulmonary fibrosis and, when infection is present, according to the type, extent, and duration of the complicating pulmonary infection.

Early in the course of the disease, signs of the condition may be absent or slight, except for the changes revealed by X-ray examination. When sufficiently developed, the most common subjective signs are shortness of breath upon exertion, cough, and

depending upon degree of development, the chief objective signs are shortness of breath, cough, prolonged expiration, decreased chest expansion, altered breath sounds and rales, impaired resonance, and characteristic radiographic appearance of the lung fields.

Diagnosis is based upon historical data, including the complete occupational history, past and present medical history, clinical, laboratory, and X-ray findings. Except by autopsy, silicosis cannot be diagnosed definitely until examination of the X-ray film reveals characteristic nodular shadows throughout both lung fields. A complete diagnosis should include, in addition to the decision that silicosis is present, a statement as to whether or not there is evidence of a complicating pulmonary infection; the type of infection if possible, and its extent; and, finally, a statement as to whether or not the individual manifests any decreased capacity for doing his usual work and an estimate as to the degree of his disability. Individuals with silicosis and active pulmonary tuberculosis are considered seriously disabled, and for the safety of themselves and others they should be barred permanently from work affording further exposure to a silica-bearing atmosphere, regardless of the manner in which the tuberculous infection may respond to treatment and rest.

As indicative of the affected individual's status under compensation laws, it has been customary to define the disease arbitrarily into stages—first, second, and third (*1*). From a medical point of view, there is no such clear line of demarcation.

First stage.—Chest film indicates early nodular fibrosis, the individual may or may not exhibit such clinical evidence as slight shortness of breath upon exertion, and some cough. The general appearance is that of a healthy individual, and there is no appreciable decrease in capacity to perform usual duties.

Second stage.—More advanced nodulation is shown by X-ray, and there may be some evidence of localized aggregation of nodules and pleural thickening. Definite shortness of breath upon exertion, cough more pronounced, chest movements sluggish, and expansion usually decreased. The individual may be able to continue at his usual job, although less effectively.

Third stage.—Fibrous changes as shown by X-ray film are further accentuated. Nodules are larger and assume conglomerate form, showing large shadows corresponding to areas of dense fibrosis. Shortness of breath is marked and distressing upon slight exertion. Cough is increased, and may be dry, but is usually productive. Chest expansion is decreased even upon forced inspiration. The individual's capacity for work is seriously and permanently impaired.

The stages just discussed would seem to indicate in a way the extent of disability or potential disability. However, those interested in silicosis are tending toward the opinion that it is best to make a

decision as to whether the individual has silicosis or not, and to determine the extent of disability, if any, due to the disease, without reference to stage.

TUBERCULOSIS AND SILICOSIS

In 1905 the Miners' Phthisis Committee of Australia, reported that gold miners there who contracted silicosis died of tuberculosis (*26*).

The increased incidence of tuberculosis among occupational groups exposed to silica has been clearly shown in every instance where this hazard exists. Britten (*27*) summarized the report of the Registrar-General of England and Wales for 1921–23, and showed the occupational mortality rate for the group of trades classed as "Dusty Trades" to be from 3 to more than 10 times as high as the rate for all occupied and retired males.

TABLE 6.—*Standardized mortality from respiratory tuberculosis in occupations with rates above average, males age 20–65, England and Wales, 1921–23*

Occupation	Mortality rate (standardized)
All occupied and retired males	149.6
Tin and copper mines, underground workers not superintending staff (III)	1,880.0
Tin and copper mines, not superintending staff (III)	1,322.5
Grinders in the cutlery trade (IV)	1,172.5
Metal grinders (IV)	636.7
Slate masons and slate workers (III)	512.5
Potters' mill workers, slip makers, potters (III)	441.4

Lanza and Vane (*5*) show by an analysis of the mortality experience of 12 life-insurance companies, for the period of 1915 to 1926, that the actual mortality from respiratory tuberculosis among the silica-exposed persons was about three times that of a non-silica-exposed group. When this comparison is limited to the rates for some of the occupations with a very great silica exposure, such as metal mining, sandstone, and granite quarries, the excess is still more striking, the rate being about 10 times that obtained in the non-silica-exposed group.

Gardner (*28*) has shown that animals exposed to silica when inoculated with a strain of tubercle bacilli of low virulence will develop systemic tuberculosis and die, while control animals not so exposed usually are not seriously affected by injections of such organisms.

PROGRESSIVE TENDENCY OF THE DISEASE

Since infection has been shown to play an important role in the advanced seriousness of silicosis, the possible relationship of this infective element to the progressive tendency of the disease cannot be overlooked.

Irvine (*29*) has stated that it is not so much what the condition of

emphasizes the tendency of the fibrosis to progress, even though removed from exposure, and expresses his opinion that it is one of the most serious aspects of the whole silicosis problem. No remedy has been shown to be of value in the elimination of the pulmonary fibrosis although improvement in symptoms may be noted after the victim is removed from exposure. According to the observations made by Bohme (Bochum) (*30*), silicosis progressed, after removal from exposure, in 20 percent of the cases diagnosed as having silicosis in grade 1, in 40 percent of the cases in grade 2, and in practically all cases in grade 3.

EFFECTS OF OTHER DUSTS

It seems to be the consensus of opinion that silica dust is the most harmful, and it is the principal one with which industry is concerned. However, asbestos is reported to be the one which produces a condition which is disabling and causes death.

Asbestosis is relatively a new disease in industrial workers, as asbestos has not been in general use for so many years. Asbestos is an anhydrous form of magnesium silicate. The symptoms of asbestosis are insidious in onset and irregular in the course of the disease. Dyspnea and cough are the most prominent, and as the disease progresses the cough becomes productive. Elastic fibers can be found in the sputum in advanced cases, indicating that there is destruction of the pulmonary tissue. Asbestos bodies have been found in the sputum and the finding of them is a diagnostic aid, showing that the worker has been exposed to the dust. These particles of asbestos in the sputum usually show evidence of disintegration. Asbestosis usually develops after exposure to the dust (excessive quantities) for a period of approximately 7 to 11 years (*31*). It has not been shown that asbestosis predisposes to tuberculosis as does silicosis. Death usually results from pneumonia, bronchitis, or influenza and less often from tuberculosis. When tuberculosis complicates asbestosis, the clinical picture is not the fulminating type.

The X-ray characteristics of the chests of asbestos workers differ from those of workers exposed to silica dust. The markings are less distinct and are described by some as having a "ground-glass appearance." The nodular fibrosis of silicosis is conspicuously absent.

Another form of pneumoconiosis occurs incident to exposure to magnesium silicate dust in the form of talc. This dust is a hydrous form of magnesium silicate, $H_2Mg_3(SiO_3)_4$, and is used in a number of industrial processes. The mining and milling of talc entails exposure to excessive amounts of the dust. Dreessen (*32*) reports the results of a study of exposure to talc dust. He states that a fine bilateral fibrosis of the lungs occurs, which is demonstrable by the X-ray; and that while very dusty conditions prevail in the production of talc, it cannot be said that the resultant pneumoconiosis has led to disability

ANTHRACO-SILICOSIS

Anthraco-silicosis is the term used to describe a certain form of pneumoconiosis, commonly called miners' asthma. It is a chronic condition due to breathing dust incident to coal mining. It is characterized by general fibrotic changes of the lungs with the presence of excessive amounts of carbonaceous and siliceous materials. A compensatory emphysema occurs and often cardiac changes take place in the later stages.

The coal mining employees were grouped occupationally, largely in accordance with the proportion of free silica found in the dust to which they were exposed.

No cases of anthraco-silicosis were found in a control group composed of hard-coal mining employees whose dust exposure averaged less than 5 million particles per cubic foot of air.

The prevalence of anthraco-silicosis among the entire group of employees was found to be about 23 percent. Among men exposed 15 to 24 years to dust containing less than 5 percent free silica, 14 percent of those who had worked where the average dust count was 100 million to 199 million particles per cubic foot, 29 percent of those exposed to 200 million to 299 million particles, and 58 percent of the men who had worked for this period in more than 300 million particles per cubic foot, developed anthraco-silicosis.

Among the men employed for more than 25 years in dust containing less than 5 percent free silica, the proportion of persons found with anthraco-silicosis under different concentrations of dust was as follows: 5 million to 99 million particles, 7 percent; 100 million to 199 million particles, 54 percent; 200 million to 299 million particles, 71 percent; 300 million or more particles per cubic foot, 89 percent.

With the exception of miners, their helpers, and rock workers, about 25 percent of all the men employed underground developed anthraco-silicosis after a working period of more than 25 years. This group was exposed to dust having a quartz content of about 13 percent.

The prevalence of anthraco-silicosis among persons who had been exposed for more than 2 or 3 years to dust of which about 35 percent was free silica, varied from 10 percent among those who had worked in concentrations of less than 200 million particles per cubic foot for less than 15 years, to 92 percent among those who had been employed for more than 25 years in dust concentrations exceeding 300 million particles per cubic foot, more than 2 or 3 years of which time was spent in rock work.

Age *per se* appeared to play only a minor role in the development of anthraco-silicosis.

Analysis of the data for the purpose of determining safe limits of dust exposure indicated that

taining 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 5 percent. In the gangways where the silica content of the dust was about 13 percent, a safe limit appeared to be 10 million to 15 million particles per cubic foot. The limit of toleration for rock workers was set tentatively at 5 million to 10 million particles per cubic foot of air.

Pulmonary infection increased with length of service more rapidly among the men in the haulageways than in the control group, and much more rapidly among the regular miners. The highest rates of pulmonary infection, however, were found among the rock workers of more than 15 years' service.

The prevalence of pulmonary tuberculosis among the hard-coal mining employees at ages below 35 was slightly less than that found through studies of tuberculosis among male adults in the general population of the country. In the age group 35 to 44, however, the prevalence of tuberculosis was about twice, at ages 45 to 54 about 5 times, and for the ages above 55 it was about 10 times the rate found in the general population.

The highest prevalence of clinical pulmonary tuberculosis occurred among the rock workers (men who had been employed in rock loading or rock extraction for more than 2 or 3 years). After 20 years' service, of which more than 2 or 3 years were in rock work, 37 percent presented evidence of pulmonary tuberculosis.

In a group of 135 completely disabled former anthracite workers, which did not include any known cases of tuberculosis, 10 percent proved positive for pulmonary tuberculosis.

Pulmonary infection (tuberculous and nontuberculous) was found among 58 percent of the employed men having early anthraco-silicosis, and in 92 percent of the workers in the more advanced stages.

Clinical pulmonary tuberculosis was diagnosed in 15 percent of those with early anthraco-silicosis, and in 43 percent of those in the more advanced stages.

In all groups combined, with the exception of the control group, about 20 percent of the nontuberculous workers were diagnosed as having some respiratory disease other than tuberculosis. In the control group only 6 percent had nontuberculous respiratory disease.

In the control group less than 2 percent were found with moderate or marked physical impairment causing decreased capacity for work as compared with about 10 percent among the regular miners, and with approximately 13 percent among the rock workers. With the exception of the rock workers, no group showed moderate or marked physical impairment in excess of that found among the controls when the period of employment was less than 20 years. However, an excess in the prevalence of slight impairment was found among the regular

miners and among others exposed to dust containing less than 5 percent free silica when they had worked from 10 to 20 years in atmospheres containing more than 100 million particles per cubic foot.

The correlations between exposure to dust and the evidence of constitutional changes left little doubt as to the etiological significance of the dust in the air breathed. Like correlations were found between the silica exposure and the extent of pulmonary changes.

Mortality from respiratory diseases was found to be much greater among anthracite workers than in the general adult male population of the country. The data indicated that underground work in the absence of dust did not predispose to fatal attacks of respiratory disease.

PREVENTION OF SILICOSIS

This phase of the silicosis problem may advantageously be considered under two main divisions—medical and mechanical.

Medical.—Preliminary and periodic physical and roentgenological examination should be made of all employees engaged in industrial processes where there is a potential if not actual exposure to excessive concentrations of silica in the atmosphere. Medical personnel making these examinations should be familiar with the industrial processes in which the individual is to be employed.

In fairness to the individual, his fellow workers, industry, and even the public at large, an individual with evidence of active tuberculosis or extensive healed lesions should not be employed in an atmosphere where there may be exposure to silica dust. The infected individual's condition may become aggravated by such exposure to silica dust; his fellow workers may become infected under conditions which might not be dangerous were it not for the presence of silica dust; industry may eventually bear the burden of compensation for injury to the health of the employee and possibly some of his coworkers; and last, but not least important, the prevention and control of tuberculosis among the general population will be more difficult. In the absence of infection, silicosis is a slowly progressing disease and develops at such a rate that the individual affected may not become disabled during his ordinary working life. Therefore, if infection can be lessened, the silicosis problem may be much more readily controlled.

Periodic physical examination will serve to detect early evidence of infection and abnormal pulmonary changes resulting from dust exposure, which will permit correction of the conditions causing the pulmonary fibrosis and prevent the development of disabling pulmonary disease. Unless active pulmonary infection is diagnosed, the individual in whom a beginning fibrosis or nondisabling silicosis is present may be safely continued at his employment, provided the dusty conditions causing it are eliminated.

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STUDIES ON TRICHINOSIS

VII. The Past and Present Status of Trichinosis in the United States, and the Indicated Control Measures [1]

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INTRODUCTION

Some factors regarding trichinosis that have been clarified since the problem was taken up by the Public Health Service in the spring of 1936 are as follows:

1. Abundant evidence indicates quite clearly that trichinosis is probably a greater problem in the United States than in any other country in the world. The evidence to this effect was available about 50 years ago, but was quite generally overlooked, disregarded, or misinterpreted.

* Died May 1, 1938.

2. Human trichinosis rests primarily on a basis of swine trichinosis.

3. Swine trichinosis rests primarily on a basis of uncooked or inadequately cooked pork scraps fed to swine in garbage, table scraps, swill, and similar material.

4. The indicated control of trichinosis in the United States is primarily a matter of keeping such uncooked pork scraps out of the feed of swine, and either cooking such feed as garbage or refraining from feeding any food containing pork scraps.

5. Rats appear to have a very minor role in the production of porcine trichinosis, and at this time may be given only secondary consideration in our control measures.

6. The evidence available indicates that our too casual measures for the control of trichinosis have not lowered the incidence of trichinae or trichinosis in man in this country during the past 50 years. The incidence in swine has decreased only to the extent that the once widespread practice of feeding offal from slaughtered hogs to other hogs has decreased; and this bad practice has been almost entirely abolished purely as a result of improved sanitary conditions in general and not as a definite part of any trichinosis control program.

7. Prompt action looking towards the control of trichinosis is imperative for the protection of the public health on the one hand, and the protection and improvement of the swine industry and the packing industry on the other hand. An extension of the present move by scientists, physicians, and veterinarians to protect the public health must be anticipated by the swine growers and packers to protect their industries from the consequences which may be expected from research within the next few years.

The factors just enumerated will be discussed in the following sections.

PREVALENCE OF TRICHINOSIS

There was abundant evidence 50 years ago that the United States had an extraordinary prevalence of trichinosis, but that evidence was misread, misunderstood, disregarded, or misinterpreted for the most part, and wholly unwarranted conclusions were drawn from it. Various writers, including Mark (*1*), pointed out, in the 1880's, that

[1] Following are the preceding articles of this series:
I. The incidence of trichinosis as indicated by post-mortem examinations of 300 diaphragms. By Maurice C. Hall and B. J. Collins. Pub. Health Rep., 52: 488 (1937). (Reprint 1836.)
II. Some correlations and implications in connection with the incidence of trichinae found in 300 diaphragms. By Maurice C. Hall and B. J. Collins. Pub. Health Rep., 52: 512 (1937). (Reprint 1817.)
III. The complex clinical picture of trichinosis, and the diagnosis of the disease. By Maurice C. Hall. Pub. Health Rep., 52: 529 (1937). (Reprint 1819.)
IV. The role of the garbage-fed hog in the production of human trichinosis. By Maurice C. Hall. Pub. Health Rep., 53: 873 (1937). (Reprint 1898.)
V. The incidence of trichinosis as indicated by post-mortem examinations of 1,000 diaphragms. By M. C. Nolan and John Bozicevich. Pub. Health Rep., 53: 692 (1938). (Reprint 1929.)
VI. Epidemiological aspects of trichinosis in the United States as indicated by an examination of 1 [illegible]