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INDUSTRIAL HYGIENE AND TOXICOLOGY

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VOLUME I

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PREFACE

Industrial hygiene has been recognized and practiced from the time of Pliny down through the ages. It is the present concept of industrial hygiene that is relatively new—the concept of anticipating and recognising potentially harmful situations and applying engineering control measures before serious injury results. There are some who question industry's ability to control all harmful exposures; and there is quite naturally a tendency to take the easy way out of a difficulty by substituting materials of low or moderate toxicity for those of a hazardous nature. Nevertheless, where incentives such as low cost, availability, or superior properties of a hazardous material justify the provision of positive engineering controls, they can be supplied promptly.

It is said that artisans are born rather than made, but there can be no doubt that industrial hygienists must be made, and it takes years to mellow some of us with sufficient understanding so that we can use our knowledge of the basic principles of industrial hygiene to the best advantage in accomplishing our goal.

Depending somewhat upon where we acquire our academic training and initial experience in field work, we are likely to start out with the concept that industry has one purpose—to make money—and that, in following that urge, the humanitarian aspects are apt to be neglected. So it is with somewhat of a shock that some of us learn that many industries are eagerly taking the initiative in improving the working environment of their employees. Our first ideas of introducing hygiene to industry are likely to involve some means of maneuvering into a position in which our recommendations for control measures are to be accepted as commandments not to be questioned and not, upon penalty of closing up shop, to be ignored. It is only gradually that we become aware of the fact that in promoting anything to the American public a sound idea "takes" more quickly and develops faster if it is "sold" rather than presented as an ultimatum. As a people we basically resent being told bluntly that we have to do a thing: we much prefer to "discover" for ourselves that the proposed new course is correct and therefore to our advantage. Adams* expresses these ideas well in his recent book, from which I quote:

> "The American is a composite of almost all races, nationalities, and classes . . . He hates a bit and bridle as heartily as does a young colt . . . There *are* dirty politicians, dirty labor leaders, dirty business men—black markets, some selfish and dirty consumers—but the Americans, now 138,000,000 of them, *the American people*, are the hope of the world and of the whole future of humanity."

The industrial hygienist becomes aware that salesmanship is a necessary part of his practice. The salesman will think of the "buyer's" point of view and, first of

* J. T. Adams, *Big Business in Democracy*. Scribner's, New York, 1946.

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all, develop his recommendations for environmental controls with the understanding of an economist, and then stress all the advantages. He will simplify his work by taking advantage of the views of production engineers and occasionally going them one better by saving them money in the conservation of materials or in the recovery of a by-product. One cardinal rule he learns early is not to "bluff" or try to impress his audience with his superior knowledge. Some few persons have the ability to "get away with it," but the odds are against them and it is much safer to work on the same plane as our audience whether that means a step down or a jump up, and it helps to imagine one's self in the position of the man one is trying to influence.

Industrial hygiene may be defined as *the science and art of preserving health through the recognition, evaluation, and control of environmental causes and sources of illness in industry.* It resolves itself into the problem of finding factors or conditions in workplaces that may cause or contribute to the illness or serious discomfort of employees, and of devising methods and means of eliminating or controlling such conditions.

It would be a mistake to attempt to give the impression that industrial hygiene is pure science, or that it is restricted to the art of applying scientific principles: much of it involves a liberal use of common sense or what is perhaps better known as "horse sense." The job will never become monotonous or routine because the chemist, the engineer, and the physicist will keep introducing new and more or less hazardous materials and processes that require new developments for the evaluation and control of exposures attendant to their use. Neither is the job glamorous or spectacular, and much of it is hard work bordering on drudgery, but it has its compensations.

> I should like to narrate an incident that, because of its fundamentality and at the same time dramatic departure from the daily routine of an industrial hygienist, may be worth describing without retouching. One of the pioneers in industrial medicine and hygiene whom most readers will recognise without further identification might have regarded this incident as an "acorn.*" However, since no tangible reward or token of appreciation was either anticipated or received and the only special remuneration was the feeling of satisfaction that goes with accomplishing any job, assumed or assigned, it was considered all in the day's work.
>
> While busily engaged at my office one morning in the industrial hygienist's favorite occupation of pouring over survey reports, I received a telephone call: "This is Dr. ————, chemist over at ———— Dry Dock. Here's something I think you should know about and maybe you will want to come over and look around. We just sent a man to the hospital and we have two more who are laid up and in a serious condition. The ———— was docked here just two weeks ago for repairs and conversion and out of the 2,000 men working on her, over 100 are affected with some sort of breaking out and itch and all the men are threatening to quit if we don't find out what's wrong and correct it. The boat's been in the tropics for some time and the workmen fear some tropical disease is responsible for this outbreak."
>
> Yes, I did want to look around, and within an hour was aboard the ship and observed many of the afflicted men at work. There was considerable grumbling and an abundance of dirty looks. Several men wore bandages over vesicular patches and on a few there was evidence of a generalized fine vesiculation. We went through the ship from stem to stern and forecastle to bilge. It was like a beehive: men were cutting with torches, sawing out panels,
>
> * C. P. McCord, *A Blind Hog's Acorns*. Cloud, Inc., Chicago, 1945.

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knocking off plaster, shoveling out debris and filth, scrubbing, and removing the interior furnishings preparatory to refitting the boat completely. Admittedly the ship was dirty—in fact, in some areas it was filthy—but so what! Next we went to the first-aid room to see the attendant and find out what, if any, information could be obtained there. The place was crowded with patients, and while we waited to see the attendant first-aid man we could hear the grumblings of the waiting patients, who complained of "filthy working conditions" and that, if the health department were not called in to condemn the place, everyone should quit before they became ill—that the place was "infested with fleas" and that they didn't "want any tropical fevers." Finally the harassed attendant came to us, but he had little to offer except to comment that the cases and complaints were getting more numerous and that he certainly hoped we could do something.

We had seen the situation: a once luxuriant ship, somewhat filthy in spots, an explosive outbreak of dermatitis in nearly ten per cent of the 2,000 men at work on the ship, no reported cases among the many thousands of workmen in adjacent areas of the same shipyard, and, unless something were done quickly, work on this desperately needed troop ship and possibly in the entire yard would stop.

The job looked interesting if not easy. We collected samples of everything we could get loose—plaster, upholstery, hair stuffing, sweepings, scrapings from panels and floors, and samples of the different woods and sawdusts, and returned to the laboratory. The samples were turned over to the chemists and microscopists to look for fumigants, insecticides, alkalies, and other common irritants, as well as any signs of insects or parasites. Two members of the staff were inveigled into joining me in making patch tests with some of the materials after saturating them with alcohol as a precautionary measure. While these tests were in progress a medical associate told me with an air of finality that our company had called in, as consultant, an authority on dermatology and that he planned to leave the matter entirely in the consultant's hands. When I took the story to my immediate superior he said it was very interesting, and sometime when he had more time I should "tell him all about it." I telephoned the consultant dermatologist and inquired if he had seen the cases and had any ideas of what might cause the difficulty. Yes, he had seen them, and, except for the fact that it didn't make sense, he would say that they resembled poison-ivy cases. That agreed with what we had seen on the job, but we had not seen any poison ivy! After all of the chemical and microscopic tests proved negative except for an insignificant amount of arsenic in the sweepings, there remained only the possibility that the patch tests might indicate an irritant. After 48 hours, and with all patch tests negative, the shipyard's chemist reported that there were several more cases, including an electrician working on the dock beside the boat, and that a walkout seemed imminent even though the ship was desperately needed for a troop transport.

The fact that an electrician who possibly had not been on the boat was affected gave an indication that the problem might be attacked from epidemiological approach by personally interviewing some of the afflicted men. Discussion of personal matters with employees, especially shipyard workers, is ordinarily something to be avoided, but in this instance approval of such interviews was readily obtained from all parties concerned. Aided by the chemist and two safety engineers, we went out on the job and talked to some of the affected men, including the electrician, who had never been on the suspected ship, and a security policeman, who was seriously affected and had been aboard the ship only once for a few minutes. This introduced a strong element of doubt about the ship's being the contact source and diverted our attention to other possible sources of irritant material. It developed that nearly all cases were on the day shift. The electrician's case had been diagnosed "cable rash," but, since the characteristics of his lesions were similar to those of the other workmen, we did not waste time examining the cables with which he had been working. Instead, we investigated all the possible exposure sources where the men spent their time when they were off the ship: where

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they ate their lunch, where they loafed, and the route they took to and from work. We found what appeared to be some damaged oil drums in a pile on a sand lot where several men had spent their lunch hour within the shipyard grounds, but about a block away from the dock to which the boat was tied. The drums were punctured or otherwise damaged and, for the most part, were empty, but three or four contained some dark oily liquid. Close examination of these drums revealed lettering still visible on one which read *Cashew Shell Liquid!*

The cause of the epidemic had been found.

At the shipyard, management quickly announced the facts over a public address system to the workmen so that their fears would be allayed. A strike was averted, and the ship was completed ahead of schedule. Further investigation revealed that cashew shell liquid had been spilled on the dock to some extent, in the street to a considerable amount, and extensively scattered over the sand lot in question. Many of the men working at refinishing and repairing this ship had sat on the sand, or the drums themselves, during the noon hour while they ate their lunches. Others had stretched out in the sun, bare-backed, on the contaminated sand. A check into the source of the drums revealed that a cargo of 1000 drums of the liquid had arrived and been unloaded on the opposite side of this same dock about one month previously. This work had been done by an outside contractor who had dumped the damaged drums onto the unused lot.

At our suggestion, all suspicious-looking spots were covered with chlorinated lime, the dock was cleaned and scrubbed, the drums disposed of, and dirt filled in on the sand lot. The dermatitis, which by now was starting to appear in the homes of workmen, from contact with soiled clothing, quickly disappeared. My colleague, the shipyard's chemist, however, in a sincere effort to convince himself and others of the potency of the cashew liquid, became impatient at negative results obtained in 24 hours with one tiny patch test and tried four more generous patches with samples from different drums. A few days later he joined those who had been hospitalized.

This is not a medical book nor is it intended for legal reference. Its primary purpose is not to aid in the diagnosis and treatment of disease, the winning of compensation, or the refutation of false claims. If it should prove of use in such instances by making some facts more widely available, it may be presumed that the more light that can be thrown upon these phases of industrial medicine and industrial relations the better. It is hoped that much information of use in preventive medicine is included. Diagnostic signs of absorption or of early effects, in advance of injury, are eagerly sought by the progressive industrial physician, and, although time-proved and recognized tests are disappointingly few, the research-minded industrial physician will find opportunities for increasing this field of knowledge. The object of this book is to present industrial hygiene and toxicology in simple, understandable terms in sufficient detail to be of some use to all persons interested in safeguarding the health and welfare of working people and in improving the working environment. Essential requisites for the completely successful advancement of the health and safety of the breadwinning population include: (*1*) competent persons in health and safety maintenance departments; (*2*) managerial interest and appreciation of the benefits to be derived from health and safety work; and (*3*) teamwork—camaraderie, and cooperative efforts among industrial hygiene, medical, and safety personnel.

The welfare of an individual workman involves not only a working environment that gives reasonable assurance of freedom from accidental injury or occupational

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disease, but also his mental, temperamental, and physical fitness for the work to be done. Incentive, whether in the form of tangible assets to supply the necessities and some of the pleasures of life, or some expression of interest or recognition of accomplishments, ability, and efforts may be involved indirectly, but are somewhat outside the field of industrial hygiene.

It is not possible to maintain a uniform style in a collaboration, but the collective viewpoints compensate for some variations in style. In Chapter Thirty-Five considerable variation will be noted in the length of discussions. Much of this follows a premeditated plan to give full discussion where most needed and to avoid duplication. Some, however, reflects the degree of personal familiarity of the authors of this part of the book with the occupation under discussion. Where information is desired on an industry not listed, significant processes of the industry may be found elsewhere. Many cross references of this nature have been included. The book is liberally supplied with references for the benefit of those who wish to pursue the subjects in greater detail.

Acknowledgments

The contributors are to be congratulated for their doggedness in sticking to the task through many adversities, and, in some instances, inordinate demands upon their time. To the early contributors, may I express my appreciation of your indulgence during many discouraging delays. I should like also to express appreciation to Mrs. Ruth B. Patty for valuable assistance in editing, checking, and reading proof; to Mrs. Kathleen Kumler for reading proof and assistance in preparing the index. Credit should be extended to Lucille Rokicki and Suzanne Wickett, who have done much of the secretarial work. The courtesy of various individuals, companies, societies, and publishing houses in permitting reproduction of some of the tables and illustrations is gratefully acknowledged.

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Detroit, Michigan
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Some support for this opinion is seen in the report of foundry experience in recent years.[4] Only 150 sandblasters were engaged in steel foundries prior to the war, yet 31 sandblasters were given certificates covering death, total disablement, or suspension on account of silicosis in the 1932–42 period. Congestion of operations, owing to great demands on capacity, has been credited with increasing the number of silicosis cases by increasing the concentration of dust-producing operations per unit of shop area. A committee appointed to study the situation recommended: (*1*) no silica should be used in parting compounds; (*2*) no sand should be used as an abrasive in "sandblasting"; and (*3*) no person under 18 should work as a sandblaster, in repairing a sandblast plant, or within 20 feet of such a plant. Consideration was given to prohibiting the use of free silica in molding material as well as silica paint on cores. Certain of these regulations appear unnecessarily drastic and do not take into account the possibilities of engineering control.

E. R. A. Merewether, Senior Inspector of Factories in England, recognized this lack of engineering interest and skill when in 1942 he told an American Public Health Association audience that the time had arrived when "the doctor no longer knows everything about everything" and conceded American leadership in industrial hygiene engineering. Merewether recently has attempted to inspire the English chemists to instigate the formation of a society for "the holding of annual conferences and the publication and distribution of papers and literature on the subject of industrial health and safety,"[5] much as is now done by our own American Industrial Hygiene Association and to a limited extent by the American Public Health Association.

B. RUSSIA

C.-E. A. Winslow, who visited Russia in 1917 and again in 1936, reports major advances in public health in that country. Emphasis has been placed on the health of industrial workers. Scientific studies of many occupational hazards and of means of promoting industrial health have been conducted by various research organizations, of which may be mentioned the Institute of Industrial Diseases in Moscow, the Central Institute for Nutrition in Moscow, Pavlov Institute, the Leningrad Institute of Safety, Hygiene and Technique, and States' Scientific Institute of Labor Protection.

The Soviet State has given special attention to questions involving better labor conditions, looking toward the highest possible efficiency and production, as well as the mental and physiological well-being of the workers. Early permissible standards for air pollution in factories tended to be ultraconservative on the side of safety, and rather severe standards for industry to meet. Various motives have been suggested as having contributed to the zeal of industrial hygiene investigators. Recently, however, there have been indications that standards are being revised upward to conform with experience and that in general the requirements have been

[4] "Dust in Steel Foundries," *Engineering (London)*, *158*, 152 (1944).
[5] *Chem. Age*, *53*, 353 (1945).

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arc; and (*f*) desirable air movement past the worker's head. The use of so many estimating procedures shows that requirements can not be standardized. The best approach is to figure the ventilation rates by several methods to establish a reasonable agreement in the selection of air-flow constants.

A convenient rule of thumb for checking the amount of ventilation provided in welding booths or rooms is based on the rate of electrode burning. For coated steel rods, the ventilation rate to keep the fume concentration down to 10 or 15 mg. per cubic meter of air may be estimated by the formula:

$$\text{c.f.m.} = \frac{100{,}000\ (\text{in. diam. of electrode})^2}{\text{minutes per electrode}}$$

If welding is continuous by several persons simultaneously, the *minutes per electrode* in this formula include the idle time both during welding and during the exchange of electrodes. If welding is intermittent by one person, the *minutes per electrode* should be only the elapsed time to burn one full electrode; the ventilating fan may be turned off if welding is idle for extended periods. (See also section on dilution ventilation.)

3. Abrasive-Blasting Rooms

These are complete enclosures for sand blasting, shot blasting, grit blasting, hydroblasting, etc. The concentrations of dust in these rooms are enormous, and special supplied-air helmets must be provided for the workers. Preferably, complete protective suits are used because of the destructive effect of flying abrasive. Ventilation requirements are estimated either in (*a*) air changes per minute, (*b*) c.f.m. per square foot of floor area, or (*c*) c.f.m. per blasting nozzle in operation. Method (*c*) is used when the ventilation rates of similar blasting operations conducted in rooms of greatly different volumes are being compared. It is also a more rational method of relating ventilation to the amount of dust produced when the number of blasting operators in a given room varies. The ranges of ventilation rates used with these three methods of specification are: (*a*) 5–15 air changes per *minute;* (*b*) 50–150 c.f.m. per square foot of floor; and (*c*) 5,000–15,000 c.f.m. per nozzle in use.

4. Metalizing or Metal-Spraying Rooms

These may be similar to abrasive-blasting rooms, and the required personal protective equipment likewise similar, but without the necessity of protection against high-velocity abrasive particles. Spraying of metallic lead within a room results in a tremendous potential hazard, and the best possible supplied-air respirator is necessary even though the ventilation may be excellent.[19] Spraying of steel also creates a high concentration of particulate matter, but it is possible to design ventilating equipment that can prevent dangerously high air-borne concentrations; even so, the nuisance may not be tolerable, and some form of filter respirator might be required.

[19] H. I. Miller, Jr., G. M. Hama, E. C. J. Urban, and P. Drinker, *J. Ind. Hyg. Toxicol.*, *20*, 380 (May, 1938).

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TABLE 15

Minimum Exhaust Ventilation for Grinding, Buffing, and Polishing Wheels[a]

Maximum wheel diam., inches[a]	Minimum air volumes, c.f.m.	
	Good wheel enclosure[b]	Poor wheel enclosure[c]
1 to 4	200	300
5 to 9	300	450
10 to 14	400	600
15 to 19	550	800
20 to 24	700	1000
25 to 29	900	1400
30 to 36	1200	1800

[a] Largest wheel than can be used within the exhaust hood.
[b] Not more than 25 per cent of wheel periphery and wheel sides exposed.
[c] Buffing and polishing wheels generally require air quantities listed under "Poor wheel enclosure."

Kane[11] has compiled a useful list of air flows representing good practice for control of certain dusty processes; Table 16 is adapted from his original tabulation.

TABLE 16

Volumes of Air Exhaust for Dust-Producing Equipment

Dust-producing equipment	Exhaust hood type	Air-flow requirements
Abrasive-blast rooms (sand, grit, or shot).	Tight enclosure with air inlets (usually in roof).	60–100 f.p.m. downdraft (long rooms of tunnel proportions 100 f.p.m. cross-draft). Exhaust volume should be sufficient to provide visibility for operator. Sand as abrasive, or castings with cores, or heavy molding sand deposits require highest range.
Abrasive-blast cabinets	Tight enclosure with access openings.	20 air changes per minute but not less than 500 f.p.m. through all openings.
Bagging machines.	Booth or enclosure (provide spillage hopper).	Paper bags, 100 c.f.m. per square foot open area. Cloth bags, 200 c.f.m. per square foot open area.
Barrels (for filling, or removing material).	Local hood 120 deg. around top of barrel.	Through 24-in. diam.—4-in. branch. Over 24-in. diam.—5-in. branch, at 3500 f.p.m.
Belt conveyors.	Hoods at transfer point.	Belt speeds less than 200 f.p.m.—350 c.f.m. per foot of belt width, but not less than 150 f.p.m. through open area. Belt speeds over 200 f.p.m.—500 c.f.m. per foot of belt width but not less than 200 f.p.m. through open area.
Belt wipers (may be required with high-speed belts).	Tight-fitting hood held against underside of belt.	200 c.f.m. per foot of belt width. Requires high hood suction and high conveying velocities. Mechanical brushing or wiping often used in conjunction with hood.
Bins (closed bin top).	Connect to bin top away from feed point.	150–200 f.p.m. through open area at feed points, but not less than 0.5 c.f.m. per cubic foot of bin capacity.

[10] Tentative Standard, Bur. Ind. Hyg., Detroit Dept. Health.
[11] J. M. Kane, *Heating & Ventilating*, **42**, 68 (Nov., 1945).

Common External Marks of Occupation or Occupational Diseases or Other Diseases (*continued*)

Mark or sign	Probable cause	Significance	Comment
		Skin, *continued*	
Pathological sweating.	Many nonindustrial causes, but sometimes seen in lead poisoning.	Unusual sweating may be localized, odoriferous, colored, excessive. Sometimes found in heart disease, rickets, syphilis, palsy, tuberculosis, goiter, etc.	In arsenic or lead poisoning, abnormal sweating usually local to parts involved in neuritis.
Brick red (warm weather). Plum colored (cold weather).	Polycythemia (pathologic increase in numbers of red blood cells).	May be a manifestation of repeated exposure to carbon monoxide in relatively low concentrations. R. M. I.	Engorgement and congestion of superficial blood vessels. Most prominent on face. May involve conjunctivae.
Paroxysmal face and head flushing.	Chiefly associated with the menopause, this type chiefly limited to women 40 to 50.	May be associated with mental depression, excessive perspiration, weakness, insomnia, emotional instability.	Not occupational.
Lymphangitis.	Infection.	Red streak up arm or leg. Alarming indication of extension of localized pyogenic infection. R. M. I.	Frequently associated with "kernels" such as in axilla or groin.
Keratosis (farmer's warts).	Excessive sunlight, ultraviolet light, etc.	Represent warty, flat, or horny, persistent overgrowth of outer layers of skin. Characteristic of past middle age life; usually limited to exposed parts of skin; characteristic of many trades including farmers, sailors, fishermen, gardeners, trappers.	Rarely become cancerous; readily removed by electrocauterization, by x-ray and by electric needle; less well by keratin solvents. Quite common both in and out of industry.
Asbestos warts.	Mechanical penetration by asbestos fibers; possibly chemical action akin to production of silicotic nodules.	Condition usually limited to fingers and hands.	Similar condition with glass fiber workers, but in that case always mechanical.
Tumors (on skin).	Several types, both benign and malignant. Commonest variety, epithelioma.	Apart from nongrowing warts and obvious abscesses of skin, any enlarging mass on or in skin should be investigated. R. M. I.	Pigmented moles are prone to become malignant.
Tumors (beneath skin).	Frequently harmless, fatty tumors (lipomas). Attain to large size.	Because of uncertainty, every mass beneath the skin should be investigated by a physician.	Skin over lipomas, if squeezed, becomes dimpled, "orange peel" sign. See next item.

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rate, whereas it is directly proportional to the particulate size, density of the dust suspended in air, and the extent to which the dust is wetted while passing through water. He found that retention was not affected by volume per respiration, vital capacity, or relative humidity of inspired air.

V. Dust Causing Extensive Pulmonary Fibrosis (Silicosis and Asbestosis)

A. HISTORY

Since ancient times it has been thought that dust caused disease. Perhaps the earliest reference to disease caused by dust and fume is that of Plinius,[16] who described the devices used by refiners to prevent the breathing of "fatal dusts."

In 1556, in *De re metallica*, Agricola[17] stated:

"On the other hand, some mines are so dry that they are entirely devoid of water, and this dryness causes the workman even greater harm for the dust which is stirred and beaten up by digging penetrates into the windpipe and lungs and produces difficulty in breathing."

The best of the early writings on silicosis is that of Ramazzini,[18] translated in 1705 in *A Treatise of the Diseases of Tradesmen*, in which he stated:

"For in hewing marble or stones out of the rock, in polishing and cutting them, they oftentimes suck in by inspiration the sharp, rough and cornered small splinters or particles that fly off; so that they are usually troubled with a cough, and some of them turn asthmatick and consumptive . . . And in dissecting the corps of such artificers, the lungs have been found stuffed with little stones. Diemerbrock gives a curious relation of several stone cutters that dy'd asthmatick, and were opened by him; in whose lungs he found such heaps of sand that in running the knife through the pulmonary vesicles he thought he was cutting some sandy body. He adds that he was informed by a master stone cutter that in cutting stones there rises such a subtile dust, as is able to penetrate through ox bladders hung in the shop . . . And this very dust he took to be the cause of the death of many unwary workmen."

In recent years the contributions to knowledge of silicosis have been sufficiently substantial, numerous, and varied to make this perhaps the most widely discussed occupational disease. Silicosis is of particular interest to students of preventive medicine not only because of its general distribution in industry but more particularly because of its tendency to increase susceptibility to tuberculosis—a characteristic inherent in the disease. The limited space of this chapter does not permit reference to many sound contributions which have led to our present understanding of dust disease. An exceedingly interesting and somewhat detailed review of the history of diseases caused by dust may be found in the work of Lanza.[19]

B. EXPOSURE TO SILICA IN INDUSTRY

Since siliceous material makes up the bulk of the earth's crust, it is not surprising to find silica exposures in industry, where raw materials from the earth are used

[16] Caius Plinius Secundus, *Naturalis historia*, Bk. II. Trans. by K. C. Bailey under the title, *The Elder Pliny's Chapter on Chemical Subjects*, Pt. 1. Longmans, Green, New York. 1929.

[17] Georgius Agricola, *De re metallica*, Bk. I. Trans. from 1st Latin ed. of 1556 by H. C. Hoover and L. H. Hoover. Mining & Sci. Press, San Francisco, 1912.

[18] B. Ramazzini, *A Treatise of the Diseases of Tradesmen*, English trans., 1705.

[19] A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

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to make many of the commodities necessary to our daily lives. Rocks and minerals are often intimately associated with free silica and it is obvious that those occupations concerned with mining, rock grinding, or drilling may constitute silicosis hazards, as well as occupations concerned with the processing and industrial use of siliceous products.

According to Knopf[20] 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,[21] illustrates the great variety of uses to which silica is put in industry and indicates the kind of silica adapted to each purpose.

TABLE 1

Industrial Uses of Silica and Types of Silica Used[21]

Uses	Types
Abrasives	
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, buhrstones, 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.
Refractories	
In making silica fire brick and other refractories.	Fairly pure quartzite known as gannister; not less than 97 per cent SiO_2 nor more than 0.40 per cent alkalies, tightly interlocking grains desired.
Metallurgy	
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.

Table continued

[20] A. Knopf, *U.S. Pub. Health Bull.* No. *187* (1929).

[21] R. B. Ladoo, *Silica, Nonmetallic Minerals.* McGraw-Hill, New York, 1925, p. 525.

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TABLE 1 (*continued*)

Uses	Types
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. In road asphalt surfacing mixtures.	Finely ground silica of all types.
Ceramics	
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.	Massive and ground diatomaceous earth.
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.

C. ETIOLOGY OF SILICOSIS

Silicosis has been defined by the Committee on Pneumoconiosis of the Industrial Hygiene Section of the American Public Health Association[22] as:

"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."

The definition adopted at the International Silicosis Conference in 1930 and reaffirmed at the 1939 conference is generally accepted and is somewhat simpler than the above. It is as follows:

[22] R. R. Sayers, *Yearbook, Am. Pub. Health Assoc.* (1932–33).

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"Silicosis is a pathological condition of the lungs due to inhalation of silicon dioxide. It can be produced experimentally in animals."

Lanza[23] in "Etiology of Silicosis," as printed in the National Silicosis Conference Report on Medical Control, states:

"Simple or uncomplicated silicosis is a chronic, fibrotic disease of the lungs, due to the massive invasion of the pulmonary tissue by silica (SiO_2), inhaled in the form of dust. In its early stages, silicosis may be symptom free; in its later stages shortness of breath, decreased chest expansion, lessened capacity for work, may—one or more—be present, together with an increased susceptibility to tuberculosis. The disease presents the characteristic x-ray appearance of nodulation, without which a clinical diagnosis may not be made."

The production of silicosis depends upon the following factors: the composition of inhaled dust, the number of particles of inhaled dust, the size of the particles of inhaled dust, the length of time during which particles are inhaled, and individual susceptibility.

1. Composition of Dust in Relation to Production of Fibrosis

The fact that silicosis is caused by free silica (SiO_2) is attested by a great mass of clinical experience as well as by experimental observations. Gye and Kettle[24] have shown that silica in solution, or in noncrystalline form, stimulates the proliferation of fibroblasts in tissues. Gardner[25] produced a reaction in the lungs of animals in a period as short as two years by exposure to extremely high concentrations of dust. Miller and Sayers[26] developed an intraperitoneal injection technique for testing the tissue response to dust, and their method has been used extensively by the United States Public Health Service. On the basis of this procedure it has been found that all dusts behave in one of three ways in the body tissues; they may disappear (be absorbed), cause cellular proliferation, or remain inert in the tissues.

Dust Causing an Absorptive Reaction. No cases of pneumoconiosis have been reported and confirmed among workers exposed solely to dust of the absorptive group. In experimental animals this dust disappears, leaving little or no scarring. A list of different varieties of dust investigated, which are absorbed, follows. These are of industrial origin unless otherwise stated.

Calcite—essentially pure calcium carbonate.

Precipitated calcium carbonate—containing 10.1 per cent magnesium carbonate, 0.1 per cent magnesium oxide, 0.6 per cent iron and aluminum oxides and 0.4 per cent total silica.

Gypsum—the uncalcined, natural mineral composed essentially of calcium sulfate with 1.3 per cent total silica.

Limestone—not less than 82 per cent calcium carbonate, less than 12 per cent total silica, and not more than 10 per cent free silica.

Portland cement—containing 74.4 per cent calcium oxide and 21.1 per cent total silica. Free silica not reported.

Pyrolusite—composed of 54.9 per cent manganese with no quartz reported.

[23] A. J. Lanza, Report on Medical Control, National Silicosis Conference, *U.S. Dept. Labor, Div. Labor Standards, Bull.* No. 21, Pt. 1 (1938).

[24] W. E. Gye and E. H. Kettle, *Brit. J. Exp. Path.*, 3, 241 (1922).

[25] L. U. Gardner, in A. J. Lanza, *Silicosis and Asbestosis.* Oxford Univ. Press, New York, 1938.

[26] J. W. Miller and R. R. Sayers, *U.S. Pub. Health Repts.*, 58, 264 (1941), Reprint 2234.

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Dust Causing a Proliferative Reaction. Each type of dust examined that falls into the proliferative group is known to produce nodular, pulmonary fibrosis and each is a form of free silica. In experimental animals such dust produces nodules, which progressively increase in size until a maximum is reached, in about 90 days. At first the nodule is similar to that produced by dust which is absorbed. Later the fibroblasts are replaced by macrophages, which become filled with dust particles. Miller and Sayers give the following examples of this group:

Bisque ware—ground semivitreous pottery bisque ware, fired at a relatively low temperature. Total silica, 72.0 per cent; quartz, about 40 to 50 per cent; the remainder semifused clay and feldspar.

Chert—total silica, 76.1 per cent; quartz about 25 per cent; other forms of free silica, 35 per cent.

Diatomite—total silica, 92.5 per cent; aluminum oxide, 3.5 per cent; ferric oxide, 1.5 per cent; calcium oxide, 0.4 per cent; magnesium oxide, 0.7 per cent; essentially pure diatomite.

Greenware—ground vitreous unfired pottery ware.

Porcelain enamel frit—total combined silica, 35 to 50 per cent; the remainder is oxides of antimony, zinc, and aluminum, and fluorides of sodium, aluminum, and calcium.

Quartz—a pure mineral dust; normal crystalline quartz of high purity (SiO_2).

Quartz—identical with the above sample but treated with 0.6 per cent crude pine fatty acids.

Quartz-sericite—the source of this dust is unknown. Chemical analysis: total silica, 81.04 per cent; calcium oxide, 0.30 per cent; magnesium oxide, 0.45 per cent; sodium oxide, 0.10 per cent; potassium oxide, 0.98 per cent; iron oxide, 0.25 per cent; aluminum oxide, 14.25 per cent; total water, 2.61 per cent. Petrographic examination: quartz, about 50 per cent; muscovite (variety, sericite), about 45 per cent; fibrous sericite, less than 5 per cent.

Bisqueware, chert, greenware—containing from 69 to 76.1 per cent total silica and 25 to 50 per cent of this as quartz.

Diatomite—essentially pure and containing 92.5 per cent total silica.

Quartz—samples of pure mineral dust and industrial dust containing over 99 per cent SiO_2.

Quartz-sericite—about 50 per cent quartz and 50 per cent varieties of sericite.

Tripoli—total silica, 98.9 per cent; chalcedonic silica (crystalline aggregates) with an occasional crystal of normal quartz.

Dust Causing an Inert Reaction. Pneumoconiosis resulting from some of the forms of dust that are inert in the peritoneum has been reported. Clinical and pathological examples of the reactions show modified nodular fibrosis if the dust contains appreciable quantities of free silica; with other types of inert dust, diffuse interstitial fibrosis may sometimes result. The nodules produced in experimental animals become gradually flattened, and the dust is dispersed into the adjacent connective tissue, often to a considerable distance. Inert dust must be considered as a possible potential cause of pulmonary fibrosis. It is much less likely to cause pneumoconiosis than is dust that produces cellular proliferations in the peritoneum. Dust samples of the following descriptions were shown by Miller and Sayers to be inert:

Aluminum—pure aluminum bronzing powder of the finest grade.

Alundum—total silica, 4.6 per cent; aluminum oxide, 88.4 per cent; ferric oxide, 6.9 per cent.

Asbestos—total silica 37.5 to 50.86 per cent.

Anthracite coal—total silica, 6.6 to 8.6 per cent.

Bentonite—clay, variety montmorillonite, about 97 per cent; feldspar, about 2 per cent; quartz, none observed.

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Bisque ware—ground vitreous pottery bisque ware, fired at a relatively high temperature. Quartz, about 30 to 40 per cent; the particles were wholly or partially covered by the glass phase. (This is absent in semivitreous bisque ware.)

Bituminous coal—total silica, 0.8 to 3.5 per cent.

Calcium phosphate—petrographic examination: earthy phosphates (not apatite), about 97 per cent; normal and chalcedonic quartz, about 3 per cent.

Chromite—total silica, 7.8 per cent; quartz, less than 5 per cent.

Diamond dust—pure bortz diamond dust used as abrasive.

Feldspar—total silica, 65.9 per cent; calcium oxide, 0.81 per cent; magnesium oxide, 0.10 per cent; aluminum oxide, 19.55 per cent; iron oxide, 0.28 per cent; potassium oxide, 8.98 per cent; sodium oxide, 3.18 per cent. Petrographic examination: feldspar (plagioclase-microcline), about 95 per cent; normal quartz, about 5 per cent.

Fuller's earth—filtral clay, containing from 55.7 to 62.1 per cent total silica and 1 to 10 per cent quartz.

Glass wool—finely ground sample of commercial hard glass wool.

Hematite (jewelers' rouge)—total silica, 1.5 per cent; iron oxide, 98.3 per cent.

Kaolin—china clay and hydromica predominant; quartz and feldspar, a trace.

Lanthanum sublimate—from the burning of white flame electrodes. Lanthanum, 40.0 per cent. Petrographic examination: particles too small to identify.

Mica—silica, 46.92 per cent; magnesium oxide, 0.86 per cent; aluminum oxide, 34.95 per cent; ferric oxide, 2.65 per cent; potassium oxide, 9.54 per cent; sodium oxide, 1.02 per cent; manganese dioxide, trace. Petrographic examination: mica, both as plates and fibers, plates predominating, about 98 per cent; a very small amount of quartz and feldspar.

Precipitator ash—composed of siliceous material, magnesium, iron, aluminum and calcium oxides, or rare earth oxides of the cerium group. The total silica content ranges from 0 to 48.3 per cent and the quartz from 0 to 5 per cent.

Pyrophyllite—predominantly pyrophyllite, with a small amount of rutile and a small undetermined quantity of quartz.

Rock wool—a finely ground sample of commercial, insulating rock wool.

Selenium—selenium, 98.8 per cent; tellurium, 0.01 per cent; ash, 1.16 per cent.

Selenium—a chemically prepared sample of highest purity.

Sericite—a pure mineral dust. Total silica, 51.74 per cent; calcium oxide, 0.61 per cent; magnesium oxide, 1.74 per cent; sodium oxide, 3.40 per cent; potassium oxide, 4.48 per cent; iron oxide, 5.83 per cent; combined oxides, 31.82 per cent; total water, 6.26 per cent. Petrographic examination: sericite and feldspar residues (fibrous sericite predominates), about 95 per cent; quartz, less than 5 per cent.

Shale—silica, 61.0 per cent; aluminum oxide, 12.4 per cent; calcium oxide, 4.5 per cent; ferric oxide, 5.0 per cent; magnesium oxide, 1.3 per cent; sodium oxide, 2.3 per cent; potassium oxide, 1.5 per cent; moisture, 10.3 per cent. Petrographic examination: about 35 per cent quartz; the majority of the particles appeared to be coated with clay.

Silicon carbide—pure manufactured silicon carbide. Silicon, 67.5 per cent. Petrographic examination showed no impurities.

Soapstone—total silica, 36.8 to 49.9 per cent; calcium oxide, 1.7 to 5.0 per cent; magnesium oxide, 22.7 to 26.2 per cent. Petrographic examination: talc, about 55 to 65 per cent; dolomite, about 5 to 30 per cent; tremolite, about 15 to 30 per cent. No quartz observed.

Talc—total silica, 49 to 56.54 per cent; calcium oxide, 6.25 to 8.8 per cent; magnesium oxide, 22.6 to 30.74 per cent; calcium silicate, 0 to 11.00 per cent; calcium carbonate, 0 to 1.88 per cent; iron and aluminum oxides, 0 to 1.04 per cent; ignition loss, up to 4.60 per cent. Petrographic examination: talc, mostly fibrous, about 40 to 75 per cent; tremolite, about 25 to 60 per cent; calcite and (or) dolomite, up to 1 per cent.

Titanium oxide—a finely divided, high-purity sample.

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Sodium silicate—a laboratory-prepared sample containing 1 part sodium oxide to 3.1 parts silica. Higher ratios of sodium oxide killed the animals.

Trap rock—silica, 51.7 per cent; aluminum oxide, 16.0 per cent; ferric oxide, 2.0 per cent; ferrous oxide, 9.9 per cent; calcium oxide, 10.0 per cent; magnesium oxide, 6.2 per cent. Petrographic examination: feldspar, some slightly decomposed, about 45 per cent; pyroxene, about 45 per cent; magnetite, about 10 per cent; biotite, about 1 per cent.

Volcanic ash—silica, 54.4 per cent; aluminum oxide, 14.5 per cent; ferric oxide, 3.8 per cent; magnesium oxide, 2.6 per cent; calcium oxide, 0.7 per cent; ash, 78.2 per cent. Petrographic examination: fine volcanic ash partially altered to montmorillonite. No quarts observed.

Volcanic ash—a specially treated sample. Silica, 74.3 per cent; mixed oxides, 16.8 per cent; ferric oxide, 2.2 per cent; calcium oxide, 0.5 per cent; magnesium oxide, 2.2 per cent. Petrographic examination: glass only. No quarts or calcite observed.

The immediate response of body tissue to any dust is essentially a foreign-body reaction, but the subsequent behavior of the tissue to any given dust determines whether such a dust is harmful.

In the peritoneal injection experiments of Miller and Sayers only dust containing free silica caused fibrous proliferations.

Although it has been well established that silicon dioxide is the cause of silicosis, there has been little satisfactory explanation for its action. Earlier it was thought that the fibrous nature of the proliferation was related to the hardness or sharpness of the particles. However, Gardner[27] found that no fibrosis was produced by experimental inhalation of carborundum dust, which was even harder and sharper than silica. Thus, the action is clearly chemical, but the exact chemicophysiological reaction that takes place is still unknown.

The mixed reaction sometimes produced by dusts of the inert group in which silica is mixed with other minerals emphasizes the possible effect of mixed dusts on the development of silicosis. The probable importance of concomitant exposures has also been indicated by clinical experience. Chapman,[28] MacDonald and his associates,[29] and Kilgore[30] have reported cases of rapidly developing silicosis caused by breathing air with high concentrations of silica and alkali dust. Kettle[31] and McCord[32] failed to demonstrate such action experimentally. McCord, on the basis of extensive investigations of workers in six plants where there was exposure to silica and alkali dusts, found no evidence of an accelerator action by alkalies. In fact, the absence of silicosis in this group suggested an inhibitor action which he believes may have resulted from the marked increase in solubility of silica in the presence of alkali. Peritoneal injection in animals yielded no results to prove either an accelerator or inhibitor action of alkali in the formation of silica nodules; but alkali did cause silica to spread from the point of injection and increased its primary toxicity.

The absence of silicosis in ganister-brick manufacturing was thought at first due to an inhibiting effect of accompanying dusts; more recently it has been found

[27] L. U. Gardner, *Am. Rev. Tuberc.*, *20*, 883 (1929).
[28] A. M. Chapman, *J. Am. Med. Assoc.*, *98*, 9 (1939).
[29] G. MacDonald, A. P. Piggot, and F. W. Gilder, *Lancet*, *2*, 836 (1930).
[30] E. S. Kilgore, *J. Am. Med. Assoc.*, *99*, 1414 (1932).
[31] E. H. Kettle, *Proc. Inst. Mining Metallurgy (London) 43rd Sess.*, 1934.
[32] C. P. McCord, *Ind. Med.*, *5*, 17 (1936).

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that the concentration of free silica in the work atmosphere was too low to cause fibrosis. Silicosis as found in coal miners (anthracosis) and in foundry men may be quite different in x-ray appearance and in its course from that observed in quartz grinders.

Gardner[23] investigated these effects experimentally using ferruginous chert and mixtures of quartz and calcined gypsum. He found that the percentage of free silica in air-borne dust may not be the same as in the parent substance. This fact may explain the slowness in development of silicosis in workers in certain areas and in certain types of work. When a mixture of calcined gypsum and quartz was present in air, particles of both minerals flocculated and the rate of settling of the mixture was greater than that of either dust in the pure state. Injection experiments with ferruginous chert showed that the iron in the mixture temporarily inhibited the action of silica. Gardner's summaries of these experiments follow:

1. Artificial mixture of equal parts of calcined gypsum and quartz. Materials ground separately to respirable sizes and mixed in a dusting hopper.

Quartz content of parent mixture..... 49.7%
Quartz content, air-floated dust in cages..... 29.6%
Average light-field count, air inside cages, 336 million particles per cu. ft.

No exposed animals developed silicosis within 25 months although only 15 months were required to produce nodulation with average concentration of 120 million particles pure quartz per cu. ft. of air. Of 17 animals exposed more than 25 months and up to 30 months, 7 showed nodular fibrosis of modified type. Remaining 10 developed only chronic pneumonitis.

2. Artificial mixture of 2 parts of calcined gypsum and 1 part of quartz prepared as above.

Quartz content of parent mixture..... 31.4%
Quartz content, air-floated dust in cages..... 17.1%
Quartz content, lung ash of exposed animals..... 15.4%
Average light-field count, air inside cages, 245 million particles per cubic foot of air.

No animal developed silicosis until exposures had been continued for 24 months. Mature nodules found in only 3 of 16 guinea pigs exposed 24–29 months. These lesions were of modified type. The other 13 showed nonnodular pneumonitis.

3. Ferruginous Chert I, a natural mixture ground at a mine to such size that a majority of the particles were less than 10 microns in diameter, but there were a considerable number of larger ones.

Free SiO_2 of parent mixture..... 54.6%
Free SiO_2 of settled rafter dust..... 38.4%
Free SiO_2 of air-floated dust in cages..... 10.4%
Free SiO_2 of lung ash of exposed animals..... 3.3%
Average light-field count, air in cages, 776 million particles per cubic foot.

No suggestion of silicotic reaction but merely pigmentation of lungs of animals exposed for maximal period, 18 months.

4. Ferruginous Chert II. A similar natural mixture from the same source as that used in exp. 3 but ground until all particles were 5 microns and less in diameter.

Free SiO_2 of parent material..... 67.5%
Free SiO_2 of settled rafter dust..... 61.0%
Free SiO_2 of air-floated dust in cages..... 33.5%
Free SiO_2 of lung ash of exposed animals..... 16.3%

[23] L. U. Gardner, "Reactions to Mixed Dusts," in *Fourth Saranac Laboratory Symposium on Silicosis*, B. E. Kuechle, ed., Employers Mutual Liability Insurance, Wausau, Wis., 1939.

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Average light-field count, air in cages, 776 million particles per cubic foot.

No silicosis in any animal's lungs exposed under 3 years; in 8 of 15 animals exposed for longer periods, modified nodules were present. The other 7 developed only pigmentation and chronic pneumonitis.

Thus we have both laboratory and clinical demonstration of the influence exerted by other dust on the development of silicosis.

2. Number of Particles Inhaled in Relation to Production of Fibrosis

From the findings of Gardner, quoted above, and from the discussion of flocculation and settling, in the preliminary section, it is apparent that the type of dust may play an important part in determining the number of particles in the workroom atmosphere. Not only may some components of admixtures flocculate and thus change the character of a dust, but silica itself, being a hard material with a tendency to form large particles as compared with those formed by softer minerals, may settle out more quickly than other components of a mixed dust.

However, at this point we are somewhat more concerned with the concentrations of dust that are harmful than with the factors that have produced any given concentration in the air. Determinations of free and total silica in the lungs of patients who have died after fibrosis developed and in those without fibrosis have been made by incinerating the lungs and chemically analyzing the ash for its silica content. Although there have been some variations in published data on the amount of silica in pulmonary tissue necessary to produce silicosis, Sladden,[34] McNally,[35] Badham and Taylor,[36] and Fowweather[37] found fairly comparable amounts. Drinker and Hatch[38] sum up the present knowledge somewhat as follows: as a rough guide, a total silica content as high as 0.2 per cent of dried lung can be considered normal. A content over 1 per cent is definite evidence of dust exposure, and this amount is usually accompanied by fibrosis. There is too much overlapping of data and variation in technique to be sure from published data of the significance of quantities between 0.2 and 1 per cent of ashed lung. This fact is of especial significance, as pointed out by Cummings.[39] Since there is usually no evidence of silicotic reaction in lungs containing less than 1.5 to 2 gms. of silica (1 per cent of weight of dried lung), it may be inferred that the contraction of silicosis does not necessarily follow the inhalation of silica. Silicosis occurs only after the inhalation of amounts in excess of a minimum.

In any control program we are of course concerned primarily with the amount

[34] A. F. Sladden, *Lancet*, *2*, 123 (1933).
[35] W. D. McNally, *J. Am. Med. Assoc.*, *101*, 584 (1933).
[36] C. Badham and H. B. Taylor, *Med. J. Australia*, *I*, 511 (1933).
[37] F. S. Fowweather, *Chem. Industries*, *53*, 713 (1934).
[38] P. Drinker and T. Hatch, *Industrial Dust—Hygienic Significance, Measurement and Control*. McGraw-Hill, New York, 1936.
[39] D. E. Cummings, "The Etiology of Silicosis," in *Fourth Saranac Laboratory Symposium on Silicosis*, B. E. Kuechle, ed., Employers Mutual Liability Insurance, Wausau, Wis., 1939.

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of dust in the air that will cause fibrosis. The methods of determining atmospheric dust concentrations are discussed in Chapter Eight. Cummings suggested that atmospheric concentrations of silica dust should be considered in terms of two thresholds: the primary threshold, a level at which a healthy man can be employed for his lifetime without harm, about 5,000,000 particles per cubic foot (light-field count); and the secondary threshold, a level at which a healthy man will inevitably develop silicosis, about 100,000,000 particles per cubic foot (light-field count). The National Silicosis Conference summarizes the situation as follows: "There is evidence that for prolonged exposure a concentration of more than 5 million particles per cubic foot, of a highly siliceous dust, is dangerous. Therefore it is now considered good practice to hold concentrations of highly siliceous dust at 5 million particles per cubic foot, or less," as based on light-field counting methods.

Since standards of safe atmospheric dust concentration based on medical findings have been suggested tentatively for only a few industrial dusts, and since considerable study is necessary to form a basis for such standards for other industrial dusts, a tentative arbitrary measure of what is good practice may be used. This should be within the limits of good engineering practice and yet low enough to control the silicosis hazard for most industrial exposures. The following formula is frequently used to express the maximum permissible concentration of silica in air:

> Multiply the percentage of free silica by the total dust particle count per cubic foot (light-field technique). If the result is over 5 million, the concentration may be considered too high. For example: a dust containing 10 per cent free silica with an average total concentration of 30 million particles per cubic foot would give 0.10 times 30 million, which equals 3 million (good practice); a dust containing 30 per cent with an average total concentration of 50 million particles per cubic foot would equal 0.3 times 50, or 15 million (unsatisfactory). This formula is not applicable to any dust containing less than 5 per cent free silica.

The Division of Industrial Hygiene, National Institute of Health,[40] has suggested that an attempt should be made to have highly siliceous dusts kept at a concentration below 4,000,000 particles per cubic foot since considerable silicosis occurred in the pottery industry even with low dust concentrations.

Dust may also be simply a nuisance. It is considered good practice to control even relatively harmless dust sufficiently to prevent concentrations in excess of 50,000,000 particles per cubic foot (by light-field count) in the workroom air.

3. Particle Size in Relation to Development of Fibrosis

We have already mentioned the fact that large particles settle more rapidly than small ones and that particle size is important as a determining factor in the actual amount of dust in air. Particle size is even more important in determining the chemical activity of the dust and physiological responses to it. Chemical activity is, of course, increased in small particles because of the increased surface area. McCrae[41]

[40] R. H. Flinn, W. C. Dreessen, T. I. Edwards, E. C. Riley, J. J. Bloomfield, and R. R. Sayers, *U.S. Pub. Health Bull.* No. *244* (1939).

[41] J. McCrae, *Pub. S. African Inst. Med. Research*, Report No. 3 (1913).

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found that 70 per cent of the particles in silicotic lungs were less than 1 μ in diameter and that the largest were not greater than 10.5 μ. An upper limit of 10 μ as the maximum size that will produce silicosis has been suggested because larger particles are probably collected by mucus in the upper respiratory tract and moved out of the lungs by ciliary action. The importance of small particles in the production of fibrosis has been well demonstrated by Tebbens, Schulz, and Drinker.[42] These investigators produced liver fibrosis in experimental animals by intravenous injections of suspended silica and found that particles less than 0.6 μ in diameter caused much more fibrosis than larger particles.

Briefly, silicosis is caused by the inhalation of silica particles less than 10 μ in diameter. Recent work suggests that particles of less than 0.6 μ may be of greatest importance in this respect.

4. Individual Predisposition

Detailed discussion of the role of individual predisposition in the etiology of silicosis is hardly necessary. Race and sex seem to play little part, although race may be a factor in the development of tuberculosis just as financial status may be a factor. Certainly it is known that of two persons working in the same dusty exposure one may contract silicosis in a few years while the other may wholly escape it. We have already mentioned the possible influence of differences in nasal filtration as a factor in causation of silicosis. It is likely that there are sound explanations for the many variations in individual susceptibility, but clarification is lacking.

D. PATHOLOGICAL ANATOMY AND X-RAY FINDINGS

Gardner,[43] whose descriptions of pathological anatomy have been used as a basis for much of this discussion, has classified the pneumoconioses as follows: *Nonspecific pneumoconiosis*, including all forms except silicosis and asbestosis, *silicosis* of the classical discrete nodular type, *modified silicosis* caused by the inhalation of dusts containing free silica mixed with certain other minerals, and *silicosis with conglomerate lesions*, in which healed or active infection probably plays a dominant role.

1. Nonspecific Pneumoconiosis

All dust other than that containing free silica and asbestos produces the same general reaction.

X-ray Examination. There is simply an accentuation of the normal, branching, treelike shadows cast chiefly by the pulmonary blood vessels. As an occasional variation in this picture there may be superimposed fine reticulations. These reticulations are thought to be caused by thickening of the sheaths of the pulmonary arteries and thickening of the interlobular septa. See Figures 4 and 5.

[42] B. D. Tebbens, R. Z. Schulz, and P. Drinker, *J. Ind. Hyg. Toxicol.*, **27**, 199 (1945).

[43] L. U. Gardner, in *Fourth Saranac Laboratory Symposium on Silicosis*, B. E. Kuechle, ed., Employers Mutual Liability Insurance, Wausau, Wis., 1939.

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H. CONTROL OF SILICOSIS IN INDUSTRY

1. Engineering Control

The fundamental basis for silicosis prevention is engineering control. Enclosed processes, exhaust ventilation, wet methods, and other engineering techniques can be used to insure safe concentrations of dust in the air breathed by workmen. Engineering control of dusts is discussed in Chapter Ten.

Personal protective equipment such as filter respirators and air-line or supplied-air respirators (See Chapter Fourteen) will under some circumstances afford protection, but such devices are poor substitutes for air sufficiently free of dust to prevent the development of pneumoconioses.

2. Medical Control

All employees with potential exposure to dangersous concentrations of free silica should be given annual medical examinations. These should include x-ray films of good quality competently interpreted, clinical examinations, laboratory examinations in cases where it is necessary to determine infection or concomitant disease such as cardiocirculatory dysfunction, and pulmonary function tests. When there is a question of tuberculous infection, the patient should be examined with sufficient frequency and detail to determine the exact status of the disease.

The type of routine x-ray examination used will be a major factor in determining the cost of the program to the medical department. Most roentgenologists feel that the standard 14-in. × 17-in. film is necessary for definite diagnosis. However, as a screening procedure, several less expensive films are available. Paper film is sometimes substituted for ordinary transparent film. Roentgenograms of this type usually show too little detail to be satisfactory even as a screening process. Roentgenograms of the 35-mm. or 4-in. × 5-in. size, on the other hand, may be used rather successfully for differentiating normal from pathological chests. The 35-mm. film is cheaper and requires less time for developing than the 4-in. × 5-in. It must, however, be used purely as a screening device, as there is not sufficient detail for diagnosis of abnormal findings. Films of the 4-in × 5-in. size frequently may be interpreted directly, necessitating the re-x-raying of fewer employees.

The approximate relative cost of the different sizes of film in the amounts used by large industries is as follows:

Size of film	Cost per patient (approx.)
35-mm. film	4½¢
4-in. × 5-in. film	7 ¢
14-in. × 17-in. film	80 ¢

The number of employees to be examined would alter this but the relative costs should remain fairly constant.

The value of the routine chest survey in the control of silicosis cannot be overstressed, and if photoroentgenograms are used for screening purposes costs may be reduced to a level that can be absorbed by most industries.

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The decision as to whether or not a workman should continue at his work is a serious responsibility and requires great care on the part of the examining physician as well as a thorough understanding of working conditions in the particular occupation and an appreciation of public health and moral responsibility both to the individual and to his associates. As a guide for management of the patient with silicosis or tuberculosis, Lanza[69] has formulated the following rules for the various general groups seen:

"I. Partial disability due to silicosis without infection:

"(a) May continue at usual occupation, if environment is satisfactorily controlled as regards dust concentrations and tuberculosis contact;

"(b) Degree of fibrosis and rate of development may require that the individual seek less arduous employment and avoid even minimal exposures to silica-containing atmosphere.

"II. Partial disability due to silicosis with complicating pulmonary infection:

"(a) Primary silicosis. Infection mild and nontuberculous. In most instances, after successful treatment of nontuberculous complicating infection, may be allowed to return to usual work, provided environment under which they are working is safely controlled.

"(b) Primary silicosis with pulmonary tuberculosis as complicating infection. Those individuals with silicosis who develop pulmonary tuberculosis are considered totally unfit for further employment in an industry affording even minimal exposures to free silica. This disability is obviously total so long as active tuberculosis is present. Following successful treatment, these cases regain their health to the point where they can be safely employed at other work."

3. Aluminum Prophylaxis and Therapy

The use of aluminum for the prevention of silicosis was first reported by Denny, Robson, and Irwin[70] in 1937. At about the same time similar investigations were undertaken by Gardner and his associates. Since that time considerable attention has been focused on aluminum and its compounds both in laboratories and in clinics.

Tabershaw and Tebbens[71] summarized the principles that have evolved from laboratory work on both finely divided metallic aluminum and amorphous hydrated alumina as follows: If alumina and silica localize in the same phagocytic cell the effect of the silica is neutralized. Further progression of the tissue reaction characteristically produced by silica is then arrested. Mature silicotic nodules become static, and immature lesions (inflammatory and early fibrous changes) are resolved. Aluminum may remain in the tissue and protect the individual for a long period—over a year. Aluminum does not ordinarily produce toxic effects in the tissues, although it appears to increase the susceptibility of experimental animals to tuberculosis when large doses are given.

Occasionally, allergy to aluminum has been reported,[72] but this is apparently a rare occurrence. Denny, Robson, and Irwin have preferred to use metallic aluminum, but Gardner has found that certain hydrated aluminas are more satisfactory. McIntyre Research Limited, the United States agency created for licensing the use

[69] A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938, p. 56.

[70] J. J. Denny, W. D. Robson, and D. A. Irwin, *Can. Med. Assoc. J.*, 37, 1 (1937); 40, 213 (1939).

[71] I. R. Tabershaw and B. D. Tebbens, *J. Ind. Med.*, *14*, 709 (1945).

[72] L. H. Cotter, *J. Ind. Hyg. Toxicol.*, *26*, 7 (1944).

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of aluminum by physicians for therapeutic or prophylactic purposes, makes only metallic aluminum available. Some investigators, notably Hannon,[73] have reported marked success in the treatment of patients with severe disability. However, when purely objective criteria have been used to measure disability the results have been somewhat less striking. Gardner, Dworski, and Delahant[74] have suggested that variations in response to aluminum therapy may be due in part to the nature of the dust that has caused the silicosis. They suggest that dust which is primarily free silica may react with aluminum more readily than dust which contains high percentages of iron. The use of aluminum generally in industry for the therapy or prophylaxis of silicosis should be contemplated only with the utmost caution.

I. ASBESTOSIS

Asbestos is a hydrated magnesium silicate. More than 90 per cent of the raw mineral used in this country and Great Britian is produced in the Canadian chrysotile mines. Asbestos is used in two general types of manufacturing processes. It may be used either by itself or mixed with other insulating materials such as diatomaceous earth for fireproofing, packing, or insulating; or it may be combined with cotton and woven as a textile for fireproof and heat-resistant clothing and other substances. Lanza[75] estimates that there are about 10,000 persons exposed to asbestos in the United States. Most observers feel that the incidence of asbestosis in American asbestos workers is quite low. Asbestosis has, however, been reported more frequently in England and Canada.

Lanza, McConnell, and Fehnel[76] concluded from their study of asbestosis that: Prolonged exposure to asbestos dust causes pulmonary fibrosis different from that produced in silicosis and demonstrable by roentgenogram. Clinically it appears to be milder than silicosis.

Definite cardiac enlargement frequently was found to be associated with asbestosis.

A predisposition to tuberculosis, due to asbestos dust, was not indicated although it was not known to what extent asbestosis may add to the mortality from pneumonia and acute nontuberculous pulmonary infections.

Symptoms. The onset of asbestosis, as of silicosis, is slow although symptoms are apt to be somewhat more marked than in silicosis. In silicosis there may be marked x-ray findings with little complaint of symptoms, whereas the opposite is likely to be the case in asbestosis. As in silicosis, dyspnea is the cardinal symptom. Anorexia occurs frequently in advanced stages, and cyanosis and clubbing of the fingers are apt to appear with greater constancy in asbestosis.

Pathology. The fibrosis in asbestosis is diffuse and tends to predominate in the basal portions of the lungs in contrast to the generalized nodular fibrosis of silicosis

[73] J. W. G. Hannon, *Trans. Can. Inst. Mining Met.*, **48**, 180 (1944).
[74] L. U. Gardner, M. Dworski, and A. B. Delahant, *J. Ind. Hyg. Toxicol.*, **26**, 211 (1944).
[75] A. J. Lanza, *J. Am. Med. Assoc.*, **106**, 368 (1936).
[76] A. J. Lanza, W. J. McConnell, and J. W. Fehnel, *U.S. Pub. Health Repts.*, **50**, 1 (1935)

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with a predominance in the upper portions of the lung. Bronchiectasis and bronchiolectasis are frequent, especially in the more fibrous portions. Whereas the proliferation of fibrous tissue is caused by chemical action in silica exposure, it is induced by mechanical action in asbestos exposures. Gardner[77] found that the fibrosis-producing character of asbestos could be almost eliminated by grinding the fibers so that no particles more than 2 μ in length were present. As previously mentioned, asbestos is classified among the inert dusts.

Microscopic Anatomy. Johnstone[78] described the microscopic appearance of the lungs somewhat as follows: in the early phases of the disease there is thickening of the alveolar septa which results from fibroblastic proliferation. The alveolar spaces contain numerous phagocytes. With progression of the disease fibrosis becomes more marked; the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few alveoli that remain in the area of fibrosis are lined with low cuboidal epithelium giving them an almost glandular appearance.

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures described first by McDonald.[79] These bodies are 20 to 100 μ in length and are bulbous on one or both ends so that they appear club- or dumbbell-shaped. They are brownish in color, do not stain, and give a Prussian-blue reaction for iron. Simson[80] has produced these bodies in guinea pigs by experimental exposure to atmosphere containing asbestos. Lynch[81] concludes that these "curious bodies" signify exposure to asbestos dust but do not necessarily indicate asbestosis.

X-Ray Examination. For an excellent review of roentgenographic findings in both silicosis and asbestosis the reader might well refer to Pendergrass.[82] Characteristic differences in the roentgenographic findings in silicosis and asbestosis are tabulated below:

Asbestosis	Silicosis
Fibrosis diffuse (film may have ground glass appearance from pleural involvement).	Fibrosis nodular.
Findings may be either bilateral or unilateral.	Findings characteristically bilateral.
Lesions largely in the lower one half or two thirds of the lung fields.	Lesions predominately in the upper two-thirds of the lung fields.
Emphysema in upper portion of lung fields.	Emphysema in lower portion of lung fields.

Control. Prevention of asbestosis depends entirely upon preventing exposure to concentrations of dust sufficiently high to produce the characteristic reaction.

[77] L. U. Gardner, *Ind. Med.*, **9**, 45 (1940).
[78] R. T. Johnstone, *Occupational Diseases*. Saunders, Philadelphia, 1942.
[79] S. McDonald, *Brit. Med. J.*, **2**, 1025 (1927).
[80] F. W. Simson, *Brit. Med. J.*, **1**, 885 (1928).
[81] K. M. Lynch, *J. Am. Med. Assoc.*, **109**, 1947 (1936).
[82] E. P. Pendergrass, "Roentgen-Ray Diagnosis in Silicosis and Asbestosis," in A. J. Lanza, *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.

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Dreessen, DallaValle, Edwards, Miller, and Sayers[83] have found evidence to indicate that 5,000,000 particles per cubic foot of air is a satisfactory figure for the maximum permissible atmospheric concentration of asbestos to which workers may be exposed. Definite permissible standards, however, have not been established.

VI. Dust Causing Minimal Fibrosis or No Fibrosis

Gardner[77] has pointed out that any known inorganic dust other than free silica and the asbestos silicates may produce nonspecific dust reactions, as described on page 486. X-ray markings may be found in other conditions such as chronic infection and heart disease. No interference with pulmonary function or disability is produced by dust of this type and there is no influence on susceptibility to tuberculosis.

A. SILICATES

McCord,[84] from a number of sources, has compiled a list of the commonly used silicates, which is presented here:

Olivine—a magnesium silicate widely present in all basic rocks. Many varieties of this stone are known.

Calcium silicate—nonexistent in nature, but a common product of industry; may be found at blast furnaces and in production of cement and hydrated limes. Other silicates of calcium are known.

Willemite—a zinc silicate (rare).

Sodium silicate (meta)—the well-known water-soluble silicate, commonly called water glass. This is widely used in industry, notably as a filler in soaps. This is the only crystalline silicate of sodium, the others being amorphous glass.

Tremolite—a magnesium-calcium silicate.

Asbestos—tremolite, actinolite, chrysotile, or amianthus, all of which are essentially magnesium silicates.

Jade—another form of magnesium-calcium silicate.

Crocidolite—"blue asbestos." It is a sodium iron silicate.

Talc—a hydrated magnesium silicate with extensive industrial application.

Soapstone—a form of talc.

Agalite—a variety of talc resembling asbestos. It is used in the coating of paper, and as a paper filler.

Meerschaum—also called sepiolite. It is closely akin to talc.

Serpentine—hydrated silicate of magnesium. It is a common building stone.

Sillimanite—aluminum-containing silicate. The source of many stone tools of the stone age

Topaz—a semiprecious stone of silicate origin. Chemically it is an aluminum fluosilicate.

Fuller's earth—a hydrated silica-aluminum compound, associated with ferric oxide.

Kaolin (kaolinite)—many related silicates of mixed constituency.

Clays—hydrated aluminum silicates containing iron. Titanium, quartz and mica, are likely to be present in clays.

[83] W. C. Dreessen, J. M. DallaValle, T. I. Edwards, J. W. Miller, and R. R. Sayers, *U.S. Pub. Health Bull.* No. 241 (1938).

[84] C. P. McCord, *Ind. Med.*, 2, 4 (1933).

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