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REVIEW OF LITERATURE ON DUSTS

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A summary of some of the points mentioned or discussed follows:

1. Dust has been defined as solid particles (usually dry, but they may be wet) ranging in size from less than 1 micron (1/1000 mm. or approximately 1/25000 inch) to about 150 microns.

2. Physiologically, there are almost as many different classifications of dusts as there are authors on the subject; these classifications probably are not particularly important, because in daily practice in industry the harmful effect is produced more frequently (almost invariably) by dusts of mixed origin. An attempt to group dusts under the headings "active" and "inert" (with reference to effect on health) has proved unsatisfactory; the further the subject is investigated, the more it becomes apparent that virtually all dusts may be harmful if breathed in large amounts over long periods.

3. The extent of the dust hazard in industry in the United States is not known definitely, and exact data on the number of persons exposed are not available although some rough estimates have been made.

4. So far as known at present, silica dust apparently is the most harmful of the dusts ordinarily encountered. Silicosis (so-called), the disease caused by breathing silica dust, is a widespread industrial hazard which probably is increasing and affects appreciably the death rate among industrial workers exposed. The Silicosis (Medical-Arrangements) Committee of Great Britain reported in 1929 that "silicosis is more widespread than is generally believed and occurs to some extent in a number of industries and occupations where its presence has not been suspected." Some authorities in the United States, particularly lawyers who have handled any considerable number of dust cases legally, have expressed essentially the same opinion.

5. Various writers on industrial diseases have referred to harmful effects of exposure to dust for centuries, at least as far back as 75 A. D., when Pliny referred to it.

6. Dust diseases are found among workers in dusty trades all over the world but are more prevalent, or at least have received maximum attention, in such industrial countries as Great Britain, Germany, Australia, South Africa, Canada, and the United States.

7. In the United States dust disease, usually designated "silicosis," is more or less common among workers in the mining, grinding, granite, and foundry industries; it also is found to some extent in more than 100 other occupations or industries.

8. As regards types of dust injurious to health, it appears that any dust insoluble or difficultly soluble in the fluids of the respiratory passages and in sufficiently finely divided form to float in the air and be breathed by workers in considerable quantities over long periods ultimately will be harmful. Although free silica dust (SiO_2) has been considered the most harmful, silicates (for example, asbestos) have been found almost as harmful, and other dusts certainly have a detrimental effect upon health under certain conditions.

9. The following terms are commonly used to describe dust diseases of the lungs: (*a*) "Pneumoconiosis" (from Greek *pneumon*, lung, and *konis*, dust), a general term for all dust diseases of the lungs; (*b*) "silicosis," a condition of the lungs due to inhalation of silicon dioxide or free silica; (*c*) "silicatosis," a disease of the lungs thought

to be caused by silicates; (*d*) "anthracosis," a dust disease of the lungs of coal miners; (*e*) "siderosis," a diseased condition of the lungs of metal workers, particularly in iron or ores of iron; and (*f*) "asbestosis," a lung disease caused by breathing asbestos dust. The generic term "pneumoconiosis" or possibly the simpler one "dust disease" appears to represent more accurately the common types of dust disease, because numerous dusts usually are involved.

10. Silicosis is characterized by fibrotic changes in the lungs which are said to be more clearly noticeable and easier to diagnose than changes due to other dust diseases of the lungs. The dust of free silica is considered more dangerous than other dusts, chiefly because its victims are thought to be more liable to contract tuberculosis than persons exposed to nonsiliceous dusts.

11. The symptoms of silicosis are shortness of breath (dyspnea), pains in the chest, cough, expectoration (coughing and spitting material), hemoptysis (spitting blood), night sweats, loss of strength, and gastrointestinal symptoms; most of these symptoms also accompany diseases caused by at least some other dusts.

12. As outlined by Pancoast, the pathological features of silicosis are: (*a*) Entrance of dust; (*b*) "dust cell"; (*c*) entrance of dust cell into the lymphatic system of the lungs as a carrier of silica particles; (*d*) influence of deposited silica in the production of fibrous tissue; (*e*) action of silica; (*f*) elimination of dust; (*g*) predisposition of the fibrotic and silica-saturated lung to respiratory infections, especially tuberculosis.

13. The mechanisms provided to protect the lungs from accumulation of foreign particles are the mouth and nose, through which the respiratory tract opens to the surface of the body, the latter of which is guarded by a course filter of hair; behind this a series of tortuous passages with moist walls which trap many smaller particles; and cells covered by the cilia (minute vibratory hairs) which line the nasal cavities, portions of the upper respiratory tract, the pharynx, the trachea, and bronchi. The wavelike vibrations of the cilia tend to carry particles lodging on their surface away from the lungs and back toward the surface. These mechanisms, with others, are adequate to protect against ordinary amounts of atmospheric pollution, but if work is continued in a very dusty atmosphere for long periods, they cannot cope with the situation, the devices themselves are damaged, and the dust particles collect where the air should be or possibly prevent air from going where it is expected to go and is needed.

14. In some countries silicosis has been divided arbitrarily into three stages for convenience of description and possible compensation purposes: In the first stage (as designated in the United States) the symptoms are few and often indefinite, and the working capacity is not noticeably impaired; in the second stage a definite shortness of breath on exertion is usually experienced, work cannot be performed as well as formerly, and the chest expansion is noticeably decreased; in the third stage the shortness of breath is marked and distressing on slight exertion, the cough is more frequent, the capacity for work is seriously and permanently impaired, chest expansion is greatly decreased, flesh is likely to be lost, pulse rate may be increased, the heart may become dilated, and usually tuberculosis has intervened.

cations may ensue in 2 years or even less from the date of first exposure to heavy concentrations under unfavorable conditions of temperature, humidity, noxious gases, exposure, etc.

26. Determination of dust in air breathed by workers is held to be necessary before measures can be taken to prevent dust disease; moreover, it is economically important as evidence of the extent of the dust hazard involved in dusty processes and as a measure of the efficiency of protective devices introduced to mitigate the hazard. The various methods that have been and are being used are described. The final choice of an instrument for sampling dust depends on its efficiency, its relative freedom from errors in analysis, its ruggedness, portability, and weight, and the difficulty or ease with which the samples obtained may be analyzed. The instrument or method should eliminate as far as possible variation or errors due to the "human equation"—a difficult requirement and one apparently not yet reached. A suitable instrument or method for determining air dustiness in one industry may not necessarily be applicable to conditions in another, though, as far as feasible, instruments and methods should be kept standard r all industries to permit correlation of results.

27. Engineering meth ds of controlling dust in the mining, abrasive, grinding, foundry, and granite industries are discussed.

28. Textbooks on ventilation and engineering handbooks devote little or no space to methods of controlling industrial dust; less than 1 percent of the articles in an extensive bibliography on dust diseases deals with engineering control.

29. No definite, absolutely reliable standards of allowable dustiness are known; therefore the aim in the prevention of dust disease should be to eliminate dust from the atmosphere breathed by workers or at least to reduce the dust content to the lowest figure feasible. Considering the many uncertainties of the present day, standards—especially in legal requirements—should be tentative and flexible rather than permanent and rigid.

30. The remedy for the apparently concerted effort to hide the facts regarding dust diseases in industry is education of workers in the necessity of taking available precautions, including physical examinations; of employers in recognizing the seriousness of the situation and in providing devices and methods to reduce or prevent the incidence of disease and, if necessary, force their adoption on the workers; of doctors in correctly diagnosing disease, giving publicity to its prevalence, seriousness, preventive remedies, etc., and assigning in death certificates dust disease as the cause if such is the case; and of merchants, newspapers, and other influences in the community in trying to prevent the disease rather than to hide its existence.

31. Employers should not conclude that they have eliminated the danger of silicosis or of any type of pneumoconiosis simply because the air is seemingly free from dust; as far as now known, the particles likely to do the most harm are invisible and remain long in suspension in the air unless the latter is replenished by fresh, pure air at frequent intervals through ventilation.

32. The following methods of reducing dust in South African mines are said to have been effective in preventing the incidence of dust diseases for the past several years: Wet drilling, water spraying (by

water blast), filtration, requiring adequate time between blasting and return to the working face, air sampling, ventilation, and wearing of masks.

33. The main sources of dust in metal-mine air in the order of importance are: Dry-drilling holes for blasting, blasting, shoveling or "mucking" very fine dry material at the working face which is usually poorly ventilated, loading cars from chutes, dumping loaded cars into chutes, and timbering. Dry crushing and other occupations in metal-mine mills are also likely to be dangerously dusty.

34. The most dangerously dusty occupation in coal mines is cutting or loading dry coal by machines; blasting, shoveling, drilling, and rock dusting by machinery are also very dusty operations. In addition to the miners and their helpers, rockworkers and timbermen and drivers who must enter the miners' workplaces when the air is particularly dusty are exposed to relatively large amounts of dust.

35. The best remedy for the dust menace in mines, other than preventing its formation, appears to be the universal coursing of currents of air to remove the dust, as it has been proved that the very fine, most dangerous dust in metal mines remains suspended in still air for several hours. Dust-prevention devices of proved success, such as the present-day self-rotating wet stoper and other wet-drilling equipment, should supplant dry drilling; their use should be enforced upon both miners and operators in metal mines. Care should be taken, however, that holes are not started or "collared" dry. To date no workable device is available for removing dust in dry drilling in underground mines.

36. Various "dust traps" to prevent dissemination into the atmosphere of dust produced by drilling in rock with pneumatic percussion drills are described; these devices are used in mines in the United States to only a very limited extent.

37. The advantages and disadvantages of dust respirators are discussed. On August 20, 1934, the Federal Bureau of Mines issued an approval schedule for dust respirators describing the procedure for testing filter-type dust, fume, and mist respirators for permissibility; a number of respirators have been approved under this schedule.

38. Dust filters developed in South Africa for removing dust produced in ore bins and at tipple stations are said to have dust-catching efficiencies of 98 percent or more.

39. In industries other than mining the processes used vary so widely and call for such different treatment that each must be studied separately. Methods recommended for eliminating dust are: Local exhaust ventilation to remove the dust at point of origin; substitution of non-silicosis-producing material as a metal abrasive for sand, nonsiliceous parting compounds for molds in foundries, and clay instead of flint in bedding potteryware for baking; enclosure and segregation of dusty processes, as the use of sandblasting cabinets; suppression of dust by water spray and steam; general mechanical ventilation; plant cleanliness, which is as important as upkeep of protective equipment; protection of workers by masks and helmets, although such devices should be considered only emergency equipment as dustiness should by all means be controlled by such measures as dust-collecting equipment and exhaust fans if feasible; medical supervision with

periodical physical examinations of workers; education of workers; and dust counting to check the efficiency of the measures.

40. If much progress is to be expected engineers, operating officials, and doctors must cooperate in the control of dust diseases. The most important measure in the medical control of dust diseases is physical examination of workers before employment and at stated intervals thereafter. It has been found that certain defects and diseases are predisposing causes of silicosis. According to German investigators, workers with poor nasal filtration are more susceptible to dust diseases. A man with a good, sound physique and with good reserve respiratory capacity is said to be best-suited for underground work in mines. Age is also important; a man with a good chest and good reserve can carry without inconvenience or loss of health enough fibrosis to seriously affect one with poor reserve or a chest becoming rigid with age. All persons with signs of tuberculosis taint or tendency should be rigorously excluded from dusty occupations, irrespective of silica content of the dust.

41. A plan for medical control includes establishment of a medical department adequately equipped, routine examination of applicants for employment, rating and placement of applicants, periodical physical examinations, and provision for the disabled. Methods of conducting examinations are outlined.

42. No cure is yet known for silicosis. At one time it was thought that removal of the victim from further contact with dust would arrest the condition at the point of development reached when discovered; although this practice is effective in some instances, generally it is not now considered the solution, and in some mining localities the removal system is not utilized.

43. Earlier investigators believed that so-called "protector" dusts would cause excretion from the lungs of such harmful dusts as silica by exciting the physiological process of elimination and that these protective dusts acted in some way to prevent the development of tuberculosis; some still hold this opinion. Coal dust, iron-ore dust, clay dust, and dusts of calcium minerals have been suggested and tried in the treatment of silicosis; there has been great divergence of opinion of various investigators regarding the harmfulness or harmlessness of these dusts and their efficacy in preventing silicosis and tuberculosis, when mixed with dusts definitely agreed upon as harmful, or their harmfulness when breathed alone. However, the belief is growing that any dust or combination of dusts if breathed in large enough quantity over a long enough period will cause diseases of the respiratory organs.

44. Experiments by Canadian investigators led to the suggestion that the inhalation of aluminum dust might prevent if not cure silicosis. The method of application was developed at the McIntire Mines, Ltd., of Canada and is being tried at a number of mines in the United States and some other countries. The theory of aluminum therapy in silicosis is based on the hypothesis that chemical action, not physical irritation by silica, is the cause of silicosis. It was observed by some investigators that aluminum almost completely inhibited solution of siliceous material.

45. Animal experiments indicate that the prophylactic use of aluminum inhibits the toxic action of relatively pure quartz. The therapeutic use in man appears to relieve symptoms in some cases. As human silicosis develops slowly, only prolonged, unbiased observation, with adequate control cases, will demonstrate its value in the treatment and prevention of silicosis.

46. It has been recommended that general application of aluminum therapy in industry be delayed until adequately and impartially controlled clinical observation demonstrates its effectiveness in preventing or alleviating silicosis in man. Meanwhile, there should be no slackening in the control measures that have been found effective in reducing the incidence of dust diseases in industry.

47. It is recognized that, in extending compensation coverage to occupational diseases, the subject is much more complex than that of accidents. In 1948, 39 States included silicosis in their occupational disease compensation laws.

48. The status of compensation for silicosis in the United States, Canada, Great Britain, South Africa, and Australia is summarized, and the important features of the laws and methods of enforcing them are more or less briefly described.

49. The main effectiveness of the preventive aspects of silicosis schemes is said to lie in their power to set up a standard of physique for new entrants into the industries, based on periodical medical examination; this procedure would have ramifications affecting safety, production, and probably other phases of industrial work also.

50. The pressure of occupational disease (especially dust disease) on insurance companies increased so much as to threaten financial ruin in some instances; consequently, insurance companies are decidedly active in connection with investigations, research, and all other phases of occupational disease, especially dust disease.

51. Much of the information regarding the cost of legal compensation for silicosis has been obtained from foreign countries, especially the British Empire, as most of the silicosis-compensation laws in the United States are too recent to determine the cost of their operation.

52. The most complete figures available are probably those for South Africa. The total amount paid for silicosis compensation since enactment of the miners' phthisis laws of South Africa to 1934 exceed £14,000,000 (about $70,000,000). The cost per ton of ore milled was about 6d. (about 12 cents) and per ounce of gold recovered, 1s. 7d. (about 38 cents); the cost per underground European shift was about 4s. ($1). In Canada in 1934 the cost of silicosis compensation was 1¼ percent of the total mine pay roll, although only 2½ percent of the men employed by the mining companies were actually exposed. The estimated cost of each case to the company was $11,000 to $12,000. It was also said that, for every $5 spent in mining and concentrating 1 ton of gold ore, $1 was required for silicosis.

53. The cost of cases of silicosis in Wisconsin for 1942 was $122,039; for 1943, it was $30,199; for 1944, it was $44,837; and for 1945, it was $48,898.

54. No question offers greater difficulty in the administration of workmen's compensation laws than that of determining the nature and extent of disability in silicosis. It is known, however, that silicosis

can be present in diagnosable form in a person who will still possess enough capacity to permit full employment. Information is given on various methods for the determination of vital capacity and working capacity.

CONCLUSIONS

The literature on the effect of breathing dusts abounds in various experimental, theoretical, and factual data; it comes from nearly all civilized countries; it covers nearly all phases of the subject but lacks conclusiveness in almost any or every phase except possibly that harm to health can be expected from prolonged breathing of excessive amounts of dust. There appears to be good reason to believe that this applies to virtually any or all dust or combination of dusts, organic or inorganic.

Almost innumerable uncertainties are connected with the harmfulness of dust, but dust is now generally believed to be detrimental to health through its effect of one kind or another on throat, bronchial passages, lungs, stomach, and other internal organs, as well as eyes, ears, nose, other external organs, and the skin covering the entire body. Many dusts (metal, mineral, and organic) are explosive; others fire spontaneously with more or less readiness under varying conditions; and virtually all dusts have almost universally detrimental effects upon settlement, whether in the home, in the office, or on the external surfaces of buildings or on the roads, streets, or sidewalks. All of these facts (and others can be added) make dust one of the real scourges of present-day life, and certainly no sane person can deny that a most determined effort should be made to reduce dust or at least to confine it in such manner as to minimize its numerous harmful effects.

It now seems probable that the most harmful of the ordinary dusts is that of silica; on the other hand, not even free silica (supposedly the most detrimental of the silica dusts) is harmful unless it is breathed in considerable *quantities* and over comparatively long periods; no human being has ever lived any length of time without breathing silica dust (free silica dust), yet by no means all of the people of the world have or have had silicosis. In other words, the *quantity* of dust taken into the respiratory organs is a controlling factor in dust respiratory harmfulness, and common sense indicates that quantity of dust breathed is one of the dominant factors as to possible harmfulness from any kind of air-borne dust. What constitutes that quantity limit no one knows, notwithstanding the fact that numerous so-called threshold limits are now being announced, some of them embodied in State regulations or laws. Predicating dust respiratory harmfulness on a sliding scale in proportion to the free-silica content of the dust is anything but logical; in the first place, no one knows whether a dust of 1-percent silica is or is not more harmful than a dust of 2- or 3-percent or even of 30-percent. Moreover, air with a certain number of dust particles per cubic foot with a silica content of 1 percent (or any other percentage) probably would be far more harmful to a person working on a contract basis than to one on a day-pay basis, or more harmful to a person working in an atmosphere of 85° or 90° F., relative humidity 90 or 95 percent, than to one working in an atmosphere of 60° F., relative humidity 60 percent. Numerous other more or less

similar contributing factors make unworkable or futile the establishment of regulations as to allowable number of dust particles on a sliding scale in proportion to silica content of the dust. In some industries, such as metal mining and tunneling, there may be as many different percentages of silica in the mine air as there are working places, and in many metal mines it is very unlikely that the percentage of silica in the air of a working place today will be at all close to the percentage of silica in that same place tomorrow; to apply to most metal mines the sliding scale of allowable number of dust particles in the air in proportion to silica content would be utterly impossible of enforcement or fulfillment.

Much should be done to clarify numerous uncertainties as to air-dust harmfulness before any attempt is made to establish more or less rigid air-dust standards by legal means. At present there is a definite lack of knowledge as to the size of air-borne dust likely to be harmful, and present-day so-called standards of air dustiness are built on numerous uncertain premises on this point alone. Different authorities have found, in the lungs of deceased silicotics, varying maximum and minimum sizes of dust, ranging from as small as 0.2 micron to as large as 10 microns; this fact leads to the conclusion that, as far as harm to the lungs is concerned, the dust to be avoided is that from 0.2 to 0.5 micron on the low side to 6 or even as high as 10 microns on the high side; this leaves much leeway, because an air-dust sample containing 10,000,000 particles (0.5- to 6-micron size) per cubic foot of air might easily have 30,000,000 particles 0.2 to 10 microns in size. Moreover, if the solution theory of dust harmfulness to the lungs is correct, there is the best of reasons to think that particles much smaller than 0.2 micron are very likely to be harmful. To hold that the only harmful sizes are those found in the lungs is anything but good reasoning, because certainly dusts ranging from 10 to 100 microns or more when floating in the air are drawn into the nostrils and other protective air passages and if in large numbers soon clog these protective agencies and passages, permitting virtually free unobstructed entrance of the so-called harmful dusts (say, 0.2- to 10-micron sizes) into the lungs to perform maximum injury. It does not seem logical to disregard these larger sizes entirely, as is now done almost universally in the so-called dust standards being considered and announced.

Again, the present-day instruments and methods of sampling air-borne dust and determining the number of particles present are anything but accurate or definite, even when handled by technically trained experts, and methods or instruments applied to certain industrial conditions are wholly unsuited to others. A condition as to air dustiness found in one sample in a certain place is unlikely to be anything like another sample taken immediately afterward in the same place and in the same manner; this is true where the sampling is done instantaneously by grab method or over a period of several minutes. In other words, the technic of determining quantity of dust particles in air is by no means definite, accurate, fair, or dependable, not only as regards conditions between different plants but between different time periods in the same place in any one plant. In the face of all these (as well as numerous others) uncertainties as to airborne dust, the attempt to embody in laws and regulations having

the force of law rigid standards as to air dustiness would seem to be a travesty on justice. If quantity standards as to air-borne dust are made, they certainly should be labeled tentative, and the technic of taking the air samples and making the particle determinations should be outlined and enforced very carefully; otherwise, much injustice can be inflicted through inexperience or the malice of the persons making the determinations.

This review of the voluminous literature on harm to health caused by breathing inorganic dusts has been confined to readily available data and almost wholly to respiratory disease, chiefly lung affections. It gives little or no consideration to the numerous ill effects to human organs, other than the lungs, due to breathing more or less insoluble dust or the harm to other internal organs (stomach, liver, kidneys, etc.) or to the nose, throat, and bronchial tubes from breathing more or less soluble or so-called poisonous dusts; nor does it take into consideration the numerous hazards to human beings in breathing organic dusts, with accompanying hay fever and kindred ills. Moreover, numerous dusts, both organic and inorganic, have harmful effects of various kinds on eyes, ears, and other parts of the body and on the skin from external contact; indeed, some dusts cause harm to health by absorption through the skin. Hence, while pulmonary disease due to the breathing of air-borne dust is unquestionably of great importance, its various ramifications (pneumoconiosis, silicosis, anthracosis, or these combined with tuberculosis), while vitally important, constitute by no means the only detrimental effect to human beings in coming in contact with dust, externally or internally.

Close analysis of the status of dust in industry (and in general life outside of industry also) indicates that numerous—one might almost say innumerable—uncertainties still exist as to specific features connected with dust harmfulness. So numerous and far-reaching are these uncertainties (only a few of which have been indicated in these conclusions) that almost the only definite fact is that dust is a menace and that all kinds of it likely to come in contact with human beings should be reduced to a minimum or at least be held under positive control until much well-planned, well-correlated research and investigation (field and laboratory) have been conducted on almost every phase of the subject.

DEFINITION AND CLASSIFICATION OF DUSTS

According to Webster's Unabridged Dictionary, dust may be defined as fine, dry particles of earth or other matter so comminuted that they may be raised and wafted by the wind; that which is crumbled to minute portions; fine powder. In 1876 Richardson (*1*) [1] included in the term "dusts" all those fine, solid particles thrown off from various substances in the processes of manufacture or treatment of articles in common use in daily life. Drinker (*2*) in 1930 defined dusts as solid particles ranging in size from less than 1 micron [2] to about 150 microns; this definition is probably as applicable as can be had insofar as concerns respiration.

[1] Italicized numbers in parentheses refer to bibliography at end of bulletin.
[2] A micron is 1/1,000 mm or approximately 1/25,000 inch.

Physiologically, there are almost as many different classifications of dust as there are authors on the subject. Richardson (*1*) suggested the following:

(*a*) *Cutting dusts*, formed of minute, hard, crystallized particles with sharp, cutting, and pointed edges and composed of iron or steel, stone, sand, or glass, dried silicates in earthenware, lime, and pearl.

(*b*) *Irritant dusts*, derived from woods, ivory, textile fabrics, fluffs of wool, silk, cotton, flax, hemp, hair, and clay.

(*c*) *Inorganic poisonous dusts* charged with arsenical salts, derived from poisonous chemical compounds used for coloring artistic products or for preserving organic substances, such as furs.

(*d*) *Soluble saline dusts*, organic poisonous dusts thrown off during the making of tobacco into cigars and snuff and carrying with them particles of the dried tobacco plant.

(*e*) *Obstructive and irritating dusts* composed of carbon, fine particles of coal dust, scrapings of carbon or soot, dust of rouge, and flour.

Baskerville (*3*) in 1912 classified dusts as:

(*a*) *Insoluble inorganic dusts*, including metals (antimony, arsenic, type metal, brass, bronze, copper, aluminum, iron, steel, lead, manganese, vanadium and ferrovanadium, silver, tin, zinc, and solder) in a state of fine division (dusts, atomized metals, and metallic powders); flue dusts; various ore dusts (iron ore); silica; sand, emery, flint, and glass powders; carbon graphite, diamond, coal, and soot; brick dust, marble, granite, cement, and terra cotta; lime, gypsum, plaster, and meerschaum; phosphates; and guano.

(*b*) *Soluble inorganic dusts*, including substances likely to be swallowed and absorbed, such as metal particles, including lead, brass, copper, zinc, arsenic, mercury, and silver, as well as soluble inorganic salts.

(*c*) *Organic dusts*, comprising such widely varying materials as sawdust, fur, skins, feathers, broomstraw, grains, flours, jute, flax, hemp, cotton, wool, carpet dust, street sweepings, tobacco-box dust, hides and leather, felts, rags, paper, and horsehair.

In 1918 Hoffman (*4*) considered the following classification to be in strict accord with the facts as they were known and understood at that time:

(*a*) *Inorganic dusts*, including metallic dust, mineral dust, and dusts of the mineral industries.

(*b*) *Organic and miscellaneous dusts*, including vegetable-fiber dust, animal and mixed-fiber dust, organic dust, and mixed organic and inorganic (public) dusts.

Thompson (*5*) classified dusts as: (*a*) *Insoluble inorganic dusts* (irritating the respiratory passages), as flint, silica, sand, carbon (coal soot), brick dust, marble, granite, terra cotta, cement, asphalt, enamel, glass, quartz, lime (gypsum, plaster), meerschaum, phosphate (fertilizers), guano, emery, diamond dust, metal filings (lead, brass, iron, steel, etc.), pumice, and ashes.

(*b*) *Soluble inorganic dusts* (liable to be swallowed and absorbed), as soluble arsenic, mercury, lead and silver compounds, metal filings of lead, brass, and zinc.

(*c*) *Organic dusts and fibers* arising from handling or manufacture of wood, bone, and shell, fur, skins, hides and leather, feathers, brooms and straw, flour and grain, jute, flax (linen), hemp, cotton, wool (worsted, etc.), tobacco, felt, carpets, rags and paper, horsehair, and street sweepings.

De Balsac and Agasse-Lafont (*6*) suggested the following classification:

(*a*) *Active dusts*, which are immediatley disseminated and radiate beyond their point of application; toxic dusts (lead, arsenic, and mercury); caustic dusts, chromates, etc.; infectious dusts.

(*b*) *Inert dusts*, soft, flexible felting dusts (wool, cotton, and leathers); hard, troublesome, wounding dusts (ligneous, metallic, stone, and coal dusts).

Schürmann (*7*) differentiated dust, according to origin, into animal, plant, and mineral dusts and dust from artifacts.

Animal dust is evolved with the working of ivory, horn, whalebone, bones, mother-of-pearl, hides, leather, bristles, sheep's wool, hair (horse, rabbit, and cow), and feathers.

Plant dust originates in industries working with grain, medical powders, spices, cotton, hemp, jute, flax, tobacco, wood, thick-shelled nuts, bark of plants (tanning industry), rags, shoddy, and paper.

Mineral dust is formed in working hard coal, marble and other limestones, and in connection with clay and porcelain industries, also pumice, sandstone, granite, and slates.

Dust from artifacts is encountered in the glass, glazing, enameling, tile, cement, Thomas slag, celluloid, iron, steel, bronze, galalith, and lead-alloy industries and in the chemical (especially dye) industry.

Schürmann (*7*) defined mixed dust as a mixture of polishing materials and the fragments of the object being polished.

According to Drinker (*2*) all these classifications have little practical importance, since in daily practice in industry the harmful effect is exercised more frequently by dusts mostly of mixed origin. Most important, moreover, is the physical and chemical constitution of the dusts and consequently their action on the human system. An effort has been made to group under the heading "Inert" a certain number of dusts; but the number is becoming more and more reduced since certain dusts, such as talc and asbestos and even coal dust, until recently considered as inert nevertheless have caused serious organic lesions among certain classes of workers. The majority of experts admit that the so-called inert dusts are not really inert when considered from the pathogenic point of view. Although at the outset these dusts are not harmful and even when present in the workroom in large quantities some of them cause only transitory discomfort (sneezing, watering of the eyes, and cough), it is certain that, as the organic reactions diminish and the system seems to have recovered from the effects, they nevertheless finally attack it in a subtle manner, sometimes even seriously. The mucous membrane of the nose, the conjunctiva, first the upper and later the lower respiratory passages, the teeth, the skin, and often the digestive tract are subjected to the harmful action of these dusts.

As indicated by the above discussion, an absolute classification for dusts is difficult. Opinion regarding so-called inert or harmless dusts

has changed as further investigation has proved some of them to be injurious. A convenient classification, according to physical characteristics and physiological effect, has been used by Sayers (*8*). He classified dusts into the two main categories of organic and inorganic. The organic he divides into nonliving and living organic dusts, with further subdivision of the nonliving into toxic and (or) irritant dusts and allergic dusts and the living into bacteria and fungi. The inorganic dusts are subdivided into toxic and (or) irritant, fibrosis-producing, and non-fibrosis-producing dusts.

EXPOSURE TO DUST

Prehistoric man, who originated the trade of making stone implements, probably started the first industrial hazard, the extent and severity of which the medical profession, as well as the industries concerned, has begun to realize only comparatively recently. In fact, dust as a factor in the causation of disease had not received much attention before 1900. At the site of one of the Swiss lake dwellings where flint implements were found, although the flint must have come from a distance (probably from the south of France), the chippings of material were in such profusion as to imply that the implements were manufactured on the spot. In other words, a prehistoric flint-knapping factory was probably located there (*9*). Some of the persons who prepared these implements no doubt suffered from respiratory diseases. According to Collis (*10*), the flint knappers of Brandon, the lineal occupational representatives of this oldest of industries, who still use tools similar in shape to the deerhorn picks of their prehistoric ancestors, suffer a terrible mortality from phthisis induced by flint dust generated in their work. The work is carried on in small workshops at the back of cottages in the rural district bordering Norfolk and Suffolk, where large flints or potstones are found; the district has been known from remote antiquity for the manufacture of arrowheads and other prehistoric implements, tinder boxes, and in later years flints for flintlock guns.

Carozzi (*11*) has questioned the hypothesis of an occupational pneumoconiosis in prehistoric man. The conditions requisite for the development of this occupational disease do not seem to Carozzi to have been present in that distant epoch. For example, if it is true that even today, in dusty industrial surroundings, a long period of exposure to the dust is necessary, and if it is true that the average duration of life did not exceed 30 years, it is doubtful that mortality from a form of pneumoconiosis attained a high value. Carozzi (*11*) states, however, that it is certain that prehistoric man suffered from a disease of the nature of tuberculosis.

Dusts of various kinds are carried in the atmosphere in all parts of the world; and the inhalation of these dusts over periods of years inevitably produces changes in the lungs, as, for example, the pigmented lung of the city dweller (*12*). The following table by Landsberg (*13*) indicates the amount of dust that may be present in the air under different conditions:

Kind of air:	Particles per cubic centimeter (large particles only)
Mountain	2
City, residential quarters	15
City, business center	100
Cement factory	1,200
Granite cutting	2,100
Bituminous-coal mine	3,900
Anthracite mine	4,400
Grinding shops	6,700

Landsberg (*13*) has estimated that the air inhaled by a normal person during a working day contains roughly 12 billion dust particles. Pfaff (*14*), however, found the values for city dust in Saarbrücken, Germany, to lie between 250 and 2,800 particles per cubic centimeter; in streets with heavy traffic he found 4,000 particles. This difference in results probably is due, at least in part, to the size of particles; Landsberg (*13*) states that he counted "large particles only" but does not say how large, while Pfaff (*14*) counted those for the most part under 1/15 micron. The composition of the dust is not given in either instance.

Soper's (*15*) report on the air and dust in the 21-mile subway of the Interborough Rapid Transit Railroad of New York City, published in 1906, likewise deals with general exposure to dusts. Chemical analysis of the dust underground showed that it contained 61.30 percent of iron, nearly all of which was in the metallic state, 21.94 percent of organic matter of vegetable and animal origin, 15.58 percent of silica and other matters insoluble in acid, and 1.18 percent of oil. There were, on the average, 61.6 mg. of dust in 1,000 cubic feet of air; the maximum weight was 204 mg. The dust in the air of the subway was 11 to 800 percent heavier than that in the street air. Although some of this dust was carried in from the streets, much of it was of underground origin from gradual wear and tear of the wood, cement, and other materials used in constructing the subway and from operating the trains; the largest percentage was iron dust from the grinding action of the powerful brakes on the cars. The loss of weight in brake shoes alone was estimated as about 1 ton per mile of track per month, and when the loss from the wheels and the railway track is added to this, the origin of the metallic dust is explained. The men had not been employed underground long enough at that time to show the effect on health of inhalation of the dust. Although no serious disease was found among them, many suffered from inflammatory affections of the nose, throat, and windpipe and from "dry pleurisy" unaccompanied by pain.

Hoffman (*4*) prepared an occupational grouping to emphasize in a general way the principal dust hazards in 118 occupations or groups of employment, which he said was in strict accord with the facts as they were known and understood at that time (1918). Under the heading "Organic and miscellaneous dusts" he listed 65 occupations; under the heading "Inorganic dusts" he listed 52 occupations.

Using the United States census for 1919, he estimated that 3,928,978 persons were employed in the dusty trades; of these 1,667,181 were listed as employed in occupations exposed to metallic and mineral dust and dust in the mineral industries. He considered that quantitatively

the most important kind of metallic dust encountered under typical industrial conditions is the dust of iron and steel, which, however, is generally more or less intermixed with dust of other metallic or mineral substances. Exposure to mineral dusts is most common in the stone industry, among potters, in cement manufacture, and in mining.

In a paper on the extent and severity of the health hazard from dust in mines and allied industries in the United States van Siclen (*16*) gave the following estimates, based on the 1929 decennial Federal census, of the number of men exposed to dust hazard in various types of mining and industries working in metals and minerals:

	Total workers exposed
Metal mining	62,268
Nonmetallics mining, excluding tunnel and foundation excavations	23,665
Bituminous-coal mining, underground	450,513
Bituminous-coal mining, open pit	8,219
Anthracite mining, underground	143,083
Anthracite mining, independent washeries	269
Smelting, nonferrous plants	13,166
Smelting, ferrous plants	24,980
Cement plants	33,368
Abrasive industry	3,873
Asbestos products	8,092
Clay products	93,336
Cutlery	14,091
Glass manufacture	67,527
Granite, slate, marble, and other stone products	28,715
Hones, whetstones, and similar products	174
Iron and steel	39,697
Mineral fertilizers	20,926
Minerals and earths, ground	1,679
Nonferrous-metal alloys and products	79,183
Pottery, including porcelainware	33,409
Sand-lime brick	500

In his conclusion he stated:

> No totaling of the number of workmen exposed to the dust hazard in the mining and allied industries in the United States, as estimated in the preceding pages, has been undertaken, first because the estimated figures themselves are subject to revision after more detailed study of the mineral industries and manufactures, and second, because the degree of the dust hazard varies with the several industries, with the manifold occupations in each industry, and between similar positions in different plants of the same industry. A lump-sum total would therefore have no real meaning. The separate totals, however, are sufficient to indicate the wide extent of this kind of hazard.

In 1947 the weekly average employment in the hard-coal mines of Great Britain (*17*) exclusive of ancillary plant and office workers was 718,000. During the same year 320,000 men were employed in the hard-coal mines of France and 160,000 in the mines of Belgium. As of December 31, 1940, about 400,000 men were employed in the coal and metal mines of South Africa (*18*). Probably 150,000 persons were employed in 1930 in the quarrying, cement, pottery, and stoneware industries of Germany (*19*). Of 217,000 men employed in the coal mines of the Ruhr district in 1933, 11,500 were said (*20*) to have been employed in work involving exposure to rock dust. In 1947 the average daily employment in the British zone of Germany was 287,000 and in the American zone 8,000; 41,000 were employed in the Saar,

39,000 in the hard-coal mines of the Netherlands, and 27,000 in the Italian mines (*17*).

The above figures on number of employees in the dusty trades are given merely to indicate the possible exposure to the dust hazard; they are in no way complete and, of course, do not show the actual number subjected to the hazard.

In 1934 Lanza and Vane (*21*) stated that silicosis was associated with certain industries as a major hazard, and the number of persons engaged in these industries could be estimated with some degree of reliability. In a great many other industries, however, isolated jobs or processes might involve exposure to silica dust, and only a guess could be hazarded regarding the number of workers in these processes. From a summary of the facts, these authors concluded that approximately 450,000 workers are exposed to silica dust in the chief mining, quarrying, and manufacturing industries, and 100,000 in other industries and processes; the 500,000 they gave as a final conservative estimate of the number of workers in the United States exposed to silica dust to a harmful degree probably is still applicable. It is apparent, therefore, that silicosis has been and still is a widespread industrial hazard, is probably increasing, and affects appreciably the death rate among industrial workers exposed.

Only dusts encountered in the mining and allied industries and the diseases caused by them are considered in this paper.

PHYSIOLOGICAL EFFECTS OF BREATHING DUST

HISTORICAL RÉSUMÉ

Following is a brief reference to some of the many authors who have added to our knowledge of the history of silicosis. For a more detailed and complete historical résumé, the reader is referred to Carozzi's contribution covering the period from the 27th century B. C. to 1871 A. D. (*11*).

Probably the oldest published statement regarding the harmfulness of exposure to dust is one by Pliny (*22*):

> Minium refiners in the factory envelop their faces with loose bladders, which enable them to see without inhaling the fatal dust.

Hippocrates (*23*), who was born about 460 B. C., called attention to the difficult breathing of the metal digger. Celsus, a Roman medical writer who lived in the first century, stated:

> By far the most terrible form of emaciation is that which the Greeks call phthisis. It spreads to the lungs. On top of this, ulceration occurs and a slow fever which at times disappears and at other times reappears.

In quoting this passage Mavrogordato (*24*) said that when early writers discussed dust phthisis one may assume that the disease they had in mind was of the nature mentioned by Celsus.

In 1551 Amatus Lusitanus (7) reported that most workers occupied with the preparation of gypsum and lime died of lung phthisis.

Agricola (1494–1555), in his De Re Metallica (*25*), published in 1556, described mining as—

> A perilous occupation to pursue because the miners are sometimes killed by the pestilential air which they breathe; sometimes their lungs rot away.

* * * Some mines are very dry and the constant dust enters the blood and lungs, producing the difficulty of breathing the Greeks call asthma. When the dust is corrosive it ulcerates the lungs and produces consumption; hence, it is that in the Carpathian Mountains there are women who have married seven husbands, all of whom this dreadful disease has brought to an early grave.

In the sixteenth century, according to Georgius Agricola, it was known that permanent injury to the lungs resulted from exposure to certain kinds of dust but not to all kinds (*25*). Two kinds of injurious dusts were recognized—a corrosive and a noncorrosive type. Each of these classes was associated with a different kind of lung change, "simple" and "infective" silicosis, respectively, described in the 1916 General Report of the Miners' Phthisis Prevention Committee of South Africa.

Paracelsus, a Swiss alchemist and physician, in his book (*26*) on miners' phthisis and other miners' diseases, described especially the chronic lung troubles of miners as "lung consumption," "asthma," and "dyspnea." He was the first to list briefly the occupational diseases of miners and smelters as well as the first in the world literature to prepare a monograph on industrial medicine (*26*). From 1531 to 1534 Paracelsus revisited the mines and smelters of Schwaz in the Tyrol, where he had worked as a laborer between 1510 and 1520, and wrote his book, which was published in 1567 after his death. He found that—

Miners in metal mines, when they are occupied in digging, smelting, and washing gold, silver, salt, alum, sulphur, lead, copper, zinc, iron, and quicksilver in the refining of vitriol, suffer from various disturbances of the lungs, stomach, and intestines; they are then said to have "miners' diseases." However, there is nothing found in the works of the old writers in regard to these diseases.

His theory was that lung diseases contracted above ground depended upon climatic conditions as influenced by the rays of the stars. Similarly, pulmonary disease of miners underground was caused by mineral rays. However, he philosophically reminded his readers that—

We need metals and, therefore, we must risk life and health for them, since everywhere in nature good and evil lie together. As the crocodile distresses and kills men by his breath, likewise also the vapors (fine dust?) of such metals kill us. * * * The organism must be prevented from coming in contact with the metal emanations; for if the organism is once injured there is no cure.

The untimely death of Paracelsus was attributed to injuries to health received during his activities in the mining and metallurgical industries.

Pancoast, a more recent author (*27*), likewise referred to the increasing interest in the subject as a recognition of the conditon of pneumoconiosis as a more or less necessary risk of commercial development in the progress of civilization in recent years.

Pansa, in 1614, was the first to discuss in detail lung disease of miners and the asthma of grain measurers (*7*). In 1649 Van Diemerbrock was reported (*28*) to have made the first section of a stonecutter's lung which, in a case of fatal asthma, revealed "lung vesicles completely clogged with fine dust"; as quoted by Ramazzini (*29*), he found such heaps of sand that in running the knife through the pulmonary vesicles he thought he was cutting some sandy body.

Carozzi (*11*) quoted Fourcroy regarding this incident as follows:

> In dissecting at the hospital the domestic of a lapidary, who died of asthma, he found the pulmonary vessels filled with dust of the diamond.

In 1652, Ursinus (1618–64) described (*30*) the lung disease that attacked especially the miner but also the smelter. He divided lung diseases into two classes—one was caused by poison, and the other was not. The principal symptoms were coughing and shortness of breath. Etiology was a question of breathing dust, catching cold, fatigue, injury, and the like. He said the lung disease due to "poison" was not so clear and was unknown before Paracelsus.

In his book published in 1656, Stochausen (*31*) defined miners' phthisis as "difficult breathing, chest asthma with disagreeable hard cough and considerable hoarseness, which effects generally degenerate into fatal consumption." He attributed these symptoms to metallic vapors, dust, and meteorological conditions, from which miners suffer more or less and call "miners' consumption" because the affliction arises especially in connection with mining. The actual causes, he said, were—

> especially thick, lowering clouds and mine dampness, watery humidity, all sorts of vapors of earth and metal, various fumes and smoke, different dusts and dirt.

He also said that much dust arose from grinding, so that workers were forced to cover nose and mouth with cloths.

Etmüller, 1648–82, a professor at Leipzig about 1670, described (*32*) a case of a miner who—

> was seized first with cough and then with great anxiety and difficulty of breathing, especially at night; he used to jump out of bed and open the window for fresh air.

In 1690 Löhneiss (*33*), referring to miners, gave the morbid sequence of events as follows:

> The dust and stones fall upon the lungs, the men have lung disease, breathe with difficulty, and at last take consumption.

Ramazzini (*7*) was the first to recognize the social significance of industrial diseases from chemical substances. In his book published in 1700, he mentioned the harmful effect of dust on the respiratory organs. For information he went to the workers in the occupations, entered the mines, ascended the mine shafts, and collected the experiences of his occupational colleagues. His book was based on a study covering more than 40 years.

Flint dust has long been recognized in England as injurious; a patent for grinding flints by a wet method was granted in 1713 to Thomas Benson, of Newcastle-under-Lyme (*34*); before this date, flints had been pounded dry, which process proved very destructive to mankind, insomuch that any person, ever so healthy and strong, working in that business, could not possibly survive over 2 years, occasioned by the dusk sucked into his body by the air he breathed.

In 1770 Scheffler (*32*), a mine physician in Saxony, wrote that miners were attacked by an asthmatic condition which he called sicum-drum asthma. According to the Mining Journal (*32*), the older mine physicians called it asthma metallicum or montanum-metal or mountain asthma.

Scheffler (*35*) realized that the description of miners' phthisis given by previous medical men was insufficient. His conception of it was a chronic slow fever with hardening of the glands and obstruction of the lungs, with lack of elaboration, secretion, nutrition, and apposition which, through the arsenical or other dusts, dries out the soft lymph of the glands in the lungs and air passages. The symptoms were slight fever, loss of appetite, cough, shortness of breath, and swollen feet, the patients finally becoming bedfast and showing great weakness, irregular pulse, sleeplessness, dry skin, usually constant fever, often bloody sputum, hemorrhage, sweating, and diarrhea until death claimed them.

Von Linné (1707–78) reported (*36*) from his travels in Dalarna in 1734 that the stone workers in Orsa, as a result of their health-destroying work, seldom attained a greater age than 20, 30, or 40 years.

In 1727 Wepfer (*36*) pointed out that there must be a connection between the phthisis from which the stone worker suffered and the dust particles which each breathed with the exercise of his activity.

In 1771 Bubbe (*36*) described the "Seeberger stone breakers' disease."

In 1780 Ackerman (1756–1801) referred (*37*) to miners' phthisis as the most "frequent and specific" mine workers' disease and said that physicians did not agree regarding the true definition of this disease.

As a result of a special inquiry into the prevalence of dust phthisis Allison (1790–1859), a professor of medicine at Edinburgh, found (*38*) that rarely did a mason, regularly employed in hewing stones in Edinburgh, live free from phthisical symptoms to the age of 50. According to Mavrogordato (*24*), he established the association between dust phthisis and tuberculosis, that is, between dust phthisis and true phthisis. Not dust per se but a secondary complication was responsible for the "ulceratio" of Celsus. The corrosive dust of sixteenth-century Agricola and eighteenth-century Ramazzini became the "tuberculosis" of Allison. Allison, like Laennec, knew of the anatomical tubercle but not of the tubercle bacillus.

Hugh Miller (*39*), the famous writer, geologist, and stonemason, described his own narrow escape:

> The dust of the stone which I had been hewing for the last 2 years had begun to affect my lungs, as they had been affected in the last autumn of my apprenticeship, but much more severely; and I was too palpably sinking in flesh and strength to render it safe for me to encounter the consequences of another season of hard work as a stonecutter. From the stage of the malady at which I had already arrived, poor workmen, unable to do what I did, throw themselves loose from their employment, and sink in 6 or 8 months into the grave—some at an earlier, some at a later period of life; but so general is the affection that few of our Edinburgh stonecutters pass their fortieth year unscathed, and not 1 out of every 50 of their number ever reaches his forty-fifth year.

In 1783 Hoffman (*40*) stated that metal fumes and vapors, as well as dust, caused the disease picture of "miners' phthisis," but Henkel (*41*) in 1745 attributed the disease to such external causes as dust from stones and metals, lack of air, bad air, or bad fumes or vapors.

Thackrah (1786–1833) wrote the first general work on occupational medicine in English (*11*), with the exception of the translations of Ramazzini's treatise. He (*42*) claimed that such generalization was not justified—that "dust of every kind irritates but not in an equal degree"—but recognized that masons inhaling particles of sand and

dust which arise from chipping stone were short-lived, generally dying before they attained the age of 40. He also discussed the prevalence of phthisis among the metal grinders of Sheffield and quoted Knight's opinion that fork grinding ought to be confined to criminals. A few years later Holland (*43*) portrayed the conditions of fork grinders—

> a picture of wretchedness which has no parallel in the annals of any country, or in the record of any trade. Fiction can add no color or touches to a picture like this. Truth transcends the gaudy embellishments of imagination. The distempered fancy has here no room to exercise her powers.

INCIDENCE OF DUST DISEASES

According to Greenburg (*44*), data on prevalence of occupational disease in different countries at different periods of time must be interpreted with the greatest caution, because industrial processes differ so widely and change so frequently. To be conclusive, such data should be available in the form of actual death rates, based upon knowledge of the population exposed as well as on the number of deaths occurring, and properly corrected for the age distributio of group involved.

A brief summary of some of the voluminous information available on the incidence of dust diseases in some of the principal mining countries of the world follows. For more detailed information, the reader is referred to Carozzi's Bibliographic Contribution to the History of the Pneumoconiosis "Silicosis" (*11*), published in 1941–42, and to Rosen's interesting and comprehensive History of Miners' Diseases (*45*), published in 1943.

GREAT BRITAIN

Statements published in England (*45*) before 1850 indicate that miners apparently suffered more with pulmonary complaints than other population groups and that the mean duration of life of miners was less than the average duration for the population as a whole. The Mining Journal (*45*) for 1858 contains the following report:

> At the age of 20, the total average sickness of coal miners exceeds that of other people by 46 percent; at the age of 30, it rises 70 percent; at 40, it is 78 percent; at 50, they have an excess of 76 percent; and at the age of 60 about 53 percent. Between the ages of 15 and 25, one-third of the deaths among coal miners are due to diseases of the respiratory organs, while one-third of the miners die a violent death.
>
> It is an undeniable fact that the average duration of life of miners is only 7.7 years, while agricultural workers attain an average age of 42.3 years.

In 1862 royal commissioners were appointed in England (*10*) to inquire into the health of men employed in metalliferous mines. Notwithstanding the evidence of several witnesses, particularly the miners themselves, that dust was far worse than anything else with which they had to contend, the commissioners concluded in a virtually unanimous medical opinion that the influence of dust was subsidiary to the many other adverse conditions of ventilation, exposure to fumes of explosives, and variations of temperature which at that time were prevalent in the mining industry. Collis (*10*) said that this conclusion was unfortunate, as the prevalence of phthisis in certain indus-

tries was attributed to imperfect hygienic conditions rather than to dust inhalation, a point then in dispute.

In 1902 a departmental committee (*10*), of which Haldane was a member, was appointed to reinvestigate the cause of the still persistently high phthisis mortality among Cornish tin miners. This committee decided that—

> So far as the Cornish miners are concerned it seems evident enough that stone dust which they inhale produces permanent injury of the lungs—gradually in the case of ordinary miners, and rapidly in the case of machine drillmen—and that this injury, while it is apparently capable of gradually producing by itself great impairment of the respiratory functions, and indirectly of the general health, also predisposes enormously to tuberculosis of the lungs, so that a large proportion of miners die from tubercular phthisis. That the primary injury to the lungs is due solely to inhalation of stone dust would seem to be practically certain.

From February 1, 1919, when the first silicosis scheme of compensation for the refractories industry became effective, until the end of 1928 compensation awards were made in England in 423 cases, including 121 deaths (*46*).

During the period 1921–23, 328 deaths were recorded in England and Wales under the generic title "chronic interstitial pneumonia", which included such diseases as fibroid phthisis, fibrosis of the lungs, silicosis, and miners' phthisis, when returned as nontuberculous (*47*). In 1926 the question was raised in Parliament (*48*) as to the number of men not affected by the Silicosis Act, but when examined by officers of the Home Office during the preceding 5 years had been found suffering from the disease. The reply was that the medical inspectors had made no routine examination of workers in factories except sample examinations in connection with special inquiries into the grinding and other industries. Of 1,106 workers employed in processes involving silica dust so examined, 556 were found to be affected by fibrosis of the lungs; 528 of these men were employed in the grinding industries and 28 at steel works.

A memorandum on industrial silicosis and asbestosis issued by the Home Office in July 1932 (*49*) contained the following statement:

> The incidence of silicosis continues to be serious and widespread, as is shown by the following figures of cases in which compensation has been paid under the special schemes made under section 47 of the Workmen's Compensation Act, 1925. During the last 3 years there have been 80 cases, including 30 deaths, amongst workmen employed in ganister mines and silica brick works; 179 cases, including 25 deaths, in the getting and manipulation of sandstone at quarries or on premises worked in conjunction therewith; 322 cases, including 87 deaths, in the pottery industry; 81 cases, including 32 deaths, in the metal industries, including metal grinding and sandblasting; and 91 cases, including 20 deaths, in coal mines.

The number of silicosis cases (fatal cases excluded) in Great Britain receiving compensation in different industries in 1933, 1935, and 1938 is given in table 1. The compensation in these cases was granted under section 47 of the Workmen's Compensation Act, 1925, as amended by the act of 1930 (*50*).

TABLE 1

Compensation scheme	Disablement cases receiving compensation; total, and new cases in each year [1]					
	Number			Percentage		
	1933	1935	1938	1933	1935	1938
(1)	(2)	(3)	(4)	(5)	(6)	(7)
Refractories Industries Scheme	270 (13)	274 (8)	262 (9)	21.7 (2.9)	15.8 (1.8)	10.4 (1.4)
Sandstone Industry Scheme	242 (76)	350 (75)	372 (69)	19.4 (17.0)	20.2 (16.6)	14.8 (10.7)
Metal-Grinding Industries and Various Industries Schemes						
China and earthenware industry	241 (99)	282 (69)	249 (47)	19.4 (22.1)	16.2 (15.3)	9.9 (7.3)
Metal industries	46 (16)	61 (17)	55 (14)	3.7 (3.6)	3.5 (3.8)	2.2 (2.2)
Coal-mining industry	229 (126)	545 (217)	1,279 (430)	18.4 (28.1)	31.4 (48.0)	50.8 (66.7)
Builders, etc	94 (45)	123 (28)	110 (21)	7.6 (10.0)	7.1 (6.2)	4.4 (3.3)
Miscellaneous [2]	123 (73)	101 (38)	189 (55)	9.9 (16.3)	5.8 (8.4)	7.5 (8.5)
Total	1,245 (448)	1,736 (452)	2,517 (645)	100.0 (100.0)	100.0 (100.0)	100.0 (100.0)

[1] The unbracketed numbers and percentages are the total cases of disablement (that is, new cases—cases continued from previous years) in which compensation was paid in the given year; the bracketed figures are the new cases only.

[2] Including metalliferous mines.

SOURCE: Home Office (1933, 1935, 1938)

Fatal cases are not included in table 1. However, in an analysis by Bridge (*50*), based upon data from death certificates supplied by the Registrar-General, and upon investigations of occupational history of deceased persons, carried out by the Factory Department of the Home Office, 41.2 percent of the deaths attributed to silicosis in England and Wales in 1938 were assigned to the coal-mining industry.

Since 1929, when compensation for silicosis first became payable to men employed in the coal-mining industry, the number of disablement cases among coal miners has increased rapidly until pulmonary disease in this industry has come to dominate the silicosis problem of Great Britain (*50*).

According to Stewart (*51*), in districts of Lancashire County, where the industry of coal mining predominates (40 percent of the occupied males being engaged in mining) the male death rate from pulmonary tuberculosis is higher than the corresponding rate for the whole administrative county.

Sladden (*52*) stated that, during the 4 years 1925–28, he had examined the bodies of 20 coal miners; 1 had advanced silicosis and 7 had slightly degrees of the condition (*52*). Since 1929, 63 examinations were made, 27 of which showed definite and serious degrees of silicosis and 16 others slighter degrees.

In a discussion of Prof. S. Lyle Cummins' paper, The Need for Dust-Prevention Measures in the Coal Industry, at a meeting of the South Wales Institute of Engineers, December 1931, the Colliery Guardian quoted Haldane (*52*) as follows:

Dr. J. S. Haldane (Oxford) said the opinions he had already expressed were largely in agreement with those brought forward by Prof. Lyle Cummins. The main new point that had emerged was that in a quite appreciable proportion of colliers who had never worked in stone, nor, in some cases, ever passed along stone-dusted roads, X-ray appearances indicative of scattered fibrosis and similar to the appearances in undoubted silicosis could be found after long service in an appreciable proportion of apparently healthy colliers, among whom no tendency to tubercular infection could be discovered. But when they came to the interpretation of the fact there was a divergence of opinion. The mortality among colliers from both phthisis and bronchitis, as well as from almost every other cause, had fallen steadily during the last 70 years, but so had the corresponding death rates among the general population, though not in the same ratio, till after about 1900. In the last decennial returns published there was still, however, a well-marked excess of deaths from bronchitis among colliers above about 55. He could not find, however, any justification in the official statistics for Dr. Tatham's statement that bronchitis had increased since about 1890. There had been a steady decrease all the time. * * *

In the anthracite districts of South Wales, for instance, the death rates were unusually high; and this suggested that there might be something specially irritating in anthracite dust. The recent X-ray investigations described by Prof. Lyle Cummins seemed to confirm this and also showed that it was not uncommon to find among elderly colliers of all sorts scattered fibrosis due to dust. He would like to know what the death rate from bronchitis was among the rest of the population in the anthracite area. Almost the only point in which he did not agree with Prof. Lyle Cummins was in extending the term "silicosis" to cover any form of fibrosis due to the inhalation of dust. This seemed inadvisable since there was no evidence that various forms of actual fibrosis were in fact due to inhalation of free silica and not to other forms of dust, such as coal dust or shale dust, which produced no tendency toward tubercular phthisis. It seemed preferable to distinguish these latter forms as "pneumoconiosis," since the word "silicosis" had come to have a very sinister significance.

The 1930 report of the Chief Inspector of Workshops and Factories (*53*) stated:

By a recent arrangement the Factory-Inspection Service receives copies of all death certificates in which death resulted from pulmonary disease involving fibrosis of the lungs. Of 700 such certificates received in 1930, 241 gave silicosis as the cause of death, and in the great majority of cases it was found that the previous occupation of the persons concerned was one in which there was recognized exposure to silica dust.

The industries furnishing the greatest number of such cases were the pottery trade, 52; sandstone, 49; coal mining, 39; sandblasting, 10; tin mining, 10; and other industries, 90. The report also said that recent investigations of the effects of exposure to asbestos dust had resulted in the adoption of measures to control dust in the textile branch of the asbestos industry. Data regarding 20 fatal cases of asbestosis without tuberculosis show a serious hazard in continued exposure to heavy concentrations of asbestos dust.

The frequency of silicosis in English porcelain plants was referred to in 1926 by Sutherland and Bryson, quoted by Bruce (*36*), who examined 568 porcelain workers, 250 with roentgen ray. Silicosis was determined among 87 workers employed in the slip and molding departments and in the kiln house.

Middleton reported, quoted by Bruce (*36*), in 1930 on 35 porcelain workers whose death was referred to silicosis. In 17 cases the occupational disease was complicated by tuberculosis. Three of the deceased had an exposure time of 20 to 29 years; 7, 30 to 39 years; 18, 40 to 49 years; and 7 had an exposure time of more than 50 years.

In 1931 Wood and Gloyne (*54*) were prompted by the recent discovery that asbestos workers are often the victims of occupational disease to determine whether pulmonary asbestosis also paves the way for a tuberculous invasion of the lungs or aggravates an existing infection. They studied 57 cases of pulmonary asbestosis, including 18 males and 39 females, 8 of whom were young girls from 18 to 21 years old; 12 of the 57 cases showed evidence of pulmonary tuberculosis, 4 of the 12 died, and on post mortem examination evidence of obsolescent tuberculosis was found in 2 and of active tuberculosis in 2.

Meriwether, quoted by Gloyne (*55*), found 4 active cases of tuberculosis and 33 inactive on examination of 374 asbestos operatives while at work. According to Sparks (*56*), 2,000 workers in England are exposed to asbestos dust.

An inquiry by Sutherland and Bryson (*57*) on the occurrence of silicosis among sandstone workers in England since February 1, 1929, under the Workmen's Compensation Act of 1925, disclosed that one of every four men at work appeared to have "mason's disease." As only men at work were examined, cases as far advanced as third stage and many in second stage, not being at work, were not included in the survey.

The outstanding feature of Ferguson's (*58*) investigation on the occurrence of silicosis among sandstone workers in Scotland and the North of England was the high degree of pulmonary morbidity existing throughout the industry. Of 1,000 working people examined, 433 showed clinical evidence of pulmonary fibrosis. Radiological examination of all the workers showing clinical evidence of fibrosis was impracticable but doubtless would have disclosed other cases of silicosis; most of the cases X-rayed by Ferguson were those that showed the severest fibrosis and those that gave the most reason for suspecting the presence of silicosis. One hundred and seventy-three of these workers showing clinical evidence of pulmonary fibrosis were X-rayed; 97 showed first-stage and 12 second-stage silicosis (advanced nodules coalescent).

The report of the Chief Inspector of Workshops and Factories in Great Britain for 1932 (*59*) considered particularly 42 deaths from asbestosis or asbestosis with tuberculosis and 281 deaths from silicosis or silicosis with tuberculosis distributed among the industries as follows: Pottery, 147; sandstone, 60; grinding materials, 30; sandblasting, 23; scouring powder, 5; miscellaneous, 16.

Under Parliamentary Intelligence, the Colliery Guardian (*60*) on August 4, 1933, published the following:

> In the House of Commons on Friday, on the motion for the summer adjournment, Mr. R. Davies introduced a discussion on the annual report of the Chief Inspector of Factories, in the course of which Mr. Tinker raised a number of questions relating to industrial diseases and the solvency of employers, as a remedy for which he demanded a system of compulsory insurance. Mr. R. T. Evans suggested that the bronchial diseases particularly affecting miners should be placed in a separate category and dealt with by the Mines Department under a new order. Mr. E. Williams complained that scores of men had died from silicosis in South Wales, whose dependents had not received a farthing of compensation and that there were also scores who were dying but were not receiving compensation after having worked for years in hard ground because the rock did not contain a certain amount of silica. He also urged that anthracosis should be scheduled as an industrial disease.

On February 16, 1934, the Colliery Guardian (*61*) published a statement from the Home Office that, since June 1, 1931, when the coal-mining industry first came within the scope of the general medical board under the silicosis-compensation scheme, 432 men in the industry had been certified by the board as disabled or having died from silicosis or from silicosis accompanied by tuberculosis and that 385 of these cases occurred in the South Wales coal field. Earlier comparable figures were not available.

On March 16, 1934, the Iron and Coal Trades Review (*62*) quoted the following statement by Prof. J. S. Haldane from the British Medical Journal:

> Cases of silicosis in the sense normally accepted end nearly always in death from phthisis, and if there were actually a large number of cases of real silicosis in this sense among South Wales colliers, their phthisis death rate would be considerably increased above the normal figure. There seems to be no sound reason for believing that many more cases of real silicosis occur among South Wales colliers than among other colliers, and I think the widespread alarm produced in the South Wales colliery district by the numerous certified cases of silicosis has no substantial basis so far as silicosis is concerned.

However, Harper (*63*), a radiologist practicing in the South Wales coal field, in a letter to the British Medical Journal published in May 1934, said that, when the full facts were known, there was little mystery in the many cases of silicosis in that part of South Wales. Deductions are made from the pay of the coal miners at the colliery offices in behalf of the medical men in the district; the workmen can therefore have roentgenological examinations if the family doctor is doubtful of their condition, and silicosis at present is the fashionable disease in the area. Harper (*63*) believed that more colliery workmen had been X-rayed in this area than in any other part of the coal field; hence, more cases of silicosis had been found than in other areas where no such examinations were made. This course partly explains why there were more known cases here. In fact, Harper said, there were more cases of silicosis than had been either examined or passed by the board because some refused to be examined, preferring to continue at work regardless of the cost to their health rather than be retired by compulsion with partial compensation of about £1 a week to meet the necessities of life and no further hope of employment. Kettle (*12*) said that nearly all the specimens sent to the board from this area were those of infective silicosis, and the infecting organism was most commonly the tubercle bacillus (*63*). Harper considered this statement much more reliable than any returns of the Registrar General. Therefore there would seem to be no difference between the silicosis in South Wales and the commonly accepted type, and he could find no roentgenological difference between cases of silicosis in South Wales and cases he had seen in the Belgian coal fields.

In March 1936, the Chief Medical Officer of the Silicosis Medical Board (*50*) informed the committee that the claims for compensation made by coal miners in South Wales were increasing in number. The total claims in 1933, 1934, and 1935 were 234, 316, and 398, respectively.

Cases and incidence of certified silicosis among coal miners underground in various parts of the British coal fields during the period 1931–37 are listed (*50*) in table 2.

TABLE 2

Coal field	Total number of new cases [1]	Average number of men employed underground [2]	Average annual incident-rate per 1,000 employed underground [3]
(1)	(2)	(3)	(4)
Anthracite mines of South Wales	749	22,033	5.23
Nonanthracite mines of South Wales	608	94,351	.99
Coal mines in remainder of Great Britain [4]	200	507,755	.06

[1] Total number of new cases of coal miners certified (under the various industries (silicosis) schemes, 1931 and 1934) during the period June 1931 to December 1937, to be disabled (totally or partly) or to have died from silicosis.

[2] Average number of men employed underground in December of each year from 1931 to 1937.

[3] Column (2) divided by column (3) and multiplied by $\frac{1,000}{6.5}$.

[4] All nonanthracite except for a few typical anthracite mines in Scotland.

SOURCE: Mines Department (1931–37, and unpublished records).

It will be seen that in 1931–37 the absolute number of new cases from South Wales as a whole was more than six times that from the remainder of Great Britain and that the number of cases from the anthracite mines of South Wales was greater than from its nonanthracite mines. When these figures are related to their respective populations, to give comparative incidence rates, the geographical differences are even more striking. The incident rate of certified silicosis in the anthracite mines is more than five times that in the other mines of South Wales and nearly 100 times the rate for Great Britain other than South Wales.

For 1940 the total number of new cases was as follows: South Wales anthracite mines, 267; South Wales nonanthracite mines, 176; remainder of Great Britain, 41. For the South Wales subdivisions the new cases were thus about twice the annual average for 1931–37, whereas for the remainder of Great Britain they were approximately the same (*50*).

According to Fisher (*64*), for the 3 years preceding 1943 there were 1,000 cases of dust disease annually in South Wales alone. In 1944, the first full year in which "pneumoconiosis, including the condition known as dust reticulation" was certifiable for compensation, the figures increased enormously. In 1945, in South Wales, 7,376 coal miners applied for submission to the medical board and for certification; 5,074 of these were certified; 513 were certified as totally disabled, and 4,561 were suspended as partly disabled. In 1946, 8,866 applications were received, 3,678 of which were certified. Of the 3,678 certified, 327 were totally disabled and 3,351 were suspended as partly disabled.

Fisher (*64*) stated that, although the incidence of pneumoconiosis is much less in the country outside of South Wales, it is nevertheless significant. In 1945, 595 cases were certified, and up to June 30, 1946, there were 316 cases. There were 13 pneumoconiosis cases certified in Nottinghamshire and 21 in Derbyshire during the period 1932–45, 65 percent of which were certified during the 3 years 1943–45.

The number of applicants for examination decreased in 1947 compared with 1946 (*65*).

From January to March 1946, 2,312 applications were received and 2,424 cases considered by the medical board, 1,230 of which were certified to be suffering from pneumoconiosis. The corresponding figures for 1947 were 1,520 applications received, 2,545 cases considered by the medical board, and 840 certified. According to a statement made in April 1947 (*65*) by the Minister of Fuel and Power, during the preceding 2 years 10,500 coal miners had been certified by the Silicosis Medical Board as suffering from pneumoconiosis and suspended from work in the industry.

In 1720 Josiah Wedgwood incorporated calcited flint with his earthenware body. In 1865 Elisa Meteyard (*11*) wrote, in her biography of Wedgwood, that at the beginning the flint was kept in cellars and used parsimoniously.

A history of the occurence of dust diseases in the North Staffordshire pottery industry in England was published in the Royal Sanitary Institute in October 1946 (*66*). According to this article, at first the calcined flints were broken by hand by men working secretly in cellars.

Before World War II the pottery industry in North Staffordshire employed 67,000 persons, 55 percent of whom were women and girls. Before the industrial revolution carried the stigma of the exploitation of child labor, in 1863 evidence was given before a commission to the effect that each successive generation of potters became more dwarfed and less robust than the preceding one.

In 1726 Thomas Benson was granted a patent for wet-grinding of flints (*66*). In his application for the patent, Benson stated that no person could survive dry grinding for more than 2 years. Although improvement took place slowly, dry grinding had not ceased by 1936. In 1928 the Various Industries (Silicosis) Scheme entitled pottery workers to compensation for total disability or death.

During the period 1931–41, 2,000 persons were given initial examination, 10 percent of whom were rejected on medical grounds. In the same period 4,000 workers were examined periodically while at work, and 490 were found to be suffering from silicosis or silicosis accompanied by tuberculosis. During the period 1929–44 the Department of the Chief Inspector of Factories recorded an average of 47 deaths annually from these diseases—402 cases of silicosis and 347 cases of silicosis with tuberculosis for the 16-year period. Great improvement has followed in recent years by the substitution of alumina for flint and other siliceous materials.

GERMANY

Industrial pathology in Germany was developed at the beginning of the nineteenth century, simultaneously with the upward trend of German industry (*7*). In 1781 Tissot (*67*) graphically illustrated the health hazards encountered by stonemasons; in one place where the people formerly had been engaged in felling trees and in carving they were the "handsomest, strongest, and healthiest of persons" but for 25 years—since the inhabitants had turned to stonecutting—there had been in that region the most lingering diseases. In 1804 and 1812

Goetzinger (*68*), in describing the mountainous part of Saxony, said that the fine dust and drink brought the stonecutter to an early death.

Schürmann (*7*) stated in 1929 that only a few works had been published on the harmful effects of iron and steel dust to which the metal polisher was exposed, in spite of the fact that the works of Paracelsus, Ramazzini, and others were known in Germany, Ramazzini's work being the sole textbook in the field of occupational disease used in Germany up to the middle of the nineteenth century. In contrast to the English reports on the health picture of the Sheffield polishers, Hauer (*69*) in 1832 reported conditions among German polishers as not unfavorable to health. Nasse (*70*) was the first to report polishers' disease; he wrote that grinding workers lasted only 12 years and recommended respiratory protection for them. The first known German publication on the harmful effects of dust on polishers was issued by Pappenheim (*71*) in 1860. In 1866 Zenker (*72*) identified iron oxide in a lung and termed the disease caused thereby "siderosis." He designated the diseases of the lungs caused by dust as "pneumoconiosis." In a book on the care of health and medical statistics in Prussian mines, published in 1881, Schlockow (*73*) stated that, of 942 miners examined by Seltmann, 37.7 percent had emphysema of the lungs.

Only 5 roentgenologically normal pictures were disclosed among 25 sandblasters examined by Lochtkemper (*74*) in 1930; the remaining 20 (80 percent) revealed various stages of silicosis. In 1937 Bergerhoff (*75*) reported finding lung tuberculosis in 24 of 175 sandblasters examined clinically and roentgenologically. Thiele (*76*) found that 28 percent of the porcelain workers examined by him were affected by pneumoconiosis; Holzmann and Harms determined roentgenologically the presence of pneumoconiosis in 75.6 percent of 41 selected porcelain workers; and Koelsch found 58 percent of 19 workers affected. As given by Koelsch (*77*), the tuberculosis mortality rate for porcelain workers in various Bavarian districts was 23.7 to 96.2, whereas that for the general population was 24 to 29. Vollrath found a tuberculosis mortality rate of 24 among porcelain workers compared with 16 for the general local population in Rudolstadt and 57 compared with 20 in Meiningen. However, Böhme stated that only about half of the workers in porcelain factories are exposed to a severe dust hazard. Hofbauer-Flatzeck (*77*) found a tuberculosis mortality rate of 26.5 in the porcelain industrial town of Selb; 61 percent of those dying of tuberculosis had silicosis also. If this 61 percent is subtracted, the mortality rate is scarcely higher than the average of 9 for the Empire; therefore, Hofbauer-Flatzeck attributed the higher mortality entirely to injury through silicosis. Böhme (*78*) determined pneumoconiosis in 28 percent of 184 coal cutters who had worked more than 10 years underground and in 66 percent of hard-rock miners; in another investigation he determined by X-ray examination that 36.2 percent of 69 hard-rock miners had pneumoconiosis and by clinical examination, 17.3 percent. Komissaruk (*79*) examined clinically and roentgenologically 40 foundry workers who had worked in dusty industries for more than 10 years; 4 definitely had pneumoconiosis, 5 were doubtful, and 3 were borderline cases. In 23 cases

the X-ray revealed old inactive pulmonary tuberculosis, but the sputum contained no tubercle bacilli.

In 1932 Lochtkemper and Teleky (*80*) published the results of an extensive study on dust which covered working conditions and health of workers in many different dusty trades. The number of workers examined in each group, however, was usually too small for definite conclusions. Examination of 22 women employed in a scouring-powder factory in 1930 revealed that all but 3 were affected by silicosis in varying degree, 6 being in stage 3. Examination of 7 workers in a sand and cement plant disclosed 1 case of silicosis in the first stage and 2 in the third; the others showed no indication of silicosis or only slight pneumoconiosis, which the authors classified as "silicosis 0–I." Inquiry among workers and physicians of the region revealed that 4 had died and 2 still had tuberculosis. Six of seven shell-limestone cutters examined were diagnosed as having pneumoconiosis 0–I and 1 silicosis 0–I. Of 18 graywacke workers examined, 3 were diagnosed as negative, 4 as having silicosis 0–I, 4 silicosis I, 3 silicosis II, 1 silicosis I–II, 1 silicosis III, and 2 silicosis II with tuberculosis. From an examination of the statistics of a large sick benefit association Koelsch and Kaestle (*81*) found the illness frequency, especially for tuberculosis and diseases of the respiratory organs, less for the quarry and shell-limestone workers than for the other members of the association, but the mortality rates apparently were greater for stoneworkers (0.35 percent) than for the others (0.21 percent). These investigators examined 82 persons who had worked in shell limestone only; 14 had more or less definite signs of dust lung, but it differed in appearance from the silicosis of the sandstone worker. They concluded from these examinations that under certain conditions limestone also can cause changes in the lungs within the meaning of dust lung if it can have a correspondingly long action. Dust lung was more frequent and more pronounced among those who had worked in limestone and sandstone, the more so the closer the work in sandstone had followed upon that in shell limestone.

According to Landau (*82*), the cleaning of metallic castings presents great health hazards to the men engaged in the work. Of those examined, 69 percent had pneumoconiosis and 8 percent tuberculosis. Work with steel seemed to be much more dangerous than with cast iron, probably because compressed-air tools were used in cleaning the steel castings, which were cast in a mold of sand very rich in silica.

From 1929 to 1934, in the Ruhr, 332 workers died of silicosis; this figure was reported (*83*) not from the total number of workers but only from those working in rock, about 10,000 to 12,000.

In 1937 Holmann (*84*) published a report of extensive investigations of a large number of workers who had worked for many years with the purest carbon dust in the form of carbon black, petroleum coke, tar coke, and artificial graphite without any admixture of rock dust, especially quartz. Among these workers he found a large number of dust lungs of stages I to III.

According to Hollman (*84*) the dust lungs of persons exposed only to pure carbon dust could not be differentiated roentgenologically from silicosis produced by sandblasting. Of 90 persons with severe pure carbon-dust exposure, 23 were diagnosed as being in stage I, 10

in stage II, and 3 in stage III. Of 202 workers exposed to pure carbon dust with slight admixture of carborundum, 13 had stage I dust lung.

In 1939 Maaszen and Büttner (*85*) reported that severe silicosis as an occupational disease cost the people infinite values in health and money—costing more than all other occupational diseases together. In 1937 these authors examined 508 workers in the largest ceramic- and silica-brick factory in Germany. This factory, which had been in existence for several decades, was located in a small village where the employees had their homes. There was therefore little turn-over, and most of the workers had been employed for years. It was possible to study all stages of silicosis and get a fundamental picture of the dust hazard in the silica-brick industry.

Of the 508 employees examined, only 380 were actually exposed to a dust hazard; a total of 17.31 percent of the 508 were affected by various stages of silicosis, while the percentage for the 380 actually exposed to a dust hazard was 23.15.

AFRICA

SOUTH AFRICA

Mining was begun in South Africa on the Witwatersrand in 1886 (*86*). At first the workings were shallow and in the oxidized or "free-milling" zone where the rock was relatively soft, friable, and damp. By 1892 extensive deep-level properties had been operated, and drilling by machines was introduced. As long as the mines were working in the free-milling zone very little dust was produced, as the mines were shallow and probably fairly well ventilated, and it is unlikely that the men were greatly affected by the dust. Very few people in South Africa suspected that mine dust was in any way injurious to health until the Government mining engineer of the Transvaal Mines Department in a report in 1902 mentioned "miners' phthisis" as a disease "which seems to be peculiar to men employed in rock-drill work." This report, which covered the 6 months ended December 1901, stated that, of 1,377 machinemen employed before 1899, 225 were known to have died between October 1899 and January 1902, an average annual death rate of 73 per 1,000. These facts arrested the attention of the Government, the mining community, and the general public, and in December 1902 the first (Transvaal) Miners' Phthisis Commission was appointed "to inquire into and report on the disease commonly known as miners' phthisis." A report was issued in 1903. The commissioners attempted to procure a medical examination of all working miners, but the returns were incomplete partly because many of the miners were reluctant to submit themselves to examination. Of the 1,201 miners examined, 15.4 percent were affected by miners' phthisis, and 7.3 percent were suspected. The Report of the Medical Commission issued in February 1912 was the second landmark in the medical history of silicosis in South Africa. The actual examinations were more complete than in 1903 but did not cover all the underground employees. A general clinical examination was made of 3,136 working miners, supplemented by a special examination of 326 men, in which radiography was, for the first time, applied to examination of cases on a fairly extensive scale. The prevalence of the disease among the

working miners examined was 26 percent, with an additional 5.5 percent of doubtful cases. This figure is somewhat higher than that found in 1903 but probably reflects the result of a more extensive investigation.

According to Irvine, Mavrogordato, and Pirow (*87*), 1916 was a cardinal year in the history of silicosis on the Rand. It was marked by institution of the Miners' Phthisis Medical Bureau and publication of the General Report of the Miners' Phthisis Prevention Committee. The present-day system of detection and prevention of silicosis on the Rand dates from 1916. In all, 6,472 original compensation awards were made to miners for silicosis from 1912 to 1916.

According to the Report Upon the Work of the Miners' Phthisis Medical Bureau (*88*) for the 3 years ended July 31, 1932, the average number of new cases of silicosis and tuberculosis with silicosis among the miners of the Witwatersrand was over 800 annually during the 4-year period 1912–16. The report also states that 895 cases of "primary-stage" silicosis were detected from 1917 to 1920; 728 cases of "anteprimary stage" from 1920 to 1923; 1,235 cases of "anteprimary stage" from 1923 to 1926; and 917 cases of "anteprimary stage" from 1926 to 1929. In 1930–31 the number of new cases, for the first time in 8 years, fell below the standard level of 251 cases, the annual average for 1920–23.

The following statement by Hildick-Smith (*89*) summarized briefly the status of miners' phthisis in South Agrica in 1934. After quoting figures showing the percentage production rate of silicosis for the preceding 15 years he said:

> From a study of these figures I personally am inclined to think that the time is not far distant when the incidence of silicosis will have ceased to be a matter of serious concern to the industry. This, I think will be accomplished by the elimination, in whole or in part, of the dangerous dust from the underground air. Old habits of thought were inclined to persist in regard to the production of silicosis in the mines. The Report Upon the Work of the Miners' Phthisis Medical Bureau showed the maximum percentage production rate for silicosis had decreased from 14.5 percent after 14 years' service in the period 1918–20 to 4.5 percent after 17 years' service in 1931–32. The graduation rates for all miners, including men working underground before the inception of the Phthisis Bureau in 1916, show that the present maximum production rate is under 4 percent after 20 years' service. Of the 157 miners notified last year that they contracted silicosis more than one-half began work in 1916–17, 1 in 1922–23, and 1 in the following year. Since 1923 there have been no cases of contracted silicosis—in other words, no new Rand miner has been found suffering from silicosis in the last 10 years. These results are confirmed by the fact that less than 50 percent of the patients at the Springkell Sanitorium are of the miner class

With regard to the employment of silicotics, Hildick-Smith said that in 1934, 978 were employed by the mines, an increase of about 110 since November 15, 1933.

The average number of miners examined annually during the period 1938–41 by the Miners' Phthisis Medical Bureau of the Union of South Africa (*90*) was 30,635, the average annual number of new cases of silicosis was 260, and the production rate per thousand was 8.50. For the period 1941–44, the average annual number of miners examined was 27,558, the average annual number of new cases of silicosis for the same period was 261, and the average annual incidence rate per thousand was 9.47 (*90*).

During the triennial period 1938–41 the annual average number of "new Rand miners" with 19 to 24 years service was 562, and they produced during that period 71 new cases; during 1941–44 the annual average of new Rand miners with 19 to 24 years service was 787, and they produced 99 new cases.

The number of new cases of silicosis among new Rand miners has now become significant and must become more so as larger numbers of them pass into the long service periods.

The position as regards the general incidence of silicosis remains on the whole satisfactory, but the gradual improvement culminating in the low rates for the triennial period 1935–38 seems to have been checked in 1938, since when a slight rise in the rates has become apparent.

WEST AFRICA

An investigation was made in 1940 (*91*) of the coal mines of the Tarkwa area of West Africa. Of 560 men examined, 21 had definite silicosis, 10 without and 11 with accompanying active tuberculosis. As the incidence of pulmonary tuberculosis was higher than that of silicosis, the authors regard tuberculosis as the greater menace to the health of the mine employees. At that time, nothing was being done for native miners who were suffering from either of these diseases.

In 1946 the same authors (*92*) reported a series of 1,002 consecutive examinations of underground employees, including the 560 referred to above. The mines concerned are in the banket deposits of Tarkwa, the Akropong-Kononge area, the Prestea coal field, and the Ashanti banket area. Of the total of 1,002 miners examined, 155 had abnormal increase in striation, 46 had definite silicosis, 19 had silicosis with tuberculosis, and 115 had tuberculosis alone.

UNITED STATES

Data on the general incidence of silicosis in the United States are not available. Such data are difficult to obtain in the existing state of American vital statistics; exact knowledge of the population at risk in a given occupation, classified by age, is obtainable only with great difficulty and by special and intensive research (*44*). In an analysis of statistical data of this kind, Greenburg (*44*) called attention to the importance of considering groups that are fairly comparable, so that the effect of industrial hazards will not be complicated by the influence of social and economic factors of a more general nature. He stated, for example, that in Hoffman's studies, published in 1918, ratios presented for the various dusty trades were based on the industrial experience of the Prudential Insurance Co. but that the ratio used as a norm for comparison of males in the registration area was obtained from data of the United States Census Bureau. On this basis, almost all the industries that he tabulated showed a surprisingly high tuberculosis ratio, including many trade designations of workers, such as "iron and steel workers," who could hardly be considered as generally exposed to a serious dust hazard; his abnormally high ratios, therefore, probably were due to the general social and economic conditions of the wage earner's life and to the

fact that the group was an industrial one. It seemed evident to Greenburg (*44*) that a comparison between the Prudential figures for a given dusty trade and the Census figures for all occupied males, which gave Prudential ratios 25 to 50 percent higher for tuberculosis than those for the registration area, was not a fair one and that the conclusion reached through such a comparison was unwarranted.

Several special investigations, carried out in certain industries in the United States that have attracted attention because of the high death rate from tuberculosis (*93*), are described below.

MINING

The first investigation of silicosis in the mining industry in the United States was made in 1914–15 by the Federal Bureau of Mines in cooperation with the United States Public Health Service in the Joplin (Mo.) mining district (*94*). Of 93 men examined, 64 showed plain and definite evidence of pulmonary disease, 3 were suffering from nonpulmonary disease, and 26 were seemingly well. To gain a more accurate idea of the prevalence of consumption among the miners of the Joplin district it was decided to examine a larger number, and from May 15 to December 31, 1915, 720 miners were examined: 45.7 percent had silicosis and tuberculosis and 5.3 percent had tuberculosis (*95*). A later report (1927) of this same investigation by Lanza and Childs (*96*) included the results of the first detailed X-ray studies of silicosis made in the United States.

Investigations of mining conditions by Harrington and Lanza (*97*) in Butte, Mont., in 1921 revealed that 42.4 percent of 1,018 miners examined showed definite signs of lung damage due to dust. An examination in 1921 of 303 gold miners in Nevada (*98*) disclosed that 81 percent had silicosis.

In 1923 the mining companies of the Tri-State district (comprising the zinc- and lead-mining areas of southwestern Missouri, southeastern Kansas, and northeastern Oklahoma) asked the Federal Bureau of Mines to determine whether measures in use were adequate to prevent silicosis and, if not, to recommend improvements. The 1923 investigation (*99*) included the examination of 309 miners, 101 of whom were found to be negative, 114 doubtful, and 94 positive cases of silicosis. Of the positive groups, 52 were in the first stage, 22 in the second stage, and 20 in the third stage of silicosis.

From July 1, 1927, to June 30, 1932, the Metropolitan Life Insurance Co., the Tri-State Zinc & Lead Ore Producers Association, and the Federal Bureau of Mines operated a cooperative clinic at Picher, Okla. (*100*), to demonstrate to industry a workable method for diagnosing silicosis, to educate workers in preventive measures, and to serve as a model to industries presenting a dust hazard. Of 27,553 individuals examined during this period, 5,366 had silicosis, 742 silicosis plus tuberculosis, and 320 uncomplicated tuberculosis.

According to the United States Public Health Service (*101*), the percentage of anthracite miners and their helpers showing signs of pneumoconiosis increased from 2 percent among those with less than 5 years' service to 16 percent among those with more than 15 years' service (for persons under 45 years of age). Of 95 X-rays of bitumi-

nous-coal miners 40 (42 percent) showed generalized fibrosis, chiefly linear in character. The percentage of deaths caused by all respiratory diseases among anthracite miners in Pennsylvania was definitely higher than that of other adult males in the general population—57.6 percent in contrast with 37.2 percent among other men of the same age in the Wilkes-Barre coal field (*102*).

The following summary (*103*, *104*) by the United States Public Health Service indicates the prevalence of dust diseases in anthracite mines:

1. A study of health conditions, including the physical examination of 2,711 men (about 96 percent of the number on the pay roll) was made in three representative anthracite-coal mines in Pennsylvania.

2. The men examined were divided into occupational groups, largely in accordance with the proportion of free silica found in the dust to which they were exposed.

3. No cases of anthracosilicosis (miners' asthma) were found in a control group composed of hard-coal-mining employees whose dust exposure averaged less than 5 million particles per square foot of air.

4. The prevalence of anthracosilicosis among the entire group of employees was found to be about 23 percent.

5. Among all except rockworkers, less than 2 percent of the men developed anthracosilicosis, when the duration of employment was less than 15 years, regardless of the amount of dust in the air.

6. 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 to 199 million particles per cubic foot, 20 percent of those exposed to 200 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 anthracosilicosis.

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

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

9. The prevalence of anthracosilicosis among rockworkers who had been exposed 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 rockworkers who had been employed for more than 25 years in dust concentrations exceeding 300 million particles per cubic foot.

10. Age per se appeared to play a minor role in the development of anthracosilicosis.

11. Analysis of the data for the purpose of determining safe limits of dust exposure indicated that employment in an atmosphere containing less than 50 million dust particles per cubic foot would produce a negligible number of cases of anthracosilicosis 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 to 15 million particles per cubic foot. The limit of toleration for rockworkers was set tentatively at 5 to 10 million dust particles per cubic foot of air.

12. 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 rockworkers of more than 15 years' service.

13. 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.

14. The highest prevalence of clinical tuberculosis occurred among the rockworkers. After 20 years' service 37 percent of these workers presented evidence of pulmonary tuberculosis.

15. Pulmonary infection (tuberculosis and nontuberculosis) was found among 58 percent of the men having early anthracosilicosis and in 92 percent of those in the more advanced stages.

16. Clinical tuberculosis was diagnosed in 15 percent of those with early anthracosilicosis and in 43 percent of those in the more advanced stages.

17. In the control group 1.7 percent were found with moderate or marked physical impairment causing decreased capacity for work as compared with 9.9 percent among the regular miners and with 12.6 percent among the rockworkers. With the exception of the rockworkers, no group showed moderate or marked 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 in group A who had worked from 10 to 20 years in atmospheres containing more than 100 million dust particles per cubic foot.

18. 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.

19. 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.

20. The term "anthracosilicosis" is used in this report as a descriptive title for the form of pneumoconiosis commonly called miners' asthma.

21. 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.

Kibbey (*105*) reported in 1931 that virtually 100 percent of the miners who worked in the several coal mines operated by his company in Alabama had anthracosis and that they had a higher mortality rate from tuberculosis than any other wage-earning group.

From a study of industrial morbidity statistics for the period 1924–27 Bloomfield (*106*) found that 38 cases of pneumonia had developed among the 1,637 bituminous-coal miners employed during the same period in the mines operated in connection with an iron and steel plant. Occupational analysis disclosed that 33 of the 38 pneumonia cases were associated with only 2 of the 69 different occupations in the mines—pick mining and loading coal. The pneumonia rate per 1,000 for miners and loaders was 31, whereas the rate for all other mine workers was only 8.5 per 1,000.

In an investigation carried out in 1938 and 1939 in Utah, the United States Public Health Service (*107*) found the incidence of anthracosilicosis among 316 underground bituminous-coal miners to be 4.6 percent; among 727 nonferrous metal miners, 9.1 percent had silicosis; and among 1,391 smelter workers, 2.7 percent had the disease.

Pulmonary tuberculosis was found in 6.2 percent of the coal miners who had silicosis; in 13.6 percent of the metal miners who had silicosis; and in 16.2 percent of the smelter workers who had silicosis. This is in contrast to the 2.1 percent, 1.0 percent, and 4.9 percent of the nonsilicotic workers in these respective groups. The incidence of tuberculosis among the workers with no silicosis in the coal and metal mines was about that of the general population, whereas the incidence of

tuberculosis among the nonsilicotic smelter workers was about twice as high as among the general population.

Jones (*108*) discovered 86 cases of silicosis during 4 years of medical practice among coal miners in West Virginia. These cases were determined by careful fluoroscopic and roentgenographic study of the chest and routine sputum and blood analyses. These cases included only those in which exposure to dust had been in bituminous-coal mines alone.

The apparent similarity of the risk in mining and the risk in the processes involved in excavating and tunneling, so far as the silicosis hazard is concerned, led to an investigation of such processes in New York City by Smith and Fehnel in 1929 (*93*). Of the 208 drillers, blasters, and excavators examined 42 percent showed early and 15 percent showed well-developed silicosis. Evidence of tuberculosis, including both active and inactive cases, was found in 9 percent of the total number.

Although rock drilling where injurious amounts of dust are produced may require only a brief period on many contract jobs, the importance of dust-control measures is indicated by the following statement in the Connecticut Health Bulletin (*109*):

> It must be remembered that drilling is an occupation calling for experienced men and that the duration of exposure of the drillers is not limited to any one job but is continued over a period of years as the driller goes from job to job. It is accordingly fallacious to state that, because the driller's exposure in any one location is of a brief duration, adequate protection need not be provided inasmuch as it requires a considerable period of time for silicosis to develop.

In 1942 Porro, Patton, and Hobbs reported (*110*) 15 cases with 5 postmortems of pneumoconiosis in the talc industry in New York State. They attributed disability or death directly to pneumoconiosis in 13 cases; the pneumoconiosis was contributory to death in one case and incidental to death in another. The conclusion was that pneumoconiosis causing disability and death occurs in talc workers, the disabling tissue changes being due principally to talc itself.

Greenburg (*111*) reported in 1947 a study of the tremolite talc mining and milling industry in northern New York State. In the initial roentgen-ray survey in 1940 advanced fibrosis was found in 32 of the 221 men examined, a rate of 14.5 percent.

In comparison of talc workers with carpet workers, Greenburg found that fibrosis was present in only 2.5 percent of all carpet workers and in 4.9 percent of those 50 years of age or older; it was present in 51.5 percent of talc workers in this age group.

The fibrosis found in this study was of a fine, diffuse type, with a roentgenographic appearance of granulation or nodulation in a hazy background. It tended to be disabling and was frequently accompanied by dyspnea, cough, and fatigue. There was also some evidence that this fibrosis was characterized by increased susceptibility to tuberculosis, as clinically significant tuberculosis was diagnosed by roentgen examination in 8 of 18 cases of tremolite-talc fibrosis where there had been no other dust exposure.

Analysis by the New York State Division of Industrial Hygiene of six samples from two talc mines showed 1 percent or less of free silica. Gardner (*111*), however, found amounts of quartz ranging from 12 to

20 percent in mineral samples from two mines in the same area. Greenburg (*111*) stated that it was difficult to explain this marked difference in findings, except on the supposition that Gardner's samples included some of the quartz-bearing stratum overlying the talc deposit proper.

GRINDING INDUSTRY

According to Hoffman (*4*), the industrial insurance mortality statistics of the Prudential Insurance Co. from 1897 to 1914 showed that 143 (46.9 percent) of 305 deaths among grinders were due to pulmonary tuberculosis. In another group of 5,988 grinders, which included cutlers, scissors grinders, and ax, plow, and other steel grinders, but excluded foremen and superintendents, the actual mortality from all causes was 17 percent over the expected mortality; in other words, for every 100 deaths expected on the basis of normal experience there were 117 deaths in this group.

In 1920 Winslow and Greenburg (*112*) investigated the dust hazard in an ax factory. They quoted Drury's exhaustive statistical study on the incidence of tuberculosis among grinders and polishers as showing that, from 1900 to 1918, the death rate in a group including 90 polishers, 85 wet grinders, and about 25 dry grinders was 1,000 percent. Their investigation revealed dust conditions serious enough to cause such an excessive death rate.

To indicate the effect of silica as a predisposing cause of tuberculosis Riddell (*113*), in a paper published in 1926, quoted the following mortality table prepared by Drury for a Connecticut community (agricultural except for an ax factory employing 800 men):

Death rate from tuberculosis in a Connecticut community

	Per 100,000
Entire population of factory district	200
State as a whole	150
Factory population	650
Polishers and grinders	1,950

In a review of pulmonary tuberculosis developed in a large grinding-wheel plant, Clark (*114*) found that from January 1, 1918, to December 31, 1939, there were 42 cases of active pulmonary tuberculosis, 38 of whom were men and 4 women; 37 of the group were employed at factory work of some kind, while 5 did clerical work. Of the 42 workers who developed tuberculosis 20 were dead at the time Clark prepared his report, 18 were living, and 4 could not be traced. Of the 18 living, 13 were working, 3 were at home under care of a physician, and 2 were in hospitals. The average number of employees during the 13-year period was 2,460.

According to Kessler (*115*), over 100 cases of silicosis were alleged to have occurred in the abrasive-powder industry in one State, involving four companies which pumped sand by hydraulic pressure to a plant, where it was washed, steam-dried, screened into various sizes, and then pulverized in closed tube mills. Six of the workers had died and been autopsied, and many others were reported to have died, but the results of the autopsies were not known. Clark (*116*) observed for 30 years employees who had been exposed to the inhalation of artificial abrasive dusts in amounts very much greater than in the use of

grinding wheels. Several studies supported by continuous observation, examinations, and X-rays failed to disclose any definite health hazard to healthy workers. As a further check, Clark (*116*) reviewed all those (96 in number) who had been exposed for 25 years or more; 87 of these were in good health and working, and 9 were pensioned but in good health. None of these men seemed to have been affected by their many years of exposure to the constituents of artificial-abrasive grinding wheels.

To check the generally accepted beliefs concerning the harmfulness of exposure to artificial abrasive dusts, Clark (*116*) wrote to the physicians heading the medical departments of 22 companies, which were the largest users of grinding wheels in the United States, for their experience with pneumoconiosis caused by the dust of grinding wheels. Among the 18 replies received, none reported pneumoconiosis as a problem, and the incidence of tuberculosis was low.

Clark (*116*) concluded that artificial abrasives belong to the nontoxic mineral dusts mentioned in the following quotation from the Journal of the American Medical Association (*117*):

> Only silica (SiO_2) is capable of inducing silicosis but any other mineral dust under conditions of prolonged and gross exposure may cause some increase in pulmonary fibrosis. If the relative potential harm of silica is rated as 100, these other nontoxic mineral dusts may be rated only on an order of 5 or 10. Moreover, the fibrosis itself is not pathognomic, since many other dusts, alkalies, acids, or vapors may induce somewhat similar, if not identical X-ray markings.

GRANITE INDUSTRY

In his report of dust phthisis in the granite-stone industry, published in 1922, Hoffman (*118*) summarized the results of his investigation as follows:

> The granite-stone industry is carried on by wage earners who, broadly speaking, live under sanitary conditions above the average, so that possibly unfavorable environmental factors are of decidedly secondary importance.
>
> The housing conditions under which granite workers live are also above the average, so that in this respect the environmental factors are favorable to a low mortality rather than otherwise.
>
> Anthropometric records clearly establish the fact of a superior physique, indicative of a higher degree of disease resistance, as determined by a relative weight above the average. From this point of view, therefore, granite workers should experience a relatively low mortality from pulmonary tuberculosis instead of a mortality decidedly above the average normal to industrial occupations.
>
> Granite workers, considered by specific occupations, show wide variations in tuberculosis frequency, the excess in the death rate being most marked among the men employed in granite-stone cutting, it being especially severe among men employed in the use of pneumatic tools. Certain occupations, such as polishing, tool sharpening, bed setting, etc., do not show a marked excess, if any, in the mortality from pulmonary tuberculosis, clearly indicating that the risk is practically proportionate to dust exposure.
>
> Compared with the normal death rate of adult males of the State of Vermont, or of New England, the mortality from pulmonary tuberculosis among granite-stone workers has increased enormously during the last 2 years, as contrasted with a diminishing mortality in the population at large. Against a decrease in the pulmonary tuberculosis death rate of adult males of the State of Massachusetts from 288.5 per 100,000 exposed to risk during 1895–99 to 203.2 during 1915–18, there had been an increase in the corresponding death rate of granite cutters of the New England States from 432.0 per 100,000 during 1895–99 to 1,056.7 during 1915–18. The only other occupation for which information is available for the corresponding period of time is that of glass-bottle blowers,

among whom the mortality from pulmonary tuberculosis diminished from 418.6 per 100,000 to 265.9. These statistics for the New England States are confirmed by similar data for every other stonecutting center of the United States, proving with absolute certainty that in every section of the country the tuberculosis mortality of this group of industrial workers is increasing, in contrast to a locally diminishing death rate from this most fatal of all diseases * * *.

Recalling that the normal pulmonary tuberculosis mortality of adult males in Massachusetts is only 203.2 per 100,000, it is shown that the present death rate from pulmonary tuberculosis among granite cutters is 5 times the normal experience in the population at large and probably 6 times what it should be on the basis of strictly noninjurious occupations carried on largely under hygienic conditions and in the open air.

The same conclusion applies to nontuberculosis respiratory diseases, for it is shown that the mortality from bronchitis, pneumonia, and asthma is also on the increase among granite cutters, in contrast to a diminishing rate of frequency among adult males of the general population.

In the second series of investigations on health of workers in the dusty trades, published in 1929, the United States Public Health Service studied the granite industry at Barre, Vt. (*119*). The occupational groups were divided into four general classes based on the amount of dust to which they were exposed:

(*A*) Hand pneumatic-tool cutters, 614 persons, exposed to an average of approximately 60 million particles.

(*B*) All other occupational groups exposed to more than average plant dustiness. This group contained 104 persons in occupations where the dustiness averaged between 27 and 44 million particles.

(*C*) Those occupational groups consisting of 146 persons exposed to average plant dustiness (20 million particles).

(*D*) Those occupational groups exposed to less than average plant dustiness. This group contained 108 persons in occupations where the dustiness averaged between 3 and 9 million particles.

The results of the study on prevalence of silicosis and tuberculosis among these workers are summarized in part as follows:

Having a clear-cut diagnosis of silicosis and complete information with regard to the magnitude of dust exposure, it was possible to study the proportion of persons developing silicosis by length of service. In the A group, or that group in which there was heaviest dust exposure, the first case of silicosis appeared after approximately 2 years of service, and by 4 years of service all in this group seemed to have developed at least an early case of silicosis. In the same dust-count group the first case of more-developed silicosis appeared after 5 years of service, and by 9 years approximately 90 percent had advanced to this stage. The study of the other dust-count groups showed that the development of silicosis was proportionate to the dust exposure. In the case of the *D* group 2 cases of early silicosis occurred after 10 years' exposure, and 1 case of moderately developed silicosis after 6 years' exposure.

General prevalence of tuberculosis complicating silicosis.—In plants carrying morbidity records, where it was believed certain that all cases of tuberculosis were diagnosed as such, the total rate was 5.7 percent or 6.5 percent if early cases are included. Latent or suspected cases of tuberculosis (among those given physical examinations) gave an additional 3.8 percent. These figures represent the number of cases found over a period of 2¼ years, and consequently the rate is different from what would be obtained on an ordinary cross-section survey.

Tuberculosis by dust groups and years in granite.—The association between the high tuberculosis prevalence among granite workers and the dust hazard was shown by determining the rates for that disease in the different dust-count groups by length of service in the granite industry. The comparison between *A* and *B* groups and *C* and *D* groups was extremely significant. No cases were found in the *D* group, and *C* group furnished but 3 cases, 1 of which occurred under 5 years' exposure; another, under 10 years. Of particular importance is the fact that in 35 years of exposure the rate did not rise. Rates for the

A and *B* groups rise steadily with increase of length of service to 15.5 percent in group *A* and 19.1 percent in group *B*. Even when the suspected tuberculosis cases are added, the general picture is not materially changed. In neither the *C* or *D* groups does there occur any excess of cases after long exposure, while in the *A* and *B* groups the rate rises with length of service in a similar manner as for active tuberculosis.

According to Goodrich (*120*), statistics show that the death rate from tuberculosis among workers exposed to silica dust and, to a smaller extent, other dusts is excessive. The introduction of pneumatic and electric tools in processing stone has caused a tremendous increase of tuberculosis of the lungs in those working with these appliances. In 1890, when stone was cut and polished with comparatively crude tools, the death rate from tuberculosis among these workers was 150 per 100,000. By 1910 it had increased to 1,080 per 100,000 and by 1925 had reached 1,950, an increase to 13 times that of the period before high-speed tools came into use. The explanation, of course, is that modern equipment creates many times the density of dust produced by manual labor. Cases of silicosis also became very numerous, and many of them developed tuberculosis. During this same period, when tuberculosis was increasing so rapidly among these workers, the death rate from tuberculosis throughout the country was reduced to about half the rate of 1890.

The report of the Special Industrial Disease Commission of Massachusetts (*121*) shows that the granite industry presents a severe silicosis hazard. The commission surveyed 314 granite establishments and studied dust conditions in 13; X-ray and physical examinations of 961 granite workers disclosed that 15.2 percent had silicosis and 7.6 percent silicosis with tuberculosis.

FOUNDRIES

The Special Industrial Disease Commission of Massachusetts surveyed 225 foundries (*121*), in which 12 dust studies and 1,614 physical examinations and X-rays were made. In the foundries studied, dust counts were excessive in connection with many of the operations. The need for immediate, effective control of dust conditions in foundries was evidenced by the fact that 8.8 percent of workers examined had silicosis and 2.6 percent silicosis with tuberculosis.

In a study of the foundry industry, McConnell and Fehnel (*122*) found that the death rate for respiratory diseases (including all forms of tuberculosis and influenza) for iron and steel foundry workers was about two and one-third times that for workers in all industries combined and was more than twice that for workers in any of the industries selected for comparison. More than a third of all deaths among foundry workers were caused by some respiratory disease, whereas little more than a fifth of the deaths of workers in industry were due to these causes. A striking fact revealed is the uniformly high death rate in this industry for each of the respiratory diseases throughout the working period of life. Another outstanding fact is the high rate for pneumonia, bronchitis, etc., the foundrymen's rate being nearly three times that for all workers combined. Among iron and steel molders, founders, and casters, where 12 deaths from pneumonia might have been expected, there actually were 38, a ratio of

actual to expected deaths of 315 percent; 24 deaths were recorded for respiratory tuberculosis, where 13 might have been expected, a ratio of 179 percent; the ratio for influenza was 216 percent, based on 23 actual compared with 11 expected deaths. In an analysis of the occupational mortality of adult, white, male, industrial policyholders of the Metropolitan Life Insurance Co. who died during 1922–24 the proportion of deaths due to any one cause in any one specified occupation group is compared with the proportion due to the same cause among all occupied white males. Again, pneumonia was the leading cause of death among foundry workers, a condition not true of any of the other 71 occupations included in the analysis. When differences in the age composition of the two groups were taken into account, it was determined that the proportion of deaths from pneumonia was 120 percent higher for foundrymen than for occupied males generally. Likewise, the proportion of deaths from influenza was 82 percent higher, that for tuberculosis of the respiratory system 5 percent higher, and that for other respiratory diseases 17 percent higher. McConnell and Fehnel (*122*) emphasize the harmful effects on the lungs of exposure of workers to dust inhalation; 67 of 215 X-rays taken were diagnosed as positive for silicosis. Advanced cases of silicosis were not found among those examined, but sufficient evidence of occurrence of the disease was presented.

As the result of a health survey in 1931 by the Metropolitan Life Insurance Co. (*123*) of representative foundries in and around Milwaukee, Wis., 67 of 215 employees studied (31 percent) were diagnosed as having silicosis, but advanced cases were not found. As these results indicated a definite hazard, the report recommended the selection of workers in foundries by preemployment examinations and chest X-rays, along with periodic examinations of those dangerously exposed.

In 1938 Sander (*123*) reported a 4-year survey which was begun in 1932; 4,035 foundry employees were studied. About 7 percent had definite silicosis, with half of the total group having had fewer than 10 years of foundry exposure. Of the 279 with silicosis, 60 (22 percent) had tuberculosis.

In a report issued in 1939, the Division of Industrial Hygiene of the New York State Department of Labor (*124*) stated that 2.7 percent of the whole group of foundry workers had silicosis, while 10 percent of the workers having more than 40 years' exposure were affected.

In 1931 a survey was made of 21 foundries in Wisconsin (*125*); 215 roentgen examinations were made. Of 130 men examined, 1 had second-stage silicosis plus tuberculosis; 31 had first-stage silicosis; and 30 were classed as doubtful.

Of 62 men from steel foundries, 2 had second-stage silicosis; 25 first-stage; and 6 were doubtful.

Of 18 men from the malleable iron foundries, 7 had first stage and 2 were doubtful.

In 1939 Sander reported (*125*) a study of 4,000 foundry workers, 7 percent of whom were silicotic; 2 percent of the 7 percent were also tubercular.

From the time (1937) the amended occupational disease law of Ohio (*126*) added silicosis to the list of compensable diseases to December

31,1940, 579 silicosis claims were filed; 194 of these claims were allowed, 100 of which (51 percent) originated in foundries, 52 in iron foundries, 27 in steel foundries, and 21 in nonferrous foundries.

The following summary by Williams (*127*) of the range, average, and average deviation of the free silica content of various raw materials and dusts present in foundry operations indicates the possibilities of contracting silicosis in the foundry industry:

Summary

Table	Number of samples	Average SiO_2 (*percent*)	Range of SiO_2 (*percent*)	Average deviation (*percent*)
1. Foundry sands	19	26 1	17-40	±6 5
2. Parting compounds—carbonate	23	1 5	tr-6	±1 6
3. Parting compounds—phosphate	19	3.1	tr-9	±1.7
4 Parting compounds—mis.[1]	6	1 0	tr-4	
5 Molding	30	12 1	5-49	±7 4
6. Cleaning	18	12 8	tr-61	±10 2
7. Sand conditioning	13	13. 2	4-27	±5 8
8. Shake-out	12	8. 4	2-22	±4 1

[1] Excluding silica partings.

CEMENT INDUSTRY

A 3-year study of the cement industry by the United States Public Health Service (*128*) revealed the following:

X-ray films were obtained of the chests of 53 employees in several different occupational groups, length of service varying from 6 months to 15 years. On the basis of the findings these employees were classified into five groups, as follows: (1) Those showing evidence of pneumoconiosis, (2) those showing evidence of tuberculosis and pneumoconiosis, (3) those negative for pneumoconiosis, (4) those showing evidence of tuberculosis without pneumoconiosis, and (5) doubtful cases.

The earliest case of pneumoconiosis appeared after 3 years' exposure to cement. Of the 53 men selected, 37 had been in the industry more than 3 years and are therefore used as a basis of comparison. Among these 37 men 15 showed evidence of pneumoconiosis, 3 of the 15 also having tuberculosis. In the group of 22 who showed no evidence of pneumoconiosis 8 gave evidence of arrested tuberculosis, 4 showed no evidence of either pneumoconiosis or tuberculosis while 10 were considered doubtful.

In none of the cases showing pneumoconiosis were there any clinical symptoms of the condition, although the general fibrosis present and its distribution through the lungs were indicative of the condition. Cement workers appeared to have more than a normal amount of calcified nodes in the lungs.

Of the 570 workers examined 21, or 3.7 percent, were diagnosed as either positive or suspected cases of pulmonary tuberculosis. In only two cases, however, was the disease active at the time of the first series of special chest examinations, and neither appeared to progress as the result of exposure to the dusts. In the history of both cases it was evident that the disease had developed before the men entered the cement industry. One of them died about a year after the study closed, while with the other the disease appeared to have become quiescent when the study was closed. All the rest of the cases, with the exception of three, which were doubtful as to diagnosis, appeared to have developed their lesions before they entered the industry and were continuing in their occupations without any evidence that the lesions were progressing.

Russell (*129*) calculated that the frequency of disability due to respiratory diseases among cement workers was twice as great as the average respiratory rate among employees of 11 manufacturing plants in relatively nondusty industries. The highest rate for all

respiratory diseases in any one of these establishments was 30 percent below the rate for cement workers.

In 1935 the Portland Cement Association requested the Saranac Laboratory to survey a representative group of its member plants and examine a large number of employees to determine whether there was a dust hazard in the cement industry. In the report issued in 1939 (*130*), it was stated that the survey of 17 cement plants and examination of 2,278 workers revealed that the incidence of tuberculosis and other chronic infections of the lungs was less than that in the general population. It was concluded that prolonged inhalation of cement dust has no unfavorable influence upon susceptibility to tuberculous infection. The results of this survey seem to contradict the conclusions reached by Russell (*129*).

POTTERY INDUSTRY

A physical examination of all but about 1 percent of the workers in nine West Virginia potteries was given between July 1936 and July 1937 by the United States Public Health Service (*131*). Of the 1,627 men and 889 women examined, 106 men and 17 women were in the first stage of silicosis; 55 men and 5 women were in the second stage; and 6 men were in the third stage.

In Ohio (*126*) in the four divisions of the ceramic industry which form the whiteware group—china, tableware, floor and wall tile, and insulators and sanitary ware— 42 silicosis claims were allowed in the period August 1, 1937–October 1, 1940; 21 of the claims originated in china and tableware plants, 17 in floor and wall-tile plants, and 4 in electric porcelain plants.

SILICA AND FIRE-BRICK INDUSTRIES

Pennsylvania is the largest producer of silica brick in the United States. Four representative plants engaged in this industry were investigated by the Bureau of Industrial Hygiene of the State Department of Health. The results of this investigation were published in 1941 (*132*). Physical examinations of 1,035 silica brick workers revealed that 538 (51.9 percent) had silicosis. Tuberculous infection was found in 123 (11.9 percent) of the 1,035 examined.

A study, reported in 1945, was made by Stalker (*133*) in the refractory-brick industry in Kentucky, which ranks fourth among the States in production and in number of people employed in the industry.

The manufacturing processes employed in these plants are of the conventional type and are basically similar to those described in the Pennsylvania silica-brick study (*132*). The raw materials and maturing temperatures, however, are quite different from silica brick. The raw plastic clay contains 17.7 percent SiO_2 and the raw flint clay 7.1 percent SiO_2.

To determine the hazard of silicosis to which the workers were exposed, 876 men were examined, representing 87 percent of the employees. The term "pneumoconiosis" is used by the investigators (*134*) for the characteristic pulmonary change noted in 11 percent

of the men examined. This change was not of the type or magnitude usually accepted in compensation courts as the discrete, nodular fibrosis of silicosis.

The report of this study concludes that continuous employment in the firebrick industry, without dust controls, leads to the development of characteristic diffuse, finely granular, pulmonary fibrosis. Conglomerate silicosis may develop after long employment in dry pan mills without adequate dust controls.

AUSTRALIA

In 1902, while a system of sewers was being dug in Sydney, Australia, the Sewer-Works Ventilation Board (*135*) was appointed to inquire into the working conditions and to recommend means of improvement whereby the work could be rendered less hazardous. The board found that—

> For many years past miners employed in this class of work (tunneling) suffered acutely from a disease which was for a long time known by the rather misleading term "sewer disease," but as the complaint became more widely known it was strongly suspected that dust was the chief cause of the mischief.

The board gave the fumes of explosives and the expired air from the lungs of the miners in imperfectly ventilated tunnels as causes contributory to the high mortality but stated that the dust from hammering, drilling, and use of the pickax was probably the sole cause of the disease once known as "sewer disease."

In 1912 Armstrong (*136*) investigated a reported epidemic of pneumonia at Broken Hill and found that the death rate from pneumonia among underground miners in that locality during 1910–12 was 6.5 per 1,000, or nearly 4 times as great as that for all males in New South Wales. In 1914 a royal commission was appointed to inquire into the mining industry at Broken Hill. With regard to industrial diseases, this commission stated that pneumoconiosis and plumbism were among the risks of a miner's calling, while other diseases—for example, pneumonia—were added risks but not strictly industrial because they affected other than miners. From unanimous medical testimony the commission concluded (*136*) that pneumonia was more prevalent, severe, and fatal among miners in Broken Hill than among any other class in the State. Although attributing the disease primarily to sudden changes in temperature, the commission advanced the opinion that dust inhalation in any form would predispose to disease. It also stated that pneumoconiosis was virtually unknown at Broken Hill and blamed mining in other States for such cases as had been noted. Tuberculosis, however, was a disease to which the miners at Broken Hill, as elsewhere, were peculiarly subject, and work in a mine was considered particularly dangerous for any tuberculous patients.

In 1919 the Technical Commission (*136*), constituted to examine the miners at Broken Hill, for the first time in Australia used X-rays in diagnosing silicosis as an occupational disease. Of 4,337 mine employees examined, 193 were diagnosed as having pneumoconiosis—90 in the first stage, 44 in the second, and 59 complicated with tuberculosis; 39 cases were definitely diagnosed as suffering from

pulmonary tuberculosis only, and 26 others were thought to be suffering from uncomplicated pulmonary tuberculosis. Of 2,618 underground men examined in 1922, 266 showed pneumoconiosis—113 in the first stage, 51 in the second stage, and 102 complicated by tuberculosis; 107 additional men were diagnosed as suffering from tuberculosis only.

In 1922 the New South Wales Board of Trade, in conjunction with the Commonwealth Department of Health (*136*) investigated clinically and radiologically the prevalence of pulmonary diseases among workers in sandstone and other siliceous rocks in the metropolitan district of Sydney; of 716 men examined, 123 were suffering from silicosis. Under the standards set by the Broken Hill Commission, 47 of these were diagnosed as having first-stage silicosis, 38 second-stage silicosis, and 38 pneumoconiosis complicated by tuberculosis; 16 were diagnosed as having pulmonary tuberculosis only.

The Workers' Compensation Commission of New South Wales (*137*) reported on 1,300 of 1,500 workmen examined up to August 23, 1929, as follows: Of 107 stonemasons examined, 9 had tuberculosis, 17 had silicosis of various stages, and 26 had silicosis with tuberculosis. These men had worked in the industry 10 to 55 years. Of 29 monumental masons examined 2 had tuberculosis, 1 class A silicosis, and 5 silicosis with tuberculosis. Of 37 ballast quarrymen, 1 had tuberculosis and 2 marked silicosis. Of 80 dimension quarrymen, 6 had tuberculosis, 3 early silicosis, 2 marked silicosis, and 16 silicosis with tuberculosis. Of 377 rock choppers examined, 12 had tuberculosis, 25 silicosis, and 10 silicosis with tuberculosis.

In 1931 Moore (*137*) reported an investigation in New South Wales to determine the incidence of fibrous pneumoconiosis among coal miners; 471 volunteers who had worked a least 10 years underground were examined by the Division of Industrial Hygiene; 199 came from 1 mine, several employees of which had had pulmonary fibrosis; the remaining 277 volunteers were employed in 8 other mines of the district. The percentage of cases of fibrosis for all men examined was 25.9. The percentage of fibrosis among men with coal-mining history only ranged from 22.5 to 25.7 percent; the percentage incidence among men who had worked also in metal mines or quarries as well as in coal mines was 39.7.

In Tasmania in 1928 (*135*), the various mining centers were visited in turn and men examined clinically and radiologically virtually at the mine head; 65 percent of the mine employees available were examined. Of 134 underground workers, 5.9 percent were suffering from uncomplicated silicosis and 2.1 percent from silicosis with tuberculosis. Of all the workers examined, 1.1 percent were suffering from tuberculosis only—1.2 percent of the underground and 1 percent of the surface workers.

In 1940 a royal commission appointed to report on pulmonary diseases among the miners in the Western Australia gold-mining industry (*135*) concluded that—

> The miner is more liable to lung disease generally than the average male over 15 years of age. The miner is less long-lived than the average male over 15, partly on account of greater liability to lung diseases.

Tuberculosis of the lungs is on the increase among miners and is twice as prevalent as among males over 15.

Pneumonia among the acute, and bronchitis, asthma, emphysema, and fibrosis of the lungs among the chronic lung diseases are more prevalent among miners than among males over 15.

The following principle was laid down:

Any man suffers from fibrosis to the extent to which he is exposed to the continued inhalation of mineral dust. If there is no dust, there will be no fibrosis, and conversely, the continued inhalation of dust certainly produces fibrosis.

During an investigation (*138*) of pulmonary conditions of mine employees in Western Australia in 1925–26, 3,039 men were examined at Kalgoorlie and 1,028 at 9 other mining centers throughout the State. Of the 4,067 men examined, 798 (19.6 percent) presented definite evidence of pulmonary silicosis, and 155 (3.8 percent) had tuberculosis; 12 of the latter group had tuberculosis without silicosis. Of the 1,759 surface workers, 6.6 percent had silicosis, 1.5 percent silicosis with tuberculosis, and 0.3 percent tuberculosis alone. The surface workers engaged in dry milling without previous underground experience showed the highest incidence of silicosis—9.6 percent were affected compared with 2.2 percent of the employees engaged in wet milling without dry-milling experience and 2.0 percent of those who had had no wet- or dry-milling experience. Of the 2,308 underground workers, 23.3 percent had silicosis, 5 percent silicosis with tuberculosis, and 0.3 percent tuberculosis alone. At least 51.5 percent of the workers engaged in developing and 39.2 percent in stoping were affected with silicosis or tuberculosis, in contrast with 8.4 percent of those without stoping or developing experience.

Since 1926 the mine employees in Western Australia have been examined annually by the Commonwealth Department of Health (*138*). Of 2,290 men previously classed as normal, 30 (1.3 percent) were found on reexamination in 1927 to be suffering from uncomplicated silicosis and 13 (0.5 percent) had tuberculosis only; of 491 diagnosed as silicotic, 86 (17.5 percent) had silicosis with tuberculosis. Of 2,822 men classed as normal in 1927, 48 (1.7 percent) had progressed to 1928 to silicosis only, 11 (0.4 percent) had progressed to silicosis plus tuberculosis, and 3 (0.1 percent) had simple pulmonary tuberculosis; 25 (5.9 percent) of 425 silicotics on reexamination showed signs of tuberculosis complication. Of 2,293 normals reexamined in 1929, 100 were diagnosed as silicotic. Silicosis was not disclosed in any case under 40 years of age or with less than 5 years of underground work.

Badham (*139*) reported that, of 477 coal miners working in the same pit, who were examined in 1930, 25 showed evidence of fibrosis. In New South Wales 116 of about 10,000 coal miners employed underground had been compensated.

Tabulation of data resulting from the examinations conducted from 1925 to 1939 revealed a steady decline in the incidence of advanced and early silicosis and an increase in the number of normal persons employed in the mines from 80 percent of the total to 95.63 in 1939. For example, 11.4 percent of the miners examined in 1925–26 had early silicosis, and in 1939 the percentage was 4.0; comparable figures for advanced silicosis were 4.5 and 0.1 percent, respectively, and for silicosis and tuberculosis 3.3 and 0.2 percent, respectively.

In 1907 a report on miners' phthisis to the Committee of the Bendigo Hospital, Victoria, Australia (*135*) stated that there was undue mortality among Bendigo miners due to respiratory diseases, notably tuberculosis. The cases examined were classified according to two clinical types—a pure fibrosis of the lungs, nontuberculosus in origin; silicosis; and a mixed type with a tuberculous infection in a fibroid lung. Of 61 men examined in Bendigo in 1928, 9.4 percent of the underground workers were diagnosed as suffering from uncomplicated silicosis and 7.5 percent from silicosis and tuberculosis; 3.3 percent of all workers had tuberculosis only.

CANADA

Elliott (*140*) was the first to direct attention to silicosis in Ontario, Canada; in an article on Silicosis in Ontario Gold Mines, published in 1924, he reported that 3 of 11 underground workers examined by him presented evidence of silicosis, indicating that the disease was being contracted in this mining area. In 1925 and 1926 a further survey was made by the Industrial Hygiene Division of the Ontario Department of Health. Miners with more than 5 years' experience underground in the individual mining area and with no exposure to silica dust elsewhere (a few of those examined had had some exposure elsewhere) were given a physical examination. Of 1,220 men examined, 74 showed evidence of silicosis—52 in the anteprimary, 11 in the primary, and 11 in the secondary stage.

During the period April 1, 1926–April 1, 1927, a survey of silicosis was made in the Porcupine group of mines in northern Ontario under the auspices of the mine operators in compliance with the amendment to the Miners' Phthisis Act of April 8, 1926, bringing silicosis under the status of a compensable industrial disease. Approximately 4,000 miners were examined for tuberculosis, silicosis, or both. As a result, 94 silicosis claims were submitted for compensation. According to the South African nomenclature 39 of these 94 cases were diagnosed as anteprimaries, 28 as primaries, and 27 as secondaries; in addition, 247 men showed either positive or probable tuberculosis (*141*).

From 1926, when silicosis was included in the list of compensable diseases under the Canadian Workmen's Compensation Act, to 1940, 462 claims had been accepted for compensation (*142*).

NETHERLANDS

In The Netherlands (*143*) a medical examination of 2,013 stonemasons in 1899 revealed 169 tuberculous cases and 10.8 percent strongly suspected of having tuberculosis. During the period 1891–1900 the death rate among stonemasons for all men between 18 and 50 years of age was greater than in any other occupation. Even such injurious occupations as typesetting, cigarmaking, and diamond cutting showed considerably lower death rates. The death rate in the age group 18–24 years differed little from the average for all occupations; the highest death rate was noted between 36 and 50 years. Of 952 stonemasons dying during 1886–98, 87 percent died of diseases of the respiratory organs. The first general examination of adult stonemasons

was conducted in 1923, the second in 1926, and the third in 1929. In 1923 in nearly half the cases the X-ray gave more information than the physical examination. Of 69 cases in 1929, lung affections were not shown clinically in 19 but were shown in the X-ray examination, and in 38 cases they were shown both clinically and by X-ray.

On examining stonemasons who, in the course of their lives, had worked certain kinds of stone and others who had worked more than one kind of stone, Kranenburg (*143*) reported that, of 16 sandstone workers, 2 were in the second stage and 5 in the third stage of silicosis after 20 to 30 years' employment in the occupation; of a total of 74 Belgian limestone masons, only 5 were in the second stage and none in the third stage after the same number of years' employment in the occupation. Using the third stage for comparison, the proportion was more unfavorable among the 90 stonemasons working various kinds of stone including sandstone than among the Belgian limestone workers, although relatively favorable in comparison with the sandstone workers. A striking fact was the large number (77) of Belgian limestone workers who showed no affection after 10 to 30 years' employment compared with only 4 stonemasons working all kinds of stone. Fewer cases of third-stage silicosis were found among Belgian limestone workers after 40 to 60 years' employment or longer than among sandstone workers.

In his general conclusions Kranenburg (*143*) made the following statement regarding the effects of sandstone and limestone:

> The working of sandstone alone must be regarded as more injurious to the lungs than the working of limestone alone. The working of sandstone alone does not always lead to symptoms of silicosis and, on the other hand, the working of limestone alone (Belgian limestone, marble) does not prevent silicosis in a serious form. The working of sandstone and limestone alternately appears to produce less serious results in the same period than the working of sandstone alone.

An inquiry by Vossenaar and Doubrow (*144*) into the respiratory affections of 600 men working in the coal mines of The Netherlands from 1929 to 1936 revealed only one worker with an image of micronodulation; all the others presented a perceptibly normal thoracic image. Sixty of the 600 workers had been employed at rock drilling for more than 20 years, the others for more than 10 years.

In 1935 an inquiry was made regarding silicosis in the coal mines of Belgium by 15 particularly competent medical men (*145*). One thousand working miners were given X-ray examinations; 900 of these had worked exclusively in rock (tunnelers); and 100, used as controls, had been occupied exclusively in working the coal seam. Normal subjects were the exception in all workers who had worked for at least 10 years in the mine. A considerable number of them were suffering from extensive or highly developed fibrous or even from nodular and pseudotumoral forms. The investigators were surprised to discover, in workers employed on the coal seam and not in direct contact with rock dust, as high a proportion affected as among rock workers.

During the 10 years from 1936 to 1946, periodic r ntgenologic examinations were made by the Medical Service of The Netherlands Colliers (*146*) of workers in rock in the State mines Hendrik, Emma, Wilhelmina, and Maurits. A total of 2,300 workers in rock were ex-

amined. During a period of more than 10 years, 4,977 X-ray pictures were made of the lungs of 2,320 underground hard-coal miners working in rock; 10.15 percent of the pictures revealed mild pneumoconiotic changes (reticulation and silicosis type I); 1.45 percent showed moderate changes (agreeing with silicosis type II); and 0.54 percent indicated more severe changes (as with silicosis type III). Although these figures refer to rock workers, Mey (*146*) states that:

> The thought obtrudes that not only the rock work gives rise to the appearance of pneumoconiotic changes.

DENMARK

An X-ray examination of 461 men and 337 women employed in 5 Danish porcelain works (*147*), reported in 1934, showed that 45.2 percent of those examined had silicosis and 100 percent of those over 50 years of age were affected. This investigation led to the introduction of preventive measures, the efficacy of which was studied in 1941 and 1942. Radiographic examinations were made of 39 workers, 32 of whom had been examined in 1932. This follow-up study revealed that it is most difficult to be certain of the radiographic diagnosis of early porcelain silicosis. Some of the cases reported as having mild or beginning second-stage silicosis at the earlier examination showed "no or no certain silicosis" at the examination given in 1942. The authors concluded that great experience is required to be fairly sure that an X-ray film indicated incipient silicosis. Gudjonsson and Möller (*147*) found silicosis widespread among the 951 porcelain workers they examined in Denmark.

V. Stub-Christensen (*148*) examined workers in the foundry, stone-cutting, grindstone, cement, scouring-powder, polishing, tile, and sand-blasting industries of Denmark. All of the 90 foundry workers except 5 were able to work at the time of the examination. The author observed that, with these workers, silicosis appeared between 10 and 19 years of work; beyond this period all the workers became affected.

Fifty percent of the molders in a large foundry examined by Komissarock (*148*) presented rather serious pulmonary affections, the typical disease images appearing only after 30 years of exposure.

Of 23 grindstone cutters examined 17 had silicosis.

In the cement industry Stub-Christensen found slight silicotic lesions after 10 to 20 years' work; after 40 years' work the lesions were present in almost 100 percent of the workers; 66 percent of his cases had lesions of the first stage, 8 percent of the second stage, and 5.5 percent of the third stage. The author states that, in the cement industry, vigorous physique does not appear to protect the workers against pulmonary lesions. In the polishing industry, of 40 workers examined 12.5 percent had silicosis in the second stage; none was in the third stage, and none had silicotuberculosis.

FRANCE

On October 16, 1860, the Medical Gazette of Lyon (*149*) stated:

> It has been established by recent statistics that among the workers employed in the mines of Cornouailles 61 in a 100 died of disease of the chest, while in the rest of the population 31 in a 100 died of this cause.

Between April 1942 and May 1943, Martin and Roche (*150*) examined 609 rock miners. Six of these had ordinary tuberculosis. Radiologic deviations were observed in 102 or 16.9 percent of the remaining 603 miners. Coal dust was incriminated by the authors in the genesis of 14 cases of pneumoconiosis in rock workers who had been employed in coal previously.

Experience for 12 months preceding August 1943 of the chief physician (*151*) of the new Society of Coal Mines of Bouches-du-Rhone did not reveal a single case of pneumoconiosis of silicotic appearance among lignite miners. Little dust is produced in the extraction of the lignite, but a hazard exists in working the lignite. Fifty-five workers, some of whom were the oldest and some of whom had worked for a long time drilling in the rock encasing the lignite veins, were considered as suspects. They were examined clinically and radiologically; 47 of these workers showed no marked pneumoconiosis and no trace, past or present, of tuberculosis. Eight of the 55 workers were clearly pathologic, but only 2 had spent all their occupational life in the exploitation of lignite; both of these revealed important pulmonary fibroses but did not have the classical signs of silicotic pneumoconioses.

INDIA

In the many discussions on the incidence of silicosis among quartz miners in various parts of the world, the Kolar field in India has been cited repeatedly as an exception to the general experience that silicosis is an occupational disease among such miners, as is seen, for instance, among miners on the Rand (*152*).

The Rege Committee (*153*), however, reported in 1946 that silicosis is an occupational—indeed the only occupational—disease prevalent in the Kolar. It is now so recognized, and in 1942–43 compensation was awarded in 778 cases.

According to Barenbrug (*154*), although classical silicosis does not occur in the Kolar gold field, it is not denied that pneumoconiosis (which means dust disease of the lung) does occur. After many years of exposure to dust, this disease can be incapacitating. The condition is characterized by a heavy dust deposition in the lung with an absence or a minimal degree of fibrosis. Disability is absent for many years, but susceptibility to tuberculosis is increased.

Apparently the reason for the difference in the disease as it occurs in the Kolar gold field and the Witwatersrand is due to the lower silica content of the mine dust, which is about 10 percent, and the Kolar dust is coarser than the Rand dust. The position as given by Barenbrug (*154*) is that Kolar miners inhale large quantities of a relatively coarse dust (the number of particles above 20 microns in miners' lungs is most striking) composed mainly of silicates of aluminum, magnesium, iron, calcium, sodium, and potassium and about 10 percent free silica. The Rand miners inhale relatively small quantities of a very fine dust with a free silica content of 70 percent. Barenbrug estimates that the Kolar miner inhales almost as much free silica as the Rand miner. If this is accepted, the low toxicity of the Kolar dust must be due to dilution of quartz by the silicates and/or the relatively large size of the dust

particles. It is even possible that the silicates modify or inhibit the action of quartz.

Barenbrug (*154*) concluded his article with the following statement:

> All this is conjecture but one thing is certain. If Rand miners worked in dust concentrations comparable with those existing on the Kolar gold field, silicosis would be as serious a menace on the Rand as it was at the beginning of this century. If the Rand reverted to dry mining the silicosis hazard would be the most formidable problem to be faced and unless there is a revolutionary development in methods of dust control it is difficult to foresee how it could be overcome. The problem of the ability of Rand miners to work efficiently in the altered conditions of higher dry-bulb temperatures is of secondary importance. Acclimatization to high temperatures kept at a moderate level with the aid of air cooling plants and improved ventilation is possible but as far as we know acclimatization to inhaled silica particles is not possible.

ITALY

In Italy the first clinical and anatomical observations (*155*) probably were made by Ramazzini. Biondi (*156*) in 1906 noted that the greatest damage to the miners in the mines of Sardinia was due to the action of dusts from gangue containing silicates. He also noticed that virtually all coal miners were addicted to coughing and continued to expectorate black sputum even some time after they had quit their occupation. He found minor dust injuries among the Bergamo miners in 1907, perhaps because they labored intermittently in mines and on farms. However, in a few miners, especially the older men, Biondi found pharyngitis and bronchial catarrh, with dark sputum due largely to the black smoke of the candles used; he observed that acute affections of the respiratory apparatus were more serious and more obstinate in these workers.

In 26 post mortem examinations of Sardinian miners, Frongia (*157*) in 1908 noted that the cause of death was in most cases an acute infection (pneumonia); he also found among these workers many cases of serious and diffused arteriosclerosis, emphysema, and anthracotic foci, with more or less diffused softening of the tissues and gangrenous foci containing a black brothlike matter—the characteristic finding of black phthisis. On the other hand, he found foci of bronchiectasis due to tuberculosis infection in only five cases. Eighty-five percent of the fatal cases among these miners, therefore, were not caused by tuberculosis infection.

Pesenti, Rota, and Finzi (*158*) in 1906 reported on health conditions of workers in lime, cement, and plaster. Pesenti affirmed the high incidence of pneumoconiosis among workers in cement and plaster but did not include proportional statistics. He believed that inhalation of cement dust neither produced tuberculosis nor favored the progress of the disease. Rota and Finzi found that, of 63 deaths among the permanent workers engaged entirely in lime and cement work, 28 died under the age of 40 and 11 under 50. The cause of death in 20 of the 63 cases was pulmonary diseases—9 acute pneumonia, 2 pleuromediastinitis, and 9 tuberculosis. Rota and Finzi stated that there was no truth in the statement that workers in lime-kilns enjoyed a certain immunity from tuberculosis. On the contrary, they claimed that the greatest toll to this disease was paid by kiln workers. For example, 9 of 35 deaths of such workers were due to

tuberculosis. Physical examination of 218 factory workers revealed 122 cases of harsh respiration in the upper respiratory passages, owing certainly, they asserted, to an incipient pneumoconiosis or infiltration of dust into the peribronchial lymphatic channels. Harsh respiration was more common in those employed for a long time, especially those who had worked 10 to 12 years; it was evident in 52 of 128 kiln workers, 18 of 26 workers transporting stone, and 43 of 50 crushing-machine operators, baggers, and porters of sacks. Many workers were also emphysematous.

Bianchi (*159*) made a careful clinical and radiological examination of 250 workers employed in more or less closed workrooms, as sculptors, rough hewers, modelers, and workers in grinding rooms. After excluding all cases of suspected syphilis and other chronic lesions, he studied 73 workers in marble who had no inherited disease or previous disease history and were free from definite or suspected symptoms of affections of the respiratory passages that might come under the category of common infections. X-ray examination revealed diffuse nodes of thickened tissue spread over almost the entire respiratory surface. In these cases he could determine only a slight percentage (20 percent) of functional alterations with symptoms of chronic bronchitis and pulmonary emphysema.

As the result of a radiographic and clinical examination of 105 Carrara marbleworkers employed in marble grinding and of such workers as sculptors, rough hewers, sawyers, and polishers Turano (*160*) concluded:

> The changes met with among Carrara marbleworkers may be classed in the initial stage of pneumoconiosis, that is to say, comprising the least serious forms of the disease, such as the very marked reinforcement of the pulmonary outline, due as demonstrated by personal observations, to the processes of arteritis and lymphangitis, and in certain cases equally to conditions of emphysema usually present among those workers.
>
> I have also found frequent pleuritic changes which can be related to inhalation of marble dust, but never lesions of the pulmonary parenchyma.
>
> The radiological aspect of pulmonary tuberculosis among workers examined is, on the other hand, highly important, since it shows an atypical picture on which are noted lesions with unusual sites and apical and subclavicular regions unaffected. It is this fact which has led me to admit the probability of a combination of pneumoconiosis and tuberculosis.
>
> Finally, statistical as well as radiological data justify absolute exclusion of the theory of a particular benign or malignant course of tuberculosis among marbleworkers, as likewise of any kind of predisposition to the said specific disease.

According to Parmeggiani (*161*), social consideration of silicosis, which before compulsory insurance was almost nonexistent, suddenly was pressed on the attention of doctors, industrialists, and magistrates between 1937 and 1943.

In 1941, 1,773 appeals were forwarded to the section on labor controversies of the Tribunal of Milan; 883 (or 47 percent) were for physical injuries of persons due to work. This group was divided broadly into accidents and diseases while at work. The diseases included 654 cases of silicosis (98 percent) and 11 cases (2 percent) of other diseases; 602 of the cases of silicosis were examined by Parmeggiani (*161*). Of the 602 trials for silicosis 349 (58 percent) were conducted to the end; of these, 342 were promoted by the workers who

had worked in dusty industries limited to the Province of Milan. The judgment of the cases was as follows: Favorable to the workers (silicosis ascertained) 201 (58 percent), unfavorable to workers (silicosis nonexistent) 148 (42 percent).

RUSSIA

In Russia an extensive field study was made in 1930–35 (*162*) at one anthracite and two bituminous-coal mines in the Donetz Basin; it was carried out under the Ukrainian Central Tuberculosis Institute and the Donetz Institute for Industrial Hygiene and Occupational Diseases.

In the Khrustalny (anthracite) colliery, pneumoconiosis was found in 11.1 percent of the 298 men—5.7 percent in stage I, 2.3 percent in stage II, and 3.0 percent in stage III. In the Vetka (bituminous) colliery, the incidence of pneumoconiosis was lower, being found in 3.3 percent of the 1,329 men; 2.3 percent in stage I, 0.9 percent in stage II, and 0.1 percent in stage III. In the Gorlovka (bituminous) colliery, the incidence was also comparatively low, being found in 2.3 percent of the 987 men; 1.5 percent in stage I, 0.5 percent in stage II, and 0.3 percent in stage III.

SOUTH AMERICA

BRAZIL

The high temperatures found in the lowest depths of the Morro Velho, the deepest mine in the world, do not permit wet drilling. In 1939 a commission of three doctors (*163*) examined 2,197 of the miners, 908 of whom were X-rayed; 304 were found to be definitely silicotic—that is, 33.48 percent of those X-rayed and 13.83 percent of those examined.

CHILE

During the period 1942–45, more than 1,200 persons suspected of having pulmonary silicosis entered the Instituto Medicina del Trabajo de la Coja de Seguo Obligatorio (Medical Institute of the Compulsory Insurance Fund) (*164*) in Chile. Two hundred verified cases of silicosis were analyzed; 172 were pure pulmonary silicosis and 28 silicotuberculosis. All of those examined worked in the copper mines, except 20 who were employed in industries manipulating quartz, as glassware, and in ceramic factories.

As a result of his recent studies in South America, Bloomfield (*165*) found it is well-known that in Chile many workers with silicosis are still employed in industrial establishments in which silica in the form of quartz is handled. For example, 15 percent of 1,000 copper miners examined in 1946 were found to have silicosis with varying degrees of disability. The company indemnified 62 cases that year. In the same mine, nearly 500 silicosis cases had been settled during the preceding 12-year period. Although dust hazards are now being controlled in this establishment, there are still approximately 150 men employed by the company who will eventually be compensation cases because of previous dust exposure.

PERU

According to Bloomfield (*165*), although Peru has had an occupational disease compensation law since January 12, 1935, it is virtually impossible to obtain nation-wide statistics on the incidence of occupational diseases. One firm that employs nearly 12,000 workers reported that 271 cases of silicosis were certified by its medical department for payment in 1946.

Other examples of occupational diseases in Peruvian industry are at hand. In one mine, which employed nearly 1,500 workers at its various installations, some 1,900 applicants for work were examined in 1946. Three percent of these were rejected because of silicosis, apparently acquired elsewhere. Another mine employing some 500 men examined 890 applicants for work during the first 6 months of 1947. Nine percent of these men were rejected because of silicosis and 14 percent because of tuberculosis. Still another mine, employing about 450 men, reported that 8 percent of its workers had silicosis and tuberculosis. Another mine employing 500 workers reported 26.1 percent illness among its workers. The causes were pneumonia, grippe, bronchial pneumonia, and silicosis. There were 84 cases of silicosis at this establishment. At this same mine, 19 percent of 815 persons examined for employment in 1946 were rejected because of silicosis and tuberculosis. Of the 156 men rejected for these 2 causes, 128 had silicosis. And finally, the experience of still another mine was available for the period 1935 through 1942. During those 8 years, of some 6,499 men examined, 14.3 percent were found to have silicosis. During the same period, only 1.9 percent of those examined were found to have tuberculosis.

It is obvious from the above data, even though they are from scattered sources, that high rates of silicosis and tuberculosis exist throughout Peruvian mines and mills. Unquestionably these rates are an underestimate, since it is known that a high labor turn-over tends to mask true conditions. Labor turn-over in the factories is slight, with the exception of one factory where it was reported to be 50 percent annually; but in most of the mines it is extremely high, running from 35 and 50 percent yearly to more than 100 percent, especially for unskilled labor.

SPAIN

A very high illness frequency of pneumoconiosis was reported (*166*) in 1937 among the Rodaquilar coal miners. Of 110 workers employed in the mine, 40 exhibited more or less advanced disease, although the mine had been opened only 6 years. Suspected or detectable changes appeared on the radiograms of these 40 miners, 22 of whom showed silicotic and tuberculous changes. There were 12 cases of silicosis I, 2 cases of silicosis II, and 9 cases of silicosis III. It was also determined that 10 workers had died of silicotuberculosis during 1935.

SWEDEN

In 1942 Bruce (*36*) issued a report of a clinical and industrial medical study on silicosis as an occupational disease in Sweden. The study

included porcelain factories, iron works, sand blasting, smelters, sandstone and quartz crushing, quartz mills, sandstone grinding shops, and mining.

Among the 2,631 workers examined by the investigator in the industries mentioned 99 were suspected of having silicosis, 625 (23.7 percent) had silicosis, of whom 408 were in the first stage, 141 in the second stage, and 76 in the third stage.

The highest percentage (45.0 percent) of silicosis cases was found among sandstone grinders; however, only 20 were examined. The next highest percentage (35.4 percent) of silicosis cases appeared among the 229 casting cleaners, followed in order by the furnace masons, with 31.2 percent of 80 workers examined; the iron-ore miners, with 27.3 percent of 959 workers examined; the sandblasters, with 26.7 percent of 15 workers examined; the quartz-mill workers, with 24.6 percent of 65 workers examined; the porcelain workers, with 23.5 percent of 614 workers examined; the founders, with 19.0 percent of 259 workers examined; those employed in shaking out, with 12.5 percent of 24 workers examined; employees other than casting cleaners of casting cleaning plants, with 10.5 percent of 86 workers examined; furnace workers in silicon alloy plants, with 8.2 percent of 170 workers examined; and zinc and lead miners, with 4.9 percent of 81 workers examined.

The Swedish coal mines appear in a much more favorable light than those in Germany, owing to the natural humidity of the mines which prevents formation of much dust. Since 1931, when the rock-dust lung came under the general law compensating industrial diseases, no case of silicosis has been reported in the Swedish coal mines.

Of 543 underground drillers in the metal mines of Lapland examined, 215, or 39.6 percent, had silicosis.

SWITZERLAND

According to the reports received by the Swiss National Casualty Fund (*167*), the number of cases of silicosis reported for miners had increased from 2 in 1935 to 94 in 1944. The total of all reported cases of silicosis was 850, of which 367 were miners and 483 workers in other occupations.

From January 1 to December 31, 1945, the medical service of the Central Administration (*168*) has been required to pronounce definitely on about 2,500 reports of examinations and X-rays of insured persons exposed to silicosis and examined according to the requirements of the Federal Council and about 300 reports of registered insured according to article 65 LAMA (occupations exposed to quartz dust, excluding working in galleries, tunnels, and in mines).

Of those examined, 112 were declared disabled by silicosis or silicotuberculosis, and 66 were declared able for siliceous dust-producing occupations.

Up to 1947 about 1,400 cases of silicosis have been determined in Switzerland (*169*).

TYPES OF DUST INJURIOUS TO HEALTH

The relation between dust inhalation and lung diseases was recognized very early, as indicated by the literature cited, but it was also recognized that certain dusts caused severer symptoms with quicker fatal issue than others. As the symptoms caused by inhalation of certain dusts were long delayed or were attributed to other causes, such dusts were considered harmless and some even beneficial in the prevention of lung diseases when inhaled with the more harmful ones. Ancient writers seldom distinguished between the various forms of respiratory diseases but rather referred to a general relation between lung affections and dust inhalation (*34*), and no really scientific investigations of the workers or the dust were undertaken. Ramazzini, however, described the effects on workers of various types of dust produced in such industries as the hemp and flax industry, silk combing, and corn sifting, in addition to mining and similar industries. According to Collis (*34*), the distinctions, implied or definitely stated, in Ramazzini's excellent clinical description of the types of respiratory trouble which follow inhalation of different dusts are—

> the more notable, because even today pneumoconioses are pigeonholed in clinical teaching as a single entity, ascribed to exposure to any and every form of injurious dust, of which pulmonary fibrosis sums up the pathological findings and phthisis the morbid result.

In the decade preceding 1862, when the study of public health was organized in England (*34*), Greenhow made an elaborate statistical inquiry into the influence of occupation on health in connection with lectures on public health at St. Thomas Hospital. Simon, then medical officer to the General Board of Health, considered Greenhow's investigation so important that his report was published by the Board, and shortly after when Simon became medical officer to the Privy Council he entrusted Greenhow with the duty of further investigations in the great industrial centers. The resulting reports were, according to Collis (*34*), the first example of State medical inspection of factories, and he made the following statement regarding them:

> Throughout these reports runs as a theme the influence of dust inhalation in causing pulmonary disease, whether among lead miners in Yorkshire, tin miners in Cornwall, needle pointers in Alcester, cotton operatives in Lancashire, flax hecklers in Pately Bridge, metal grinders in Birmingham and in Sheffield, coal miners in South Staffordshire and in South Wales, or stone dressers in Stroud. Why work so well started was then allowed to lie dormant for so long, while other aspects of public health were being strenuously developed by medical officers of health with inspectors of nuisances appointed for every town and district, reinforced now by a battalion of tuberculosis officers is astonishing.

As a result of investigations in South African mines and in other mining districts of the world so much attention was focused on one particular dust—silica—as the most harmful encountered in industry that most investigators had accepted other dusts as harmless or of negligible importance as health hazards in industry and the disease silicosis resulting from breathing silica dust as the only important dust disease. Other dusts, such as the silicates (asbestos, for example), have been found almost as harmful as silica dust, but the effect on the lungs is somewhat different from that of silica, and the hazard not so widespread.

From general underground experience for more than 25 years in coal and metal mines and from an intensive study of dust for 8 years, Harrington (*170*) concluded that—

> Any dust insoluble in the fluids of the respiratory passages and in sufficiently finely divided form to float in the air and be breathed by underground workers will ultimately be harmful to health if the dust is in the air in large quantities and is breathed by workers for considerable periods of time. This applies to insoluble nonmineral as well as mineral dusts or mixtures of them and includes coal dust or mixtures of coal and other dusts. There are also some definitely harmful mine dusts which are soluble, and some dust experts appear to believe that the so-called insoluble dusts under certain conditions become soluble and are harmful only when soluble.

An outstanding impression gained by Ballantyne (*171*) from the reports of the International Conference on Silicosis was that silicosis constitutes only one facet of a very large question—the prejudicial effect of dusts in general upon the human subject. He concluded that—

> It (silicosis) is only one of several close-related pulmonary affections due to dusts of different kinds. It is the most important of these, but only because it is the most definitely known and so many workpeople are exposed to the risk. In the case of asbestos dust knowledge regarding the injurious effects of inhaling it has advanced so far as to justify the adoption of legislative measures analogous to those relating to silicosis. There are many other kinds of dust, however, as to the effects of which, when inhaled, we have little or no knowledge, and the investigation of which calls for early attention.

The report of the Silicosis (Medical-Arrangements) Committee issued in London in 1929 (*172*) contains the following statement:

> We are convinced that silicosis is more widespread than is generally believed and that it occurs to some extent in a number of industries and occupations where its presence has not been suspected.

In 1930 Kettle (*12*) called attention to the harmfulness of breathing dust:

> Dusts of various kinds occur in the atmosphere in all parts of the world, and the inhalation of these dusts over periods of years inevitably produces changes in the lungs. Everyone is familiar with the pigmented lung of the city dweller, an example of a pathological change produced by the inhalation of dust which is of no clinical importance whatever, but an exaggeration of this condition in the coal miner causes definite symptoms, and when the inhaled dust is not the relatively harmless carbon, but one of the much more sinister possibilities, serious pulmonary changes develop. The *pneumoconioses have received less attention* than they deserve, even in textbooks of medicine and pathology. But to the doctors whose patients are exposed to dusty trades, to those responsible for the conduct of these trades, and to the factory inspector pneumoconiosis is of great moment.

In the report of a study of dust conditions in German industries, published in 1932, Lochtkemper and Teleky (*80*) stated in the summary:

> The breathing of any dust leads to injury of the lungs, if it is intensive enough and continued long enough.
>
> With intensive and long-continued inhalation of dust, entirely quartz free dust leads to lung changes clearly recognizable in the X-ray picture. The blood vessels appear more clearly defined; there are also more numerous and thicker honeycomb formations, also infiltrated mottling which, however, never shows the very sharp delineation and density caused by quartz. The subjective symptoms referable to the changes are slight; there is none of the lung rigidity characteristic of silicosis but a complication of diseases, especially bronchiectasis and emphysema.

It is theoretically possible from the report that many of the occupational dusts breathed in immense amounts over a very long time lead to changes not recognized by us today.

In a discussion of etiology, pathology, and physical signs of silicosis Lovey (*173*) stated:

However, a lung filled with dust is a wounded lung and should be considered as subnormal in its vital resistance toward the onset of disease. A lung containing silica dust has a strong predilection toward the onset of tuberculosis. This susceptibility is unusual.

TERMINOLOGY OF DUST DISEASES

The following terminology of dust diseases of the lungs (*174*) is now used by most writers on the subject:

Pneumoconiosis.—A general term covering all dust diseases of the lungs, fibrous or nonfibrous (from Greek *pneumon*, lung, and *konis*, dust).

Silicosis.—A fibrosis caused by free silica (or quartz) and the best-known scientifically of the dust diseases of the lungs.

Silicatosis.—A type of fibrosis found after exposure to certain mineral dusts and assumed to be caused by various silicates. It is distinct from the sharply defined, coarse, nodular fibrosis caused by silica dust.

Anthracosis.—A dust disease of the lungs found in coal miners; it is ill-defined and is presumed to depend on inorganic dust in coal. The lungs are black.

Siderosis.—A term applied to a fibrosis of the lungs found in metal workers. The condition is ill-defined. The lungs are yellow or red from metallic oxides, generally of iron.

Asbestosis.—A fibrosis of the lungs with characteristic microscopical stigmata due to breathing asbestos dust, a silicate of magnesium.

In his bibliography on silicosis, Carozzi (*11*) called attention to the arbitrary nomenclature that has been applied to dust diseases for centuries. These diseases have been named at various times and by various authors, according to (1) the lesions observed, as fibroid phthisis, lung fibrosis, cirrhosis of the lung, and chronic interstitial pneumonia; according to (2) the cause, as dust phthisis, dust fibrosis, dust lung, and dust disease; and according to (3) occupation, as miner's phthisis, collier's lung or phthisis, miner's lung, grit fibrosis, gritty phthisis, grinder's rot or lung, grinder's disease, cutter's lung, stonemason's lung of phthisis, millstone maker's phthisis or disease, knapper's rot, stone hewer's dust lung, quartz lung, and phthisis of potters. Some authors instead of phthisis used the term "chalicosis" or "asthma," accompanied by the occupation.

PNEUMOCONIOSIS

The diseases resulting from inhalation of dusts generated in industry during the progress of certain processes are now known generically as pneumoconiosis, having been so named by Zenker, as mentioned previously. In a brief summary of information regarding certain of the pneumoconioses other than silicosis, Collis (*175*)

called attention in 1931 to some of the respiratory diseases that may be caused by dust but seldom are recognized as dust diseases. He classed the inorganic dusts as insoluble, soluble and harmful, and mixed. Under insoluble dusts he mentions that those from certain materials, such as basic slag and emery, an oxide of aluminum, are insoluble in the tissues. Inhaled particles fall on the walls of the bronchi and bronchioles, where they are entangled in secreted mucus and then swept back by ciliary action to be expelled finally in sputum. If the exposure to dust is excessive over a period of time, an inflammatory hyperemia of the walls results, with excessive exudation of mucus; finally the process extends beyond physiological elasticity; then degeneration and destruction of the ciliated mucosa occur; microbic invasion follows; and chronic bronchitis is established.

The process described by Collis is a reaction to the inhalation of all dusts not rapidly absorbed; hence, bronchitis stands out as chief of the dust diseases and during middle life causes much recurrent invalidity and incapacity and, after middle life, high death rates. Dust bronchitis cannot be distinguished clinically from bronchitis due to exposure at hot furnaces, fumes in industry, or severe climatic conditions; its association with dust inhalation, therefore, lacks recognition.

If the dust particles are small enough (5 microns or less) to be drawn into the finer bronchioles and alveoli, which are the seat of attack for pneumococci, a similar reaction is stimulated, the resistance of these parts to infection is lowered, and pneumonia results. Pneumonia is even less recognized as a dust disease than bronchitis; nevertheless, mortality records of those employed in dusty occupations are high. However when fine particles of insoluble dusts are carried by phagocyted dust cells from the alveoli into the lymph stream and lymph nodes of the lungs they tend to remain there as foreign bodies and do not provoke any particular tissue reaction.

Collis (*175*) said that the most-studied dust of the soluble and harmful dusts in this group is that of silica. The dusts of silicates follow; although many silicates, such as fire clay and pottery clays, appear to exert little if any harmful influence upon the lungs, recent work has shown that certain other silicates, such as basalt and asbestos, react injuriously on the pulmonary tissues. The reason for these differences is not clear; but probably it lies in the constitution of various silicates, the SiO_2 radical being less firmly attached to the molecule in some instances than in others.

Collis (*175*) defined asbestosis as a pneumoconiosis that advances to a fatal end without the supervention of any characteristic infection. It is a simple dust condition, just as is simple silicosis; but it is more distressing in life and more rapid in its progress than silicosis. It contrasts even more strongly with pulmonary mycosis, which is due to a living infection upon otherwise healthy lungs.

Regarding mixed dusts, Collis (*175*) said that, as long as any dust consists of only one substance, its influence can be isolated and studied; but the case is different when more than one substance is present in the dust. Coal dust, which recently has been carefully investigated (*176*), is taken as an instance in point. No undue mortality from respiratory

diseases was noted among coal miners when the coal worked contained little or no mineral matter and the miners were not exposed to other dusts, such as those arising from rocks intervening in the coal measures. The miners in the Nottinghamshire coal fields furnish a good example; the lungs of these men may be as black as the coal they work, but they remain resilient and free from fibrosis.

Gooding (*177*) stated tuberculosis which accompanies pneumoconiosis often lacks its usual features, and it is probable that more detailed investigation would reveal tuberculosis in a much greater proportion of silicotic miners.

In discussing the nomenclature of dust diseases Panconst and Pendergrass (*27*) stated that, although silica (SiO_2) seems to be the active fibrosing agent in most conditions recognized as essentially a pulmonary fibrosis, adherence to the general term "pneumoconiosis" still seems advisable. Some other dusts undoubtedly cause a fibrosis directly, and still others may either tend to retard the action of silica, as coal dust and clay, or enhance its action, as alkalies in the mixtures of silica and powdered soap in scouring powders. "Silicosis" is the term much more frequently used, mainly because it is not so unwieldy and usually describes accurately the particular phase of the condition under discussion. "Miners' phthisis" appears in the laws of South Africa but is otherwise an obsolete term generally recognized as implying silicosis or pneumoconiosis. There is now reason to believe that silicates also produce considerable fibrosis through their silica content or through a specific action of their own.

Panconst and Pendergrass (*27*) stated that the occupational terms are advisable because certain peculiarities have been attributed to the dusts of those industries, even though the condition still remains silicosis or pneumoconiosis. For example, in anthracosis the action of silica seems to be modified by the coal dust mixed with it; moreover, coal dust alone may produce a mild fibrosis. Collis and Gilchrist (*176*), in an investigation of coal trimmers working at the Cardiff docks, discovered that inhalation of large amounts of coal dust alone over a period of years produced evidences of mild pneumoconiosis which presented the same roentgenographic appearances as silica. It has been suggested that clay has a similar effect in protecting the workers in some of the operations in the pottery industry but not all of them. The term "chalicosis" has been applied in the past to pneumoconiosis of potters.

In recent years certain very unusual features in connection with pneumoconiosis among asbestos workers have brought the term "pulmonary asbestosis" into active use.

According to Haldane (*178*), evidence is accumulating constantly that scattered fibrosis, giving essentialy the same X-ray picture as early silicosis, is comparatively common as a result of excessive dust inhalation but without accompanying liability to tuberculous infection. However, he stated that this condition should not be called silicosis; since it seems to arise from excess of any kind of dust, not merely silicate dust, pneumoconiosis seems the most suitable name for it.

Silicosis and asbestosis are forms of pneumoconiosis that have been clearly defined clinically; the other forms of pneumoconiosis, such as anthracosis, siderosis, and many others, still lack complete definition.

Although considerable radiographic evidence has been accumulated on these latter types of occupational-dust exposures, corroboratory evidence has not been deduced on all of them from correlations of X-ray findings with pathological anatomy, chemical examination of tissues, and studies of the industrial environment (*179*).

The condition of the lungs giving a roentgen appearance similar to that of silicosis but caused by inhaling certain nonsiliceous dusts has been called (*180*) "benign" pneumoconiosis. Pendergrass and Leopold (*180*) found it impossible to differentiate the roentgen appearance of nodulation of silicosis or the pseudonodulation of benign pneumoconiosis from the shadows cast by many pulmonary diseases unassociated with the inhalation of dust. The diagnosis of the pulmonary lesions in such circumstances depends upon the collaboration of the internist, the roentgenologist and the laboratory technician. Pendergrass and Leopold (*180*) conclude that—

> To differentiate between silicosis and benign pneumoconiosis one must have a detailed knowledge of the occupational history, the environmental conditions of the worker and the precise information regarding the nature, concentration and particle size of the dust to which he is exposed.
>
> Advanced silicosis is usually disabling and, when complicated with tuberculosis, it is fatal.
>
> Advanced asbestosis produces disability and ultimately may induce death from cardiac failure.
>
> Benign pneumoconiosis produces nothing but shadows cast on a roentgenogram.
>
> We owe to the worker, to labor and to industry our utmost efforts to distinguish between these conditions.

The first case of lung cavity due to necrosis in a case of graphite pneumoconiosis was observed in 1946 by Dunner and Bagnall (*181*). In 1939 a worker aged 64, who had been working with graphite for 17 years, was pronounced tubercular and sent to a sanatorium. His sputum was always free of tubercle bacilli. He did not return to work with graphite but was able to do light work for 6 years. When first seen by the authors in 1944, the radiologic aspects of his chest were unchanged from those revealed on the films taken in and since 1939; they showed massive opacity in the right upper zone, similar, but fainter, opacity in the left upper zone, and a rounded opacity the size of a golf ball superimposed on the lower part of the left root shadow. In April 1945 he suddenly brought up large amounts of black material containing about 0.07 percent carbon. The size of most of the particles was 5 microns or less, with only a few over 10 microns. Silica was not detected.

SILICOSIS

DEFINITION

Rovida (1844–77), the first to mention silicosis in a publication (*182*), used the term in 1871 in describing "a case of silicosis of the lungs with chemical analysis."

According to Carozzi (*11*), however, Rovida stated in his article that—

> The name comes proposed for the first time by Achilles Visconti, prosector in the Ospedale Maggiore of Milan. (A "prosector" is an anatomist who makes dissections for anatomical demonstration.)

At one time it was believed that several substances could produce the lung condition called "silicosis," but it is now agreed rather generally that all types of dust fibrosis of the lungs are due to some form of the element silicon (*183*). The recognition that silicon was the important element in pneumoconiosis was a great step forward but, according to a statement by Kettle (*183*), the teaching that only one form of silicon—the oxide silica—can produce the disease was accepted too readily. The realization in recent years that asbestos can produce serious pulmonary fibrosis should have been a warning that this view was too restricted, as borne out by the work of Jones on sericite. However, the following definition adopted at the International Silicosis Conference in South Africa in 1930 (*184*) is still accepted:

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

SYMPTOMS

Inhalation of silica dust is seldom accompanied by evidences of irritation and if free from other irritating dusts may be breathed without arousing suspicion of its dangerous character (*119*).

The following discussion of symptoms of silicosis is based on the findings of 9,662 examinations of 7,722 mine employees at the Picher Clinic of the Bureau of Mines in 1928 (*99*).

Dyspnea.—Shortness of breath has been generally recognized as the cardinal symptom of silicosis. Dyspnea accompanies exertion in the earlier stages of silicosis and increases progressively until, in the later stages, it often prevents any labor. The apparent distress on exertion, frequently noted in silicotics who deny shortness of breath, seems out of proportion to the very slight rise in pulse rate taken 2 minutes after exertion.

Pain in chest.—Chest pains were admitted frequently, generally anteriorly, and more often on one side only. In many instances the pains seemed of a reflex character. In nearly all cases they were vague and flitting and did not seem to interfere with manual labor. Pains in the shoulder and possibly in the posterior part of the chest probably are considered by many of the men as "rheumatic"; such pains seem partly responsible for the large number of complaints of rheumatism.

Cough.—This symptom is frequently admitted in silicosis and generally is unproductive.

Expectoration.—This symptom was admitted infrequently. When present in uncomplicated silicosis the sputum usually is clear or has a bluish tinge and is of a viscid, tenacious consistence, very difficult to cough up. The color of expectoration may be due to the color of rock mined.

Hemoptysis.—Hemoptysis appeared to be more common among silicotics than was expected. In some instances, particularly in second- as well as in third-stage silicosis, fibrotic areas may mask a tuberculous lesion which remains unrecognized until it becomes an open lesion, when the sputum will show the tubercle bacilli or hemorrhage will call attention to the true condition.

Night sweats.—It seems probable that some of the admitted night sweats were not true pathological night sweats, but the relatively high incidence in the silicotic groups appears difficult to explain except on the assumption that at the time of the sweats, some active infectious process was in progress. This could be due to tuberculosis; it may have been due to a pyogenic infection or malaria, as 33 percent of the silicotics examined gave a history of previous attacks of malaria.

Loss of strength.—The loss of strength admitted by silicotics in most instances probably was connected with their dyspnea on exertion and was due to respiratory insufficiency. This probability is strengthened by the small number of silicotics with poor physical development (4 of a total of 1,062).

Gastrointestinal symptoms.—These symptoms were pronounced in the more-advanced cases, and loss of appetite was admitted. Other pronounced symptoms in advanced cases of silicosis were constipation, epigastric discomfort, and other vague complaints which the men termed "indigestion." The causes of the gastrointestinal symptoms were not ascertained.

Sayers (*185*) summarizes the clinical findings in silicosis as follows:

> The practically constant clinical signs are: A certain lack of elasticity of the chest wall during the movements of respiration, together with a somewhat reduced air entry, and a characteristic alteration of the inspiratory murmur from the normal "vesicular" character to a higher-pitched or "harshened", "thinned", and commonly somewhat shortened type, the expiratory murmur, although somewhat prolonged, remaining fainter than the inspiratory.
>
> This type of breath sound is very characteristic with some modifications, of silicosis in all its stages and, this clinical sign has also the significant character of more or less complete regeneralization. It is first noticeable at the anterior, lateral, and basal regions.
>
> In the minority of cases, however, the breath sounds may be simply diminished, but breath sounds which are simply diminished or which, on the other hand, are merely somewhat louder or more "pronounced" than normal are not specially characteristic of silicosis.
>
> Usually there are no accompaniments, but a stray rhonchus may be heard here and there.
>
> This complex of physical signs is almost constantly present in cases of a slight degree of "simple" silicosis. The cough may be put down to the coincident bronchitis and bronchiolitis, the recurrent pains to slight intercurrent local pleurisies.

PATHOLOGY

According to Panconst (*27*) the pathological features of pneumoconiosis are: (*a*) Entrance of dust; (*b*) "dust cell" or macrophage; (*c*) entrance of the dust cell into the lymphatic system of the lungs as a carrier of silica or other particles; (*d*) influence of the deposited silica in the production of fibrous tissue; (*e*) action of silica; (*f*) elimination of dust; and (*g*) predisposition of the fibrotic and silica-saturated lung to respiratory infections, especially tuberculosis.

The lung, as described by Gardner (*186*) is an organ that permits interchange of gas between the blood and the external air. As it is in free communication with the exterior, it is more or less exposed to the action of atmospheric impurities, both particulate and gaseous. Certain mechanisms protect the lungs from the accumulation of such foreign particles. The nose, through which the respiratory tract opens to the surface of the body, is guarded by a coarse filter of hair.

Behind this is a series of tortuous passages with moist walls which trap many smaller particles. In addition, the nasal cavities and the remaining portions of the upper respiratory tract, the pharynx, the trachea, and the bronchi are lined by cells covered with minute vibratory hairs, and cilia. Wavelike vibrations of these hairs tend to carry particles lodging on their surface away from the lungs and back toward the surface.

Particles that succeed in passing these barriers and penetrate to the terminal air spaces of the lungs are ingested by wandering scavenger cells or phagocytes, which come from the partitions between the spaces for the purpose. These cells move independently and tend to carry the foreign particles out of the air spaces into a special drainage system known as the lymphatics. The lymphatics are minute vessels which drain into sedimenting basins known as lymph nodes. They are situated along the course of the vessels and bronchi and at the root of the lungs where the trachea divides into the two main bronchi. For ordinary amounts of atmospheric pollution, these protective mechanisms are adequate to prevent significant accumulation of foreign particles in the functional part of the lungs. If a person continues to work in a very dusty atmosphere for long periods his protective devices cannot cope with the situation, the mechanisms themselves are damaged, and the dust particles collect where the air should be (*186*).

Belt and King (*187*) visualize the sequence of events as phagocytosis, inflammatory change, organization, and reticulin fibrosis. Each is characterized by a distinctive morphological feature: (*a*) Phagocytosis by the koniophage; (*b*) inflammation by fibrin; (*c*) organization by the fibrocyte or dictyocyte; and (*d*) fibrosis by reticulin. One important cellular element, the foreign-body giant cell, is often present throughout all four stages.

All dust cells are phagocytosed in the parenchyma of the lung. The principal cell involved in this activity is a large wandering, amebic, mononuclear cell which Belt and King (*187*) have designated as the koniophage but which is also known as dust cell, endothelial cell, macrophage, or histiocyte. Particles up to 10 microns in size are dealt with almost entirely by this type of cell. The foreign-body giant cell usually takes care of the larger particles.

According to Kasten (*188*), coal dust is phagocytized more quickly and more completely than stone dust. Quartz causes violent changes, and vacuoles develop. Mature vacuoles unite to form one large one, filling the complete cell at last. The smaller the quartz particles, the more quickly and completely the process develops. In contrast to this, cells that have taken up coal dust do not show vacuole formation in such a degree but do show fatty degeneration. Kasten (*188*) concluded that the malignity of quartz is caused by its chemicophysical effects on the tissues ("surface effect").

Definite inflammatory change is indicated by the presence of extravasated fibrin. Commonly, it is mingled with the dust-laden koniophages in small shreddy deposits and accompanied by a little fluid exudate and occasional granulocytes. This initial inflammation plays an important part in determining subsequent changes. It is somewhat unpredictable and capricious in incidence. The quality of the dust has something to do with it. The incidence of initial inflammatory

change is roughly proportional to the silica content of the dust and roughly inversely proportional to the coal content. Exceptions to both these rules are frequent (*186*).

According to Simson (*189*), if the first line of defense is passed and particulate matter reaches the alveoli, the cells lining the alveoli are stimulated to activity. They swell, become detached from the walls, and develop an active phagocytic function. The particles are phagocyted and are then carried in one of two directions; some of the phagocites, now laden with dust, pass to terminal bronchioles, whence they may be removed in the sputum; others pass into the walls of the vestibules and terminal bronchioles. In time, large numbers of dust-laden cells accumulate and cause condensation of the tissues about the entrance to the primary unit. During the period of excessive dust inhalation and possibly for a short-time afterward, dust cells are continually reaching and entering the areas of condensed tissue at the entrance to the primary unit; others leave the condensed areas and enter the regional lymphatic vessels, along which they pass to the minute masses of lymphoid tissue which lie between branches of the pulmonary artery and the adjacent bronchioles, vestibules, and atria. Dust cells stimulate the lymphoid tissue and cause it to become hyperplastic. With continued arrival and accumulation of dust cells in the lymphoid masses, more or less well-defined aggregations are formed. The site of an aggregation can retain only a limited number of dust cells, so that with new arrivals an overflow results. The cells comprising the overflow and those that have escaped arrest pass onward in the peribronchial and perivascular lymphatic vessels, finally being trapped in the lymph nodes at the root of the lung. In silicosis these lymph nodes are the first sites to show fibrosis.

As late as 1947, Policard (*190*) stated that the exact mechanism of this accumulation and its physiopathologic consequences are not well known, although the phenomenon of the accumulation of dusts in the connective tissues of the pulmonary arteries and the bronchi is constant and indisputable.

He claims that, in a normal lung, the dust cells are rapidly and easily carried away by the lymphatics during the periods of beginning pneumoconiosis. There is never any permanent accumulation of dust in the arterial or bronchial tissues. There is an accumulation of dust only when conditions have become abnormal, especially if there is stasis of the lymph—for example, as a result of ganglion lesions down stream. The lymphatic current is then slowed and the dust cells immobilized.

By accumulating in the lymphatics of the tissues, the dust cells obstruct their openings, as vast as they are. The course of the lymph, already slowed before the accumulation, is impeded still more, perhaps arrested completely. From this arises an important consequence—edema of the tissues. Edema favors fibrosis. It disturbs the functioning of the tissues of the pulmonary lobes by making them rigid and abolishing their elasticity. Policard (*190*) thinks that this edematous process is not well-known and deserves more attention.

At certain points the tissues are fibrosed. There is an increase in their collagenous and reticular deposits (precollagenous). This fibrosis is irregular. A segment of about 1 to 3 mm. appears fibrosed,

with two apparently normal segments of tissue interposed between. This irregular disposition of the fibrosis in a tissue where the accumulation of dust on the contrary seems regular speaks against a direct fibrosing action of mineral particles. If they intervene in the processes it is in indirect fashion, still not specified.

Policard (*190*) refers to the British statement that fibrosis of the pneumoconioses is due to fibrils of the reticulum. In fact, the large fibers show the reactions of the collagens and the finer ones those of the reticulum. The latter are always abundant but appear to be simply collagenous fibrils in the process of formation, that is, "precollagenous" fibrils. In all the cases it cannot be a question of fibrosis exclusively due to the fibrils of the reticulum.

Another fact to which Policard (*190*) calls attention as little known is the calcareous (and probably phospho-calcareous) impregnation of the tissues having accumulated mineral dusts. This impregnation is revealed by micro-incineration. This is not a calcification. In particular, it does not increase the hardness of the tissue. The lime is on this level, not in the solid and crystalline form, consequently rigid and hard, but in the molecular form combined in protein complexes. The distinction is essential.

Policard (*190*) states that one consequence of this impregnation is important for medicine, that a normal tissue is radiotransparent. Coal or silica dust also is transparent. A tissue charged only with dusts does not give any image on the radiographic films; but if one has been impregnated by lime, the radio-opaque lime causes the tissue to become opaque, which leads to radiographic images. In particular, the aspect called "reticulation" seems to have to be connected with calcareous impregnation of sclerosed and hypertrophied tissues. The mechanism of this impregnation has not been determined but it undoubtedly exists.

If inhalation of injurious dust other than silica—for example, asbestos dust— is continued over a long period or over a shorter period when the concentration of dust is great, a diffuse, cellular fibrosis develops in the walls of the bronchioles, vestibules, and atria. The fibrosis tends to extend locally, involving the supporting connective tissue of the adjacent blood vessels and to some extent the walls of the alveoli in the immediate neighborhood. There is usually desquamation of the epithelial lining of bronchioles and vestibles, often without evidence of a definite exudate. In very advanced cases the fibrosis may extend to the septa, to the supporting tissues of the bronchi and larger blood vessels, and to the alveolar walls. Even in these cases fibrosis is localized to the supporting tissue about the entrance to the primary unit. It may advance to such an extent that the bronchioles become constricted, sometimes retaining their circular outline in cross section and sometimes being reduced to mere slitlike openings. Rounded nodules of cellular character occasionally are seen, but the dense hyaline type of nodule usually is absent. Dust containing only a small percentage of silica also may produce these changes, and the liability to generalized fibrosis is greater when a low-grade type of infection (not necessarily of tuberculous origin) complicates the dust effect.

Simson (*189*) said these changes appeared to be primary lesions caused by inhalation of any injurious dust, but when most of the inhaled particles are composed of or contain silica a specific and localized type of fibrosis—the silicotic islet—also develops. The first evidence of the specific and localized lesion, which Simson (*189*) termed the "silicotic islet", is seen in the aggregation of dust cells that probably represents the site of the lymphoid mass. A small, round area of fibroblasts appears in the center of an aggregation; on further development a central core of dense, fibrous tissue is formed. It often becomes hyaline in character and in larger and older nodules takes on a whorled arrangement. A fully formed, single silicotic islet consists of a central mass of dense, hyaline fibrosis arranged in whorls and surrounded by a comparatively narrow zone of cellular fibrosis in concentric laminae. Scattered through this mass, lying between connective tissue cells and fibrils, are scanty, large and small round cells and a little particulate matter. A deposit of fat may be seen also in suitably stained sections, the degree varying with the age of the nodule. The early fibrotic nodule thus formed is surrounded by numerous dust cells. The growth of the silicotic islet continues by successive new arrivals of dust cells and subsequent extension of the fibrosis at the periphery of the fibrotic nodule. Apart from the individual single or composite palpable islet, noninfective silicosis may appear as a "massive" type of fibrosis. Seen macroscopically, such lesions comprise numerous contiguous single and composite islets. The fibrosis is of the same character as that of the single islet; there is no evidence of breaking down. The intervening alveolar tissue is much compressed and collapsed, but there is no evidence of inflammatory infiltration or of definite matting together of the individual islets. As a rule, the larger blood vessels are not constricted.

These changes, according to Goodrich (*120*), make the lungs less elastic, increase their bulk, make aeration of the blood less efficient, and decrease circulation through the lungs. As a result, a more vigorous respiratory effort is necessary to supply the blood with an adequate amount of oxygen and to remove the accumulated carbon dioxide in the blood. An increase in inspiratory effect is needed to create greater negative pressure within the chest in order to draw the same amount of air into the less elastic lungs, and an increase in the normal tidal volume is needed because of the decreased aeration of the damaged alveolar structures and the impaired circulation through these alveoli. As the volume of the lungs increases from the continuing deposit of silica and the increasing amount of fibrous tissue, the chest gradually is held and later fixed in a position of partial inspiration, and breathing becomes more and more diaphragmatic. Even diaphragmatic breathing eventually becomes less efficient because of the usual development of a basal pleurisy which causes the inelastic lungs to adhere to the upper surface of the diaphragm. Secondary infection with bacteria may cause still greater lung damage. Chronic bronchitis or bronchiectasis may be caused by streptococcus, staphylococcus, pneumococcus, or other pyogenic organisms, as evidenced by investigations of the United States Public Health Service (*191*) on the development of pulmonary infections in pneumoconiosis. The results of the bacteriologic and experimental study show that—

In general, the silicotic lung is more susceptible to bacterial infection than the average lung. This is probably due to the irritation of the respiratory tissues by the inhaled dust particles which weakens the mucous membranes and renders them susceptible to infection. The toxic influence of certain inorganic dusts upon the tissues may be a contributing factor.

The relation of tuberculosis to pneumoconiosis has been studied to a considerable extent, but comparatively little work has been done in connection with other infectious processes of the lung, for example, pneumonia, lung abscess, bronchiectasis, and influenza. An investigation was made of these conditions, both bacteriologically and experimentally, with the view of obtaining a better understanding of the predisposition to, and the mechanism of, infection of the lung in certain dusty trades.

Bronchiectasis, lung abscess, and gangrene occur frequently in hard-rock miners. It has been definitely established that aerobic, pathogenic bacteria, and certain fungi are responsible for these conditions, but the high percentage of cases in which the anaerobic microbes of the mouth and throat have been reported would suggest that they at least participate in the etiology of the diseases.

According to Tillson (*192*), many of the phagocytes of the lungs, when carrying mineral particles, become trapped in the lymph channels and nodes and therefore are not eliminated. Those free in the blood stream are charged positively at the hydrogen-ion concentration of the blood and move to the cathode in an electric field. Bacteria carry a negative charge and therefore are attracted to the phagocytes, are adsorbed, and then ultimately absorbed. The agglomeration of bacteria increases the efficiency of this process, as the large phagocyte cell can dispose of many bacteria in one contact. This clumping of bacteria depends upon the plasma salts being at a high enough concentration; otherwise, bacterial absorption becomes a slow process.

Silicosis is a progressive disease. If enough silica has been inhaled to initiate nodule formation each focus continues to enlarge until a state of equilibrium is established. Nodules not visible by X-ray when a man leaves the silica industry may increase enough in size to be readily detectable some years later. The progression of the process is favored and accelerated by the development of pulmonary infection (*186*). In fact, many pathologists doubt whether the fibrosis of the lungs that constitutes the essential pathological nature of silicosis ever arises independently of a primary infectious process (*193*). Rist, Mayel, and Donbrow (*194*), after studying the question from the medicolegal point of view, were convinced that silicosis cannot develop other than in persons already infected with the tuberculous virus and hence refuse to regard silicosis as a true occupational disease.

Attentive and long-pursued clinical observation of cases of pulmonary fibrosis led Croizier, Martin, and Policard (*195*) to doubt the practical existence of an essentially pure silicosis. They cited as clinical anatomopathologic proof the fact that almost always tubercular infection intervenes in cases of silicosis. They considered the term "silicotuberculosis" very exact for expressing the combination of tuberculosis and silicosis. Their position on this subject was as follows:

The question has been posed as to whether in this instance it is the silica that begins or indeed the tuberculosis. In our opinion the primary intervention of the tuberculosis appears probable. The fibroses that we have observed represent bacillosis modified by the silica more than tuberculorized silicoses. The fact that miners have passed 10, 20, and more years in the siliceous dust and have

remained free of pulmonary silicosis is proof that silica alone cannot create these fibroses in a normal lung.

According to Gardner (*196*), only a limited number of individuals ever contract pure silicosis. Most of the hazardous occupations involve exposure to mixed dusts, and the pulmonary process that develops consequently is a silicosis modified by other less-active constituents.

In some cases the infection remains latent, presents none of its usual manifestations, and merely modifies the character of the reaction to inhaled silica dust. In others it develops simultaneously with the silicotic reaction with a more or less typical localization in the upper part of the lungs. Such cases may exhibit none of the characteristic symptoms of tuberculous intoxication for many years and often are discovered only in routine roentgenographic examinations of large groups of active employees. Ultimately these men develop symptoms, expectorate tubercle bacilli, and finally die of tuberculosis, but the course of their disease is protracted. There are still others whose roentgenograms show so much evidence of tuberculosis that the characteristic features of silicosis are obscured. Nevertheless, they have long been exposed to dust, and post mortem examination will reveal the nodular fibrosis produced by silica. Like those of the preceding group, these cases run a definitely chronic course and may not die of their infection until the fifth or sixth decade. Finally, there is another group, with well-developed nodular silicosis, whose members apparently have never had a tuberculous infection. Such men become infected, develop a rapidly progressive tuberculosis, and die within 6 months. In them the symptoms of intoxication are more acute, but bacilli may be very difficult to detect in their sputa and even in the lungs removed at autopsy (*136*).

Gardner claimed (*186*) there is no difficulty in demonstrating that silica produces a reaction in the body specifically favoring multiplication of tubercle bacilli. Kettle injected a definite quantity of fine silica particles beneath the skin of one flank of a white mouse and in the opposite flank the same quantity of aluminum oxide particles. A large dose of tubercle bacilli was then injected into the tail vein of the animal. The blood distributes these bacilli quite uniformly to all parts of the body, but if the animal is killed after several days large masses of them will be found at the site of the injected silica. The bacilli are no more numerous where the alumina particles have localized than in any other part of the body. Apparently the reaction induced by the silica produces a favorable medium for the growth of these bacteria, but they disappear later and may be very hard to find. The same seems to be true of the sputa of men with silicosis and tuberculosis.

Gardner (*186*) reiterated that only silica and a limited number of the silicates are known to produce definite and serious pulmonary damage. In the case of silica, this is associated with specific indisposition to tuberculosis and pneumonia. Of the silicates, asbestos is definitely recognized as a cause of pulmonary fibrosis. Its importance in predisposing to tuberculosis is not yet settled.

Apparently the "specific indisposition to tuberculosis" mentioned by Gardner is not always present in silicotics; Gudjonsson (*197*) found

that tuberculosis was rather rare among granite and sandstone workers in Denmark; of 60 workers with silicosis, 4 had old calcareous foci, and none had active tuberculosis as far as could be determined by X-ray; 16 of 218 workers without silicosis showed old tuberculous processes, indicating that tuberculosis is equally frequent in both groups. He stated:

> This result is in accordance with results from earlier investigations regarding silicosis in this country, that is, that tuberculosis is no more frequent among those suffering from silicosis than among those not so afflicted; according to this, tuberculosis might seem to have no direct relation to silicosis. This finding is in contradiction with many investigations from other countries, but it is none the less a fact.

At the International Conference on Silicosis (*184*) it was agreed that the microscopic pathological changes that may be produced by the prolonged inhalation of silica dust are:

> (*a*) The development of a condition designated in South Africa as a dry bronchiolitis, characterized by an accumulation of dust-filled phagocytes in or in relation to the terminal bronchioles, with possibly some desquamation of their epithelium.
>
> (*b*) The accumulation of dust-containing phagocytes about and in the intrapulmonary lymphoid tissue and their transportation through the lymphatics into the tracheobronchial lymph nodes. (The conditions described above under (*a*) and (*b*) do not constitute the disease silicosis.)
>
> (*c*) The gradual development of fibrous tissue within such accumulations of phagocytes and the formation of characteristic nodules of hyaline fibrous tissue.
>
> (*d*) Degenerative changes in these foci.
>
> (*e*) The hyaline nodules increase in size by extension of their periphery. Coalescence of adjacent nodules takes place and brings about involvement of further areas of the lung. (The conditions described under (c), (*d*), and (*e*) constitute the disease silicosis.)

Microscopically, the changes observed in silicosis are:

> (*a*) *In the early stage.*—A variable number of palpable pearly-white nodules up to 2 or 3 mm. in diameter on the pleural surface of the lung. On section, the cut surface of the lung is studded with pigmented foci, widely scattered, a moderate proportion of which are only just palpable. The tracheobronchial lymph nodes are slightly enlarged and deeply pigmented and may exhibit foci of fibrous induration.
>
> (*b*) *Later stages.*—The fibrotic nodules are increased in number, size, and density, and coalescence of these may be found. The portion of the lung between the fibrotic nodules may be emphysematous. The tracheobronchial lymph nodes may be smaller in size than those seen in the early stage and are fibrosed.

It was also agreed that the presence of tuberculous infection usually modifies the pathological appearance, and special attention was drawn to the following three types:

> (*a*) In which the picture of silicosis above described may be little, if at all, modified, but in which only a biological test can demonstrate the presence of *B tuberculosis*.
>
> (*b*) In which the coexistence of silicosis and typical tuberculosis lesions is easily recognizable.
>
> (*c*) In which the presence of tuberculosis is easily recognizable, but the existence of silicosis is more difficult to determine.
>
> In *massive* silicosis cardiac hypertrophy and subsequent dilatation may occur. In silicosis with infective processes cardiac changes may also occur. No evidence was adduced in regard to involvement of kidney or liver.

Belt and King (*187*) presented, as a result of their experimental studies on chronic pulmonary disease in South Wales coal miners, the following criteria as a basis for judging the dust effect in the animal

lung: (1) Initial inflammatory results; (2) effective retention of dust; (3) connective tissue reaction; (4) density of reticulin fibrosis; and (5) distribution of lesions. A summary of their detailed explanation of these criteria follows:

(1) Excluding complicating factors, *initial inflammation* is indicative of an immediate irritant action on the part of the dust. It is important also as the determinant of the type of organization to follow and therefore has a considerable influence on the end result. Except in a few cases, probably complicated by infection, the initial reaction showed no great fluctuations in intensity. No accurate means were available for assessing the extent or severity of inflammation in the animals surviving the initial reaction (and this includes the great majority).

(2) *Effective retention* is defined arbitrarily as permanent storage of dust in the fixed tissues of the lung, in contradistinction to casual retention, in which the dust is lodged in potentially mobile, loose-lying cells without engaging the fixed tissues. Effective retention is brought about only when dust deposits are overrun and secured by connective tissue, a process which is conveniently referred to as organization of the dust.

(3) *Connective tissue reaction.* Organization of dust deposits proceeds in one of two ways, or sometimes as a mixture of both: (*a*) As a foreign-body reaction, or (*b*) as an organization of inflammatory exudate hereafter referred to by the authors as "*carnification*". According to which of these characterizes the reaction, a dust is classified as of less or greater pathogenicity.

(4) *Reticulin fibrosis*, as opposed to collagen fibrosis, measured in terms of reticulin fibrils, constitutes the most important single criterion of pathogenicity. Reticular tissue is the histological basis of coal miners' pneumoconiosis and it assumes a similarly fundamental role in experimental pneumoconiosis, thereby affording a significant basis of comparison between the two.

Whether in animals or in man, the *density of reticulin reaction* produced by any given mineral dust is the best available histological index of its chronic harmfulness.

(5) *Distribution of lesions* provides a further guide to pathogenicity. The less noxious dusts, producing only the simplest of foreign-body reactions, tend to be dispersed into smaller and smaller aggregations, which become more and more widespread as time goes on. This process may be regarded as a form of resolution, by virtue of which the foreign material is fitted into the structure of the lung, presumably in such a way as to produce a minimum of embarrassment to respiratory function. More harmful dusts, on the other hand, tend to retain a nodular distribution, and sometimes coalescence is carried to the point of producing large confluent lesions. Coalescence may be evidence of an inflammatory complication—for example, pneumonia.

Fisher (*198*) has outlined rather clearly the process of acquiring pneumoconiosis as follows:

> When breathing takes place in an atmosphere containing an abnormal quantity of dust, a certain amount of this dust is filtered off by the nose; much of the remainder, as the air passes down the windpipe and bronchial tubes, settles on them, while some may reach the terminal air-sacs. The bronchial tubes, in the process of branching, become more and more narrow so that it is only the finest

dust which reaches the air-sacs (alveoli) of the lung. The inner part of the bronchial tubes is provided with a lining of tiny fingerlike projections (ciliated epithelium) which bend over with rapid whiplike movements, the whole layer acting like an ever-moving conveyor to help to carry the dust cells mixed with mucus upwards towards the throat. By "dust cells" is meant scavenger cells or "phagocytes" as they are called. These scavenger cells take up the dust which impinges on the walls of the air-sacs (alveoli).

The dust laden cells may either (*a*) return to the bronchial tubes, (*b*) migrate along the supporting fibrous framework of the lung and lymphatic channels, or (*c*) stay in the lobules or air-sacs.

In order to avoid dust accumulation in the lung, the rate of ridding the lung of dust must be greater than the rate of dust entry. In the ordinary conditions of life this is so; it is only when working conditions expose the individual to abnormal concentrations of dust over prolonged periods that the rate of elimination falls behind the rate of entry and pneumoconiosis develops.

It is generally accepted that the lungs cannot rid themselves of all kinds of dust with equal ease.

In the case of silica, a larger proportion of the dust which is breathed into the lungs seems to stay there than appears to be the case with other dusts. Thus a comparatively small daily exposure may, in time, accumulate sufficiently to bring about the condition of simple silicosis. The silica dust tends to be held up in the lobules and alveoli, probably in relation to the lymphatic system of the lung, and this is evidenced as small hard lumps symmetrically dotted about the lung. This condition is known as nodulation and the nodules, as they are called, show, when cut across, a concentric formation. These fibrotic nodules, with an increase in the fibrous tissue in their neighborhood, destroy the elastic quality of the lung, and this, together with other factors, accounts for the breathlessness which is the most prominent and constant symptom of the disease. At this point I must bring in the word emphysema. When speaking of silicosis or pneumoconiosis this term means the unnatural distention and rupture of air-sacs of the lungs. The air-sacs, having lost their elasticity, do not expand and contract normally. They increase in size and the dividing walls rupture, forming larger distended areas and lung ventilation is lessened.

The formation of hard small nodules is characteristic of simple silicosis, but before the nodules are clearly formed there usually develops a general increase of fibrous tissue in the lung and to this is given the name "arborisation." As one writer put it, "first you have the tree with naked branches, then berries appear on the branches." This brings up to the condition of pneumoconiosis among coal miners called "reticulation."

Reticulation is the term given to an X-ray appearance which usually shows a fine and evenly distributed network of shadows. The network is finer than that of arborisation. The question often discussed is whether "reticulation" in the coal miner is simply a precursor of a nodular condition as in arborisation in the Rand miner or an entity in itself. We need not pursue the matter here except to note the important fact that before the coming into operation of the Coal Mining Industry (Pneumoconiosis) Compensation Scheme, 1943, compensation would not be paid if reticulation only existed and the nodules were absent.

STAGES

The Miners' Phthisis Act of 1925 of the Union of South Africa (*199*) defined silicosis as follows:

> *For the purposes of this Act . . . the expression "silicosis" shall mean silicosis* of the lungs. A person shall, for the purposes of this Act, be deemed to have or to have had silicosis
>
> (*a*) in the *ante-primary stage*, when it is found by the Bureau that the earliest detectable specific physical signs of silicosis are or have been present; whether or not capacity for work is or has been impaired by such silicosis;
>
> (*b*) in the *primary stage*, when it is found by the Bureau that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been impaired by that disease, though not seriously and permanently;
>
> (*c*) in the *secondary stage*, when it is found by the Bureau that definite and specific physical signs of silicosis are or have been present, and that capacity

for work is or has been seriously and permanently impaired by that disease, though not seriously, and when it is found by the Bureau that tuberculosis is or has been present.

Later, in view of the greatly altered aspect of the disease in South Africa (*200*), it became necessary to recognize additional groups of cases. At the meeting of the First International Conference on Silicosis at Johannesburg in August 1930, the three clinical stages of silicosis recognized locally were accepted as follows:

First stage.—*Respiratory symptoms slight, few or no physical signs and capacity for work little impaired. Roentgenograms show the linear shadows increased and present discrete shadows of nodulation.*

Second stage.—All physical signs are increased. The nodular shadows are increased in number and size and show a tendency toward confluence.

Third stage.—All signs and symptoms are greatly accentuated, and there is a total loss of working capacity. When tuberculosis is present, the stage classification must be based more or less upon a loss of working capacity than upon physical signs and roentgenographic appearances.

It was recommended that an internationally comparable roentgenographic technic and terminology be adopted, and that a further study be made of the correlation of roentgen appearances, pathology, and symptomatology of silicosis with or without tuberculosis.

The Committee on Pneumoconiosis and the Committee on Standard Practices in Compensation of Occupational Diseases of the American Public Health Association in a report on silicosis (*201*) described the stages of the diseases as follows:

The disease is divided arbitrarily into first, second, and third stages for convenience of description and possible compensation purposes.

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

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

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

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

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

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

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

Pancoast and Pendergrass (*202*) questioned the wisdom of designating any appearance of pneumoconiosis by any term denoting numerical stages of progress. They prefer to designate the appearance by a term which implies its pathological nature:

Now this appearance of prominent hilum shadows and increased prominence of trunk shadows and linear markings, with or without the faint haze, has in this country at least been designated as the *first stage* of silicosis or pneumoconiosis. There may be some excuse for continuing to call this the first stage, but continued experience with cases of pneumoconiosis developing in various industries has led us to question the wisdom of designating any appearance of pneumoconiosis by any term denoting numerical stages of progress. * * * We prefer to designate the appearance by a term which implies its pathological nature and to call it not a stage but the *perivascular-peribronchial-lymph node* type of preponderance of the condition. * * *

In cases with silicosis which is moderately slow in its progression the microscopic nodular lymphoid deposit proliferations tend to become conglomerate and to produce a macroscopic nodular process * * *. / this appearance was found as a more advanced stage of silicosis in the earlier cases examined, and especially among miners in this country, it was and is still called the *second stage* of the condition. It is conspicuous by its absence or insignificance, however, in many industries, notably the granite cutter, sandblaster, sandstone and asbestos workers. It is especially likely to be absent or inconspicuous in those who are developing silicosis rapidly, such as in those working with pulverized sand and without adequate protection.

If this appearance is absent or nearly so as a stage of progress in so many industrial silicoses, why designate it as a numerical stage of progress at all? We have ceased to do so and have called this appearance the *nodular type* or preponderance of silicotic fibrosis, or, more correctly, silicosis.

There is another type of silicotic appearance which was very puzzling to us at first because of its exact identity and our inability to find the proper place for it in a numerical classification of progressive stages. * * * Fortunately a definite pathological place seems to have been found for the condition and a roentgenological classification for the appearance. * * * We have designated this as the *interstitial type of predominance* and have subdivided it into rapid and slow, which subclassification must depend largely upon the physical factors and time of exposure. * * *

This type of fibrosis will almost invariably progress to the terminal stage with little or no appreciable nodular appearance. Care must be exercised in differentiating it from a chronic interstitial lung change resulting from pneumonia or continued upper respiratory infection as in the sinuses. * * *

The condition responsible for this appearance in advanced form and of rapid development has been termed "acute silicosis" by Chapman and some others. * * *

There are few, if any, arguments to be recorded in connection with the terminal stages of silicotic fibrosis. Instead of the *third stage*, we have preferred to call this aspect of the disease the terminal *diffuse fibrosis*. It is more or less incapacitating in practically all instances, and all too often completely so.

With respect to the classification of silicotic appearances based upon known pathological changes, Pancoast and Pendergrass (*202*) suggest the following in place of the old numerical stages:

1. Peribronchial-perivascular lymph-node predominance or type. This may be rapid or slow, usually the latter.
2. Early interstitial predominance. This may be extremely or moderately rapid, depending on the silica intake. It may or may not have an associated slight nodular appearance.
3. Advanced interstitial predominance.
4. Nodular predominance. Rapidly or slowly progressing.

5. Advanced diffuse or terminal fibrosis. Conglomerate nodular type. Interstitial type. Massive fibrotic type.

DIAGNOSIS

According to the Silicosis (Medical-Arrangements) Committee of Great Britain (*172*) silicosis can be diagnosed, even in its early stages, but the diagnosis is not free from difficulty. For instance, the clinical signs of silicosis resemble to a great extent those of other chronic diseases of the chest; further, silicosis may be complicated by the presence of tuberculosis in any stage. Irvine (*203*) says that it is impossible to deduce a practical standard of diagnosis from accounts of the disease furnished separately by the pathologist, the radiologist, and the clinician. Clinical examination is obviously an essential factor in the decision in any individual case, because it provides important information otherwse not obtainable regarding the general and local condition of the patient, the degree of incapacitation, if any, and the presence or absence of complication by active infection or by disease of other organs; but in a disease like silicosis clinical evidence in many cases is inconclusive and may be misleading. Other means of diagnosis in the living subject are radioscopy, radiography, and in the last resort the pathological condition found after death. Radiography also has its limitations; on the one hand, not all forms of pulmonary fibrosis revealed by it, even in miners, are of silicotic origin; and, on the other hand, certain types of radiograph which do not show unequivocally "specific" signs of silicosis may under certain circumstances be regarded legitimately as affording evidence of the presence of a silicotic factor in the case. Accurate knowledge from which to formulate a reliable general standard of diagnosis can be obtained only by correlation of the results of pathological observation in a large number of cases with radiographs taken from the chests of the same individuals shortly before death and with the results of clinical examination made during life. The final adjudication in any individual case must always incorporate the additional evidence supplied by expert clinical examination, which indeed is frequently the deciding factor, particularly in borderline cases.

Sayers (*185*) included under "clinical manifestations" those factors that can be ascertained from the individual having the disease—personal data, past history of diseases, occupation, present history, physical examination, and roentgen and laboratory observations. Levy (*173*) believed that any diagnostic medium in the early stages of silicosis should not be overlooked. The importance of the serologic examination and correlation of the physical examination with the past history is so great as to render all the steps of the complete examination of almost equal importance. Although some claim that no diagnosis of silicosis can be made properly without roentgen-ray examination such examination must not be considered adequate, except in advanced stages. The examinations at the Bureau of Mines Picher Clinic (*89*) included collection of data on race, family history, personal history, past history of illness, occupational history, and physical, X-ray, and laboratory examination. A brief summary of some of the results of the examinations follows:

Family history.—The incidence of silicosis or silicosis complicated with tuberculosis was found to increase as the family histories of the men were classed as good, average, or poor.

Personal history.—The use of tea, coffee, or tobacco apparently has no relation to silicosis or tuberculosis. After silicosis has developed, however, the use of alcoholic drinks and patent medicines increased markedly; the men take these to alleviate the symptoms of silicosis so that they can continue work.

A study of age groups in published results of several investigations on silicosis shows that, in the early years of the occupational life of those in dusty occupations, the incidence of silicosis is low; but, with increase in the age group, regardless of length of employment, it increases slightly up to about the age of 40 and thereafter markedly. Age, therefore, has an important bearing on the occurrence of silicosis and should be considered in employing men for work in dusty atmospheres.

No correlation could be made between silicosis or tuberculosis and overcrowding in sleeping accommodations. The number of hours of sleep bore no apparent relation to silicosis or tuberculosis.

Past history of illness.—The data seem to indicate that all infectious diseases of childhood, except diphtheria and scarlet fever, probably decrease resistance to tuberculosis and silicosis. Picher is free from malaria, but many of the men reported they had had malaria; among these men the incidence of tuberculosis and silicosis was comparatively high. Tonsillitis, especially if the attacks are recurrent, seems to be associated with tuberculosis but apparently has no relation to silicosis. The highest incidence of silicosis or tuberculosis, or both, was among men who reported bronchitis, pleurisy, and asthma. The lowest incidence was among those who reported influenza, pneumonia, coryza, and hay fever. An unknown and disturbing factor in the attempt to discover a relationship between silicosis or tuberculosis and other diseases was the difficulty in determining in many instances whether they occurred before or after silicosis and tuberculosis had been contracted. The data show that the incidence of physical defects increases among men with silicosis and tuberculosis. Several explanations are offered for this fact: Men with silicosis or tuberculosis usually are somewhat older than those in the essentially negative group; the silicotics have had a much longer period of service in the mines than the negative subjects; certain physical defects may increase susceptibility to silicosis.

Nasal obstruction and chronic catarrh, enlarged turbinates, adenoids, or other conditions that cause mouth breathing are associated with an increase in the incidence of silicosis. Persons having a vertical-hanging heart are more likely to develop tuberculosis or already have it; this finding agrees with South African experience.

Occupational history.—A detailed statement of occupation is the most important single item in the history of silicotics. The mere statement that the man is a miner has slight value, if any. Many underground occupations expose the workmen to very little dust, especially in well-operated mines. The Picher histories included occupation before mining, past occupation underground, number of years in present

occupation, kind of mineral mined, and total years in metal mines. All examined were divided into two groups according to duties—those exposed to large amounts of dust (those employed at the face) and those exposed to relatively small amounts (those away from the face). The data indicate that the men in certain positions contract the disease more quickly than those in other positions.

With the development of first-stage silicosis, there was a drop in the number of weeks and shifts worked, and with second-stage silicosis there was a marked drop in amount of work performed on each shift. Men with silicosis and tuberculosis worked intermittently during 15 weeks of the year, while third-stage silicotics worked part of only 9 weeks; the number of shifts worked by these two groups, however, was almost the same (54 and 52, respectively).

Physical examination.—No marked symptoms of ill health, such as paleness, loss of subcutaneous fat, or decrease in muscular development, appear until silicosis reaches the third stage or until it is complicated with tuberculosis. Men with first- and second-stage silicosis appeared, on the whole, to be in better health than those in the essentially negative group.

As silicosis advances, weight shows a slight tendency to increase until the third stage is reached or until the disease is complicated with tuberculosis. This increase may be due to some extent to the increase in age.

An analysis of systolic blood pressure by age groups shows that the blood pressure of the younger men did not change appreciably but that the average of the older men who had silicosis was slightly higher than of those in the corresponding age group who were essentially negative. Clinical observation shows that the blood pressure of silicotics tends to remain normal or increase only slightly, but in tuberculous cases it shows a definite tendency to drop. Any persistent marked drop in the blood pressure of silicotics indicates probable tuberculous infection.

It is generally accepted that the cardinal physical finding in silicosis is diminished chest expansion. Emphasis should be put not upon chest expansion but rather upon the importance of comparison with a man's earlier chest expansion. Probably if the vital capacity of a large, unselected series was measured for comparison the decrease in vital capacity of silicotics would be greater than any chest-expansion measurements, however carefully taken, would indicate.

Certain breath sounds rarely noted in the chests of healthy people not working in dusty occupations are often detected in the chests of miners, particularly those with silicosis.

The slight changes revealed by examination of the heart probably could be attributed to the ages of the groups of silicotics.

ROENTGENOLOGICAL ASPECTS OF SILICOSIS

It is generally accepted that the X-ray offers the best and most reliable indication of the lung changes that occur in silicosis, particularly in the early stage. The value of this method of diagnosis depends largely upon the skill of the technician who takes the pictures and the experience of the reader who interprets them.

In fact, according to Croizier, Martin, and Policard (*195*), radiographic diagnosis often is very delicate. This is due to the unceasing movement of the organ; if even for an instant it can be voluntarily immobilized in "its incessant coming and going," nevertheless it is subject to perpetual agitation, determined by the periodic flow of the arterial blood; this is why the posing time for making an impression on the film necessarily must be extremely short (*195*).

Pancoast and Pendergrass (*202*) believed that the ability to interpret roentgenograms of cases of pneumoconiosis properly must be based upon several very important factors, which may be enumerated as follows: Knowledge of the anatomy of the chest and many of the physiological problems associated with its anatomical constituents; thorough familiarity with normal roentgenographic and fluoroscopic appearances and permissible variations therefrom within normal limits; knowledge of the histology of the lungs and especially of the lymphatic system; clear perception of the pathology of the condition of pneumoconiosis and of all conditions which may simulate it in roentgenographic appearances; experienced intimacy with the roentgenographic appearances of the condition in question and of those that resemble it, based upon fundamental knowledge of the pathology represented; knowledge of the physical factors involved in producing the suspected or alleged pneumoconiosis; and employment of the proper technic to show to full advantage any or all of the abnormalities present, for technic may fully enlighten, may so modify appearances as to be confusing, or may be quite misleading.

The fibrotic changes revealed by the X-ray study of the Picher Clinic (*99*) are classified arbitrarily as definitely negative, more fibrosis than usual, decidedly more fibrosis than usual, three stages of silicosis, and silicosis plus tuberculosis.

Definitely negative.—In the definitely negative chest the hilum shadows are not more than 7½ cm. in width from the midline and do not cover more than two interspaces and one rib at a target distance of 48 inches. The hilum shadows occasionally may show one or more fairly large calcifications.

The pictures disclosed little if any evidence of an unusual amount of peribronchial thickening in any of the definitely negative chests. The bronchial tree usually cannot be traced farther than the inner edge of the mid-third of the lung and never beyond the inner section of the outer third of the lung. The areas between the shadows thrown by the bronchial tree are clear and show no evidence of any inflammatory changes.

More fibrosis than usual.—The hilum shadows are larger than normal—that is, more than 7½ cm. wide (from midline)—and cover more than two interspaces and one rib. Most of these cover more than two interspaces and two ribs. The calcifications within the hilum shadows show a very definite increase and sometimes are very large. The bronchial tree shows definite thickening and often extends well into the apexes. In the majority of cases its branches can be observed radiating to the outer third of the lung.

Decidedly more fibrosis than usual.—This class corresponds to that sometimes referred to as presilicotic. The hilum shadows are definitely enlarged and dense. The X-ray reading showed that 14.46 percent of these cases at Picher had large, dense, hilum shadows. The bronchial tree shows a definite thickening and extends to the periphery of the lung, involving the bronchioles and possibly the air vesicles.

First-stage silicosis.—The hilum shadows are increased in density and are larger than normal. In many instances (of Picher cases) larger calcified spots were noted in or around the hilum shadows. These were larger than those generally observed as the result of tuberculosis in childhood. In several instances collections of such calcifications involved the entire hilum.

Following enlargement of the hilum shadows, the entire bronchial tree increases in density. It is very noticeable and often can be traced to the outer margins of the lungs. Along the thickened bronchial tree near the hilums are small shotlike spots, sometimes described as "beads" or bronchial "buds." When the spots become noticeable throughout the lower section of the lungs the case is classified as the beginning of first-stage silicosis. The spots are fairly dense, one-eighth inch or less in diameter, discrete, with irregular fuzzy outline. With advance in the disease the spots increase in number, density, and size.

The diaphragm was humped in only a small number of the Picher cases in early silicosis, and no displacement of heart shadow as a result of the disease in this stage was observed.

Second-stage silicosis.—The hilum shadows are large and dense but generally do not show any more definitely than in first-stage silicosis. The beads or buds along the bronchial tree become larger, more numerous, denser, and clearer in outline; the condition is best described as a general "mottling." The mottling is bilateral, and the density is about equal on both sides, indicating that the changes started in both sides at or about the same time.

The diaphragm is often humped or "peaked," and numerous bands of adhesions are noted at the bases. The heart shadows usually are normal.

Third-stage silicosis.—The spots described under second-stage silicosis tend to coalesce, forming large areas of marked density. In some of the cases very large areas of marked density were observed—usually in the middle section of the lungs—which were similar to tuberculous consolidation; these areas, however, usually were bilateral and blended so perfectly with the snowstorm appearance as to suggest silicotic areas, which the physical examinations and laboratory findings tend to confirm.

Silicosis plus tuberculosis.—The ordinary silicotic findings described under the different stages of silicosis were noted in the Picher cases (*100*). In many of them detection of the beginning of tuberculous infection was difficult because of the extensive silicotic changes. The hilum shadows were more likely to show calcified spots than in uncomplicated silicosis. In some of the cases the calcified spots or glands were very large and occasionally occurred in large numbers.

With the beginning of tuberculosis areas of marked density were observed, usually at one or both apexes. These areas were not so dense or opaque as the fibrotic areas of silicosis and appeared cottony or wooly. Usually they were unilateral in the beginning but often became bilateral before death. In some instances these areas occurred in the midsection of the lungs opposite the hilum. Large areas of marked density may occur in this region, and it was difficult to determine by X-ray whether the area was a walled-off tuberculous abscess or a dense fibrotic area of silicosis.

Pancoast and Pendergrass (*204*) summarize the roentgenological phases of the condition as follows:

1. The perivascular-peribronchial lymph-node aspect is due to the relaying of phagocyted dust to the pulmonary lymph nodes and their subsequent enlargement and ultimate partial fibrosis to the gradual enlargement of lymphoid deposits along the course of lymph vessels and the subsequent thickening of these vessels and stasis of contents. This is characterized roentgenographically by increased prominence of the hilum and trunk shadows and linear markings. This appearance is by no means characteristic of pneumoconiosis alone, and even if it does indicate the condition the phase is absolutely not incapacitating. It, together with a barely perceptible appearance of macroscopic nodules, corresponds to the so-called first stage.

2. The nodular aspect is due to the gradual enlargement of lymphoid deposits and their coalescence into quite apparent macroscopic nodules symmetrically scattered throughout both lungs. This corresponds to the so-called second stage. It is conspicuously absent in many industries, especially when the silica intake is rapid.

3. The interstitial type of the condition results from a hilumward and pleuralward block in the lymphatics and the escape of dust phagocytes in large numbers into the interstitial interalveolar tissue and subsequent fibrosis. It appears as a faint homogenous haze, first on the right side, then on the left. If the silica intake is comparatively slow, it may accompany the perivascular-peribronchial lymph-node aspect, but if more rapid, it may be associated with the nodular type

or may progress without the latter directly into the terminal stage of the condition without any evidence of nodulation. The unprotected or inadequately protected sandblaster, sand pulverizer, and sandstone-abrasive worker or granite cutter have been among those especially prone to present this rapid interstitial aspect.

4. Terminal and incapacitating silicosis is characterized by three general appearances—a terminal diffuse fibrosis of a conglomerate nodular type, one which is quite similar in appearance to a generalized chronic fibroid tuberculosis, and the terminal stage characterized by large fibrous consolidated areas, which closely resemble tuberculous consolidations quite frequently, but which we are now learning to differentiate one from the other.

According to Gardner (*186*), anatomic and roentgenologic studies of the lungs of persons exposed to various types of industrial dusts have demonstrated that all inhaled foreign substances do not produce the same kind of pathologic reaction. He divides the forms produced into three categories—linear, nodular, or diffuse in character. A linear pattern characterizes the general type of response to most inhaled inert foreign materials; nodular lesions apparently are confined to silicosis; and diffuse reaction is exemplified in asbestosis. Mixed patterns are produced by dusts such as granite, which is composed of several different elements.

Böhme (*77*) stated that the hard-rock miners of the Ruhr coal districts are peculiarly suitable for investigations of the development of tuberculosis in the silicotic lung since before beginning work they are examined medically and employed only if they prove to be healthy and free of tuberculosis

His description of the process of the development of tuberculosis in the silicotic lung follows:

By X-ray examination of hard-rock miners with finely mottled silicosis, we encounter occasionally pictures which, in addition to fine silicotic mottling, show unilateral round nodules, mostly in the infraclavicular region, what we otherwise designate as premature infiltration. However, they usually cause no clinical symptoms in silicotics as premature infiltrations so often do—loss of weight, night sweats, weakness, fever are lacking at first; indeed catarrh cannot be determined as a result of the obstruction of the drainage system by silicosis. Likewise the settling time of the red blood corpuscles at first is often normal. Nevertheless these nodules do not heal entirely but progress, although slowly, and often lead to very extensive consolidation which anatomically appears as an intimate mixture of silicotic tissue with small tuberculous foci (tuberculo-silicosis). Finally, however, these tuberculo-silicotic nodules under formation tend to disintegrate into central cavities, often rapidly increasing in size and secreting bacillus-containing sputum. From this moment on the tuberculosis dominates the disease picture. Numerous tuberculous aspiration nodules develop, especially in the lung sections of those less affected by silicosis.

Larger tuberculo-silicotic nodules also may be developed in the lungs, which otherwise show only slight silicotic changes. It appears as though in these cases the rock dust is deposited mostly where already tissue changes were determined by slight tuberculous foci. The observations of Giese may be mentioned here, according to which an enrichment of silicic acid has been detected in the tuberculous hilum cavities even when no silicotic consolidation has been formed in the lungs.

In contrast to these very chronically progressing forms with tuberculo-silicotic nodules a tuberculous focus occasionally may tend to disintegrate in silicotic lungs from the beginning without the formation of the larger tuberculo-silicotic nodules, so that the occurrence of tuberculosis with all its accompanying symptoms appears in the foreground prematurely. Such forms are frequently encountered especially when the rock-dust hazard is very severe and the silicosis develops after a few years (sandblasters, scouring-powder workers).

The infraclavicular consolidation focus is a frequent form of development of tuberculosis in silicotics but not the only one. Many times a progressive tuberculosis is developed in the silicotic lung from former apex-foci.

With another group of patients it is to be assumed again that an old inactive disseminated tuberculosis flares up under the action of rock dust and thus in numerous places leads simultaneously to the formation of foci of consolidation with which a silicotic wall is often found around the caseous tuberculous center, or tuberculous and silicotic tissue is combined in almost all foci. * * *

We can find, as mentioned above, in the finely mottled silicotic lung the same form of evolution of tuberculosis as formerly and certainly as infraclavicular foci from a chronic apex affection or as scattered foci of disease. The course of tuberculosis, however, is differentiated most from accompanying silicosis by the formation of tuberculo-silicotic nodules and the return after years of the clinical symptoms of tuberculosis.

If silicosis has already reached the corresponding third stage the moment of the appearance of tuberculosis and its extent are often very much more difficult to determine. The more severe infiltration of the lungs with silicotic nodules makes very difficult the recognition in an X-ray picture of a beginning tuberculosis, and clinical symptoms, as already stated, tend to make the tuberculosis first if it has progressed further.

My opinion is based on the tuberculosis becoming manifest with third-stage silicosis often after it has remained completely latent for years and cannot be diagnosed certainly by X-ray. This is especially confirmed by the fact that inoculation of guinea pigs with sputum from such patients frequently does not show positive results for years before the appearance of tuberculous symptoms. Often enough tuberculosis may already be present in the first or second stage. A demarcation between tuberculous and silicotic occurrence is scarcely possible during life in many advanced cases. In any case tuberculosis is much more frequent with third-stage silicosis than is clinically recognized. Wiltjen found active tuberculosis twice as frequent by lung autopsy as was determined clinically; my own experience confirms this.

The anatomical finding, however, also does not always allow a sharp differentiation between silicosis and tuberculosis in extensive consolidation.

Haldane (*205*) considered the diagnosis of silicosis, apart from a history of serious exposure to dangerous dust, extremely difficult, and the sooner an agreement can be reached as to the ground for a diagnosis of silicosis the better; but these grounds must be consistent with known facts regarding the infrequency or frequency of phthisis in the kind of occupation in which the patient has been actually engaged—for example, ordinary collier's work or work in driving a road through highly siliceous rock without adequate precautions.

Smith (*206*) has pointed out striking discrepancies between symptoms and physical signs and pulmonary fibrosis, as shown by the X-ray. In a study of rock drillers in the vicinity of New York 25 percent of those whose X-rays showed unmistakable silicosis had no symptoms whatever, and in a study of granite workers (*207*) in the same locality, 6 cases of 17 with advanced silicosis had no symptoms. Absence of symptoms does not indicate absence of silicosis. Smith quoted an incident reported by Watkins-Pitchford; of a group of 541 miners who had been receiving compensation for silicosis on the basis of a physical examination alone, 41 percent were found not to have it when X-rays were introduced. She considered a physical examination somewhat of a luxury where large groups of workers are being examined for silicosis; where economy of time and expense is desirable, it would seem legitimate to limit the examination to a good roentgenogram supplemented by occupational history.

McNally (*208*) stated that, during life, the diagnosis of silicosis depends on the history, clinical examination, and roentgenographic

evidence of the disease. Many times the roentgenograms are not decisive, so the diagnosis of silicosis, silicosis plus tuberculosis, or tuberculosis cannot be made wtih certainty. At autopsy the doubt may still linger; then a chemical examination for the quantitative determination of silicon dioxide aids in reaching the correct diagnosis.

The Committee on Pneumoconiosis of the American Public Health Association (*209*) has prepared the following tabulation of the various lesions of silicosis and has attempted to depict the character of the shadows cast on an X-ray film by these lesions. The tabulation applies only to silicosis resulting from inhalation of dust with a high silica content.

Healthy lungs and adnexa

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

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

Simple silicosis

Roentgenological appearance:	Histological appearance:
4. Nodulation—discrete shadows exceeding 6 mm. in diameter, tending to uniformity in size, density, and bilateral distribution, with well-defined borders surrounded by apparently normal lung shadows. The outer and lower lung fields characteristically show fewer nodules.	4. Circumscribed nodules of hyaline fibrosis located in the parenchyma of the lung. Occasionally some of these nodules may show microscopic foci of central necrosis.
5. Conglomerate shadows that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by— (*a*) Localized increased transparency of the lung with loss of fine detail. (*b*) Intensification of the trunk shadows by contrast. (*c*) Depression of the domes with possible tendency toward individualization of the costal components of the diaphragm.	5. The result of the coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more or less hyaline fibrous tissue. The lung architecture is partially obscured. No demonstrable evidence of infection. Emphysema is a compensatory dilatation of the air spaces with or without thickening of the septa.

Simple silicosis—Continued

Roentgenological apperance—Con.

5. Conglomerate shadows, etc.—Con.

(*d*) Lateral view: Increase in the preaortic and retrocardiac space with exaggerated backward bowing of the spine. Widening of the spaces between the ribs may or may not be present.

Silicosis with infection

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

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

Croizier, Martin, and Policard (*195*) stated that, in spite of their efforts, they found it impossible to use the nomenclature suggested by the 1936 Conference of the International Labor Office, believing the classification to be too complicated and absolutely unusable in ordinary practice. These authors (*195*), as a result of their extensive studies of underground coal miners in the region around Saint-Étienne, grouped the radiographic images into subnormal, nodular and pseudotumoral images and unilateral or bilateral densifications.

HOW FIBROSIS OF THE LUNGS IS PRODUCED

Kettle (*183*) pointed out that silicosis presents to the pathologist two fundamental biological problems: What is the exact process by which fibrous tissue is formed in the lungs in response to the presence of dust and how does dust influence the progress of a coincident infection? He considered that these problems lie "at the very root of

the matter," and even if the answers were known he doubted whether the situation as it exists in industries could be handled much more satisfactorily. He said,

> But if we cannot explain the *how* of the matter we may still hope to speak with some authority about the *when* and the *where*. We can discuss with profit such questions as the identification of dangerous dusts; whether they act chemically or physically; the influence of their physical state on their action; the influence of associated dusts; and the relationship of dust to infection.

For many years it was held that fibrosis of the lungs characteristic of silicosis was produced in response to irritation caused by hard, sharp quartz crystals; that is, the dust was believed to act mechanically. To Kettle (*183*) this point of view had some merit, as it is natural to think that hard, sharp quartz particles would wound and tear the soft tissues of the lungs; it is, of course, a commonplace of pathology that foreign bodies in the tissues excite a certain amount of reaction which is followed by the production of fibrous tissue. This fibrosis, however, is never extreme and is never comparable to the extensive fibrous tissue formations recognized as lesions of silicosis. In point of fact, if a harmless dust is inhaled in large enough quantities some of it remains in the lungs and causes mild fibrosis by the mere mechanical irritation of its presence. This fibrosis is never severe enough to interfere with the function of the lungs; and in its distribution, particularly in its amount, it bears no comparison to that seen in silicotic lungs (*183*).

Kettle (*183*) considered solubility a primary factor in the harmfulness of silica, and he believed that a perfectly insoluble substance is incapable of causing pneumoconiosis. Exactly how the dissolved silica acts in the production of fibrous tissue is not known. His suggestion that it acts chemically as a cell poison has been severely criticized, probably on the grounds that, since silica enters into the composition of animal and vegetable protoplasm, it cannot be a protoplasmic poison. His reply to this criticism was that phosphorus enters into the composition of protoplasm, yet people have been killed by phosphorus and others have been hanged for poisoning them. Koppenhöfer (*210*) suggested two theories to explain the reaction of the body fluids to quartz: (1) The superficial layers of the quartz particles corrode, resulting in a splitting off of minute portions; (2) a colloidal reaction takes place in the weak alkaline medium provided by the tissue fluids, resulting in a gradual solution of the particles. He thought that probably both factors operate but the presence of silicic acid in colloidal form has the most harmful effect. Peacock (*211*) in 1860 was the first to describe a microscopic demonstration of silica in the lungs. In 1866 Schmidt (*212*) reported sand in the lungs of all persons—except the newborn—ranging from 4.2 to 17.3 percent of silicon dioxide in the ash. According to Woskressensky (*213*) the lungs of individuals whose occupations do not expose them especially to dust inhalation contain an increasing amount of silica in direct proportion to the age. This investigator found that 3.5 to 33.7 percent of the ash of the lungs was silicon dioxide, and in the peribronchial lymph glands the amount was much higher, ranging from 18.3 to 55.6 percent of the ash. McNally (*208*) gave the silicon content of the lungs of eight persons working in dusty atmospheres as follows:

Case	Ash, per-cent	SiO_2 per gram of dried tissue, mg.	Occupation
44	10 78	8.6	Millstone sharpener.
503	17 14	14.0	Stone cutter.
327	12.36	2.4	Machinist.
429	14 58	3.6	Engineering draftsman.
463	19 99	4.3	Coal miner, 25 years.
411	8.84	5.0	Stone quarry man, 9 years.
300	8.46	26.0	Granite cutter.
446	8.59	10.9	Zinc miner.

McNally (*208*) also stated that the lungs of tuberculous subjects contain somewhat more silicon dioxide than those of normal persons; this may come from inhalation or directly from the blood stream, as the blood of tuberculous subjects contains more silicon dioxide than that of normal persons. He quoted Collis to the effect that all clinical observations have shown that a silicotic patient is particularly liable to tuberculous infection and that the silicotic process continues to advance after complete withdrawal from exposure.

Gardner (*214*) found that, within limits, silicosis is a progressive disease. A man may leave a dusty industry without demonstrable evidence of nodular fibrosis in his roentgenogram; if he has already inhaled enough quartz, tissue reaction will continue in the absence of further exposure until its effects become apparent in roentgenograms of subsequent years. However, equilibrium between the irritant in the lungs and the tissues is eventually established. New silicotic nodules cease to form, and the original ones undergo only slight increase in size. Progression is always self-limited, and most of the pulmonary tissue remains essentially normal. There is no disability because the pulmonary reserve greatly exceeds the amount of lung replaced by small localized nodules of scar tissue.

In his report on a comparative study of the solubility of finely divided rock dust in water, kerosine, and alcohol Myers (*215*) made the following statement regarding siliceous dust.

> It will be noted in test 1 that the process of solution (in distilled water) of the 5-mg. quantity of siliceous dust was gradual throughout the 28-day period, solution being nearly complete at the end of this time, while the 50-mg. weight of siliceous dust did not dissolve after the first 24 hours. It is believed that the finest particles went into solution very rapidly and that there was enough of them in 50 mg. to saturate the solution in the first 24 hours. The 5-mg. weight containing many larger particles and only a tenth as many of the extremely fine particles as 50 mg. went into solution slowly, and the solution was not saturated until the entire amount was in solution which took between 21 and 28 days.

Heffernan (*216*) summarized his theory of the action of silica on the lungs as follows:

> 1. Silicosis is a result of the local action of hydrated silica upon the pulmonary tissue. This action is of a physicochemical nature, and the speed of its development, other things being equal, depends upon the rapidity with which fresh silica hydrosol is formed and brought into contact with pulmonary tissue.
>
> 2. Substances which favor the formation of silica hydrosol from silica, when added to the silica dust, accelerate the development of silicosis—for example, the alkalies. Substances which retard or prevent the formation of hydrosol from silica, or which coagulate the hydrosol when formed, retard or prevent silicosis—for example, carbon, coal dust, clays, and probably many other substances.

3. The action of silica in producing the nodular pulmonary fibrosis which we term "silicosis" has not been paralleled so far by any nonsiliceous substance.

4. Silica is a normal constituent of plant and animal cells; its presence seems essential in certain tissues, and its absence from the food of the organism disastrous.

5. The study of the role of silica in biology suggests that many of the processes of cell metabolism belong to the realm of colloidal physics.

6. Some characteristic cellular phenomena are explained most simply by regarding the living cell as a polyphase colloidal system.

Kraut (*217*) found that the silicic acid content of the blood of various normal persons fluctuates between 1 and 3 percent of the sulfate ash but that the individual maintains his silicic acid content with great constancy. The administration of easily resorbable silicic acid increases the silicic acid content of the blood many times the absorbed amount of silicic acid.

Lieb and Schadendorff (*218*) found the SiO_2 content of pathological lungs of silicon workers to be 0.3 to 1.46 percent if dried at 120° and 4.6 to 22.8 percent of the ashed tissue. Normal lung tissue showed values of 0.08 to 0.40 percent. The pathological findings corresponded with the analytical results.

Smith and Wikoff (*219*) noticed a striking correlation between the percentage of SiO_2 in dried lung substance and the severity of silicosis as determined by an entirely independent method of diagnosis. They reached the following conclusion from their analytical findings correlated with the histological diagnosis and occupational history of nine cases of silicosis in men engaged in the construction of a tunnel through rock rich in silica:

If one compares the percent of ash in the dry lung substance in each case with histological diagnosis he will note a very good correlation, for the SiO_2 content of the lung increases with the severity of the case. On the other other hand, if the severity of the case is to be determined chemically by the percent of SiO_2 in the total ash, the correlation fails. We believe, therefore, that the percent SiO_2 in the dry lung substance is a much better criterion of silicosis than is the percent of SiO_2 in the total ash.

Haldane's (*220*) explanation of the large percentage of the silicate sericite which Jones found in the lungs he examined is that sericite is less soluble than free silica in the alkaline liquids of the lungs. Kettle mentioned (*219*) in this connection that crystalline silica when inoculated in the veins produces lesions but that amorphous silica does not; moreover, readily soluble silica and insoluble silica do not produce lesions, but between these two is a silica that "turns the trick." In 1929 Mavrogordato (*24*) gave as the cause of simple silicosis the reaction of the reticulo-endothelial system of the organ concerned to invasion by free silica; but dust phthisis, the clinico-pathological entity met in practice, is superficially a variable disease. Beyond the external factors and the reticulo-endothelial system as an apparatus lies the man. Individuals vary in their ability to resist the dust they inhale. The amount of dust that can be recovered from a lung need bear but little relation to the extent or character of the lesions found.

Travers (*221*) remarked about one or two facts that were brought to his attention following a review of the literature on silicosis but that apparently had been unnoticed before. A study by Ray (1923) showed that the solubility of mechanically ground silica was markedly higher than that of crystalline silica; this fact may go far toward accounting

for the poisonous character of mechanically produced dusts. It is suggested (*221*) that the microcrystalline material present in the lung, which is probably silica, is separated from the lung fluids already saturated with mechanically transformed silica. The presence of the microcrystalline material may have no connection with the toxic property of the silica.

From a mineralogical standpoint, on the basis of solubility ratio of quartz and the silicates, Udluft (*222*) presented for explanation of silicosis the following theory:

> The chemical and mechanical theories recommended by medicine are both partly right. Both methods of operation go along side by side. The body attempts to get rid of the infiltrated dust by mechanical and chemical means. Thereby, those minerals which are stable, unaffected, and as far as possible even newly formed under the given conditions, are to be considered as mechanically harmful; while on the other hand the easily attacked and more or less soluble minerals in a chemical way are removed and thereby can be chemically active and chemically harmful. The remarkably severer harmfulness of the free mineral acids (silicic acid, titanic acid) and acid minerals over the basic appears to be explicable in that between the alkalies of the blood or of the body and the portions of the inhaled dust particles passing into solution there exists an equilibrium which in the case of free acids and acid minerals leads to excessive alkali loss, while with the infiltration of basic minerals such an injury does not occur. This alkali interpretation should be carefully proved.

The manner in which stone dust exerts its effects on pulmonary tissue remained a matter of speculation and argument for many years (*223*). The following review of the investigations that led to the modern chemical theory of silicosis is taken from an article by King (*223*).

It was first thought that the fine particles of stone dust cut and lacerated the tissues with which they came into contact. As viewed under the microscope the mineral particles certainly showed sharp edges and points, but it was nevertheless difficult to believe that such extremely fine material could traumatize a tissue. As a consequence, doubts were early expressed as to the validity of this "mechanical" theory of silicosis. Two crucial experiments turned the attention of investigators to an alternative hypothesis. The first of these was by Gardner (*224*), who in 1923 brought convincing evidence against the "mechanical" theory by demonstrating that the dust of silicon carbide ("carborundum"), which is extremely hard and has just as sharp edges as stone dust, failed to produce the typical fibrous reaction which powdered stone dust (particularly that of quartz or flint) would cause when placed in the lungs of animals. These classic experiments of Gardner's marked the beginning of the modern theories and investigations of silicosis.

The second investigation which made the "mechanical" theory of silicosis obsolete was that of Gye and Purdy (*225*) in 1922; also Gye and Kettle (*225*) in 1922, and Gye and Purdy (*226*) in 1924. These workers produced in mice treated with amorphous silica a lesion characterized by acute inflammation and necrosis and in the liver a condition of necrosis by the injection of colloidal silicic acid. Gardner and Cummings (*227*) in 1933 obtained similar results by the injection of finely powdered quartz. All this work pointed to the possibility that stone dust might be harmful to the lungs, not because it was hard and sharp, but because it produced a soluble substance which was toxic to the tissues. Policard (*228*) in 1933 showed that cells poisoned with

dissolved silica (silicic acid) do not disintegrate and disappear as do other cells, but tend to preserve their structure in a fashion which suggests mummification. Kettle (*229*) in 1932 brought the final proof that the dissolution of a soluble substance from the stone particles was necessary to produce silicosis by demonstrating that the same quartz particles which readily produced silicosis in animals would no longer do so if they were first coated with a thin layer of iron oxide, which did not alter the sharpness but which effectively prevented any part of their substance from going into solution.

The modern "chemical" theory of silicosis, then, supposes that the fine particles of stone dust which get into the lungs are pathogenic, not because they produce a sort of microscopic trauma, but because something of a toxic nature dissolves from their surfaces. This toxic substance is thought to be silicic acid.

King (*223*), however, has noted discrepancies between silica solubility and disease symptoms. In commenting on the results of his experiments on the solubility of dusts compared with their disease-producing tendency, King (*223*) said that the solubility theory had been too useful to abandon because of the discovery of some discrepancies and anomalies, although it is difficult to reconcile these differences. He concluded that either the noxious dust releases something that is harmful to the living cell or it has special surface properties that cause abnormal reactions in certain cell constituents which become adsorbed on its faces. The difficulty of imagining what such reactions could be drove him back to the notion that a noxious dust must be toxic because it yields a toxic substance. King (*223*) suggests the following as a possible explanation:

> Certain siliceous dusts, at least, behave as if they release such a poison into the cells which engulf them. Others, which have the same effect on cells, do not liberate silicic acid so readily in the test tube. Still others liberate a little more silicic acid but do not harm cells. May it perhaps be that siliceous dusts release soluble silica differently in cells than in the test tube, and that some dusts may liberate the silicic acid in a different and more noxious state than other dusts? Silicic acid is constantly being absorbed into the body through the intestine and excreted by the kidney; it does no harm. It is possible that this silicic acid is different from that released from the surface of a quartz particle engulfed in the body of a phagocyte. It would be daring to speak of a "nascent silica," but something of this sort may actually occur. For the silica released from particles of the pathogenic siliceous dusts certainly has special properties, and these special properties may be due to the form in which it is released into the protoplasm in contact with the particles in the cell. Such a speculation may appear far fetched, but it is a possible explanation, and it may serve the purpose of leading to further experiments which may have a useful outcome.

It was agreed at the International Conference on Silicosis in 1930 (*184*) that:

> To produce the pathological condition, silica must reach the lungs:
>
> (*a*) In a chemically uncombined condition, although the dust inhaled may be either a natural mixture of silicon dioxide with other dusts, such as occurs in granite, or an artificial mixture, such as scouring powder.
>
> (*b*) In fine particles of the order of less than 10 microns. There is no evidence as to the lowest limit of size in which the particles may be capable of producing the disease.
>
> (*c*) In sufficient amount and over a certain period of time; these two factors are reciprocal variants. The maximum of these two respective factors has not yet been determined.

Silica dust plays the dominant role in the production of silicosis, admixture of other dusts tending to modify the picture in the direction of that of other pneumoconioses, in some relation to the proportion of free silica inhaled.

There is experimental evidence that the solubility of silica in the tissues is an essential factor in the causation of silicosis.

According to Badham's summary (*230*) of the information supplied by various members of the International Silicosis Conference, silicosis becomes noticeable after widely differing periods of exposure to siliceous dust, depending apparently upon:

1. The amount of dust inhaled.
2. The percentage of free silica contained therein.
3. The size frequency (or fineness) of the particles inhaled.
4. The nature and sort of such other substances (including vapors and gases) as may be inhaled simultaneously or otherwise.
5. The powers of resistance of the individual concerned.
6. The presence or absence of a complication by an infective process.

In a discussion of the sericite theory as a cause of silicosis Irvine (*231*) stated that, although it quite probably will become necessary to revise and widen the definition of the essential factors in the causation of silicosis, even so nothing has significantly altered the general conception of silicosis as a definite pathological entity, which is readily identifiable by the pathologist after death and which is adequately distinguishable during life by expert clinical and radiological examination, or the knowledge that this disease, year by year, produces many cases of incapacity or death. The position, he said, in these respects is not significantly affected by the present lack of complete knowledge regarding the exact constituents of a siliceous dust that may cause silicosis. In spite of considerable opposition from certain quarters, this view was accepted at the meeting of the International Labor Conference at Geneva in June 1934, and the conference accordingly affirmed the international recognition of silicosis as an occupational disease and described it as "silicosis, with or without pulmonary tuberculosis, provided that silicosis is an essential factor in causing the resultant incapacity or death."

In a paper on the etiology of silicosis presented at the Fourth Saranac Laboratory Symposium on Silicosis, Cummings (*232*) stated that only two important factors are involved in the formula for the causation of silicosis—the individual's susceptibility to injury by fine particles of free silica and the degree of harmful exposure offered by the environment in which he is employed.

The role played by individual susceptibility is indicated by the fact that some workmen have had long periods of uninterrupted service in some of the most dangerous occupations without having been affected adversely (*232*).

Among the important factors (*232*) affecting susceptibility to injury by dust are anatomical peculiarities of the respiratory mechanism, the physiological response to dust inhalation, previous dust exposure, the presence of pulmonary infections or other pathological processes—particularly tuberculosis—the age, nationality, and general health.

The most important factors comprising the degree of exposure to harmful dusts are composition, size distribution, and physical characteristics and concentration of dust suspended in the atmosphere at the

breathing level, together with the duration of exposure and the general conditions of work (*232*).

In the discussion of Cummings' paper, Lanza (*232*) pointed out that individual susceptibility had been exaggerated in the lay mind sometimes to draw attention from the major problem of atmospheric dust concentration. He expressed doubt of innate variations in the capacity of different individuals to react to silica dust. He admitted that there were variations in the reaction of different individuals apparently exposed under identical conditions to the same kinds of dust. He was inclined, however, to believe that the conditions were not identical as no history could reveal every detail of exposure from the time a man left school.

Cummings (*232*) expressed surprise that medical men should question individual susceptibility, which provides the real reason for their participation in the control of silicosis. Why otherwise should it be necessary to make preemployment examinations before putting men into dusty jobs if it were anticipated that all would react to their environment in the same manner? He was willing to qualify his statement to the extent of admitting that individual susceptibility was not as important as the degree of exposure to silica.

Gardner (*232*) interpreted Cummings' qualifying statement as an admission that variations in susceptibility might be of an acquired nature and due to infection or other causes; all his experimental experience indicated that the innate capacity of the cells of different individuals of the same species to react to silica was essentially the same.

In 1938 Croizier, Martin, and Policard (*195*) presented two factors as predominating in the etiology of silicosis—mineral dust and tuberculosis. They found in every case a certain extension of tuberculosis and a very special frequency of the fibrous forms of this disease wherever siliceous dusts were present. They then brought up the question as to which of these factors comes first; does the siliceous dust determine the fibrosis first and thus favor a localization and a secondary development of the Koch bacillus or is the tuberculosis the first phenomenon and under the action of siliceous dust takes, from the beginning of its evolution, a sclerous course? The authors presented arguments for both factors but stated that

> On the whole, it is only with occurrence of tubercular infection, but under very particular conditions, that formation of massive pulmonary fibrosis can be determined.

They also stated that:

> If rock dust aids pulmonary tuberculization in a manner clearly more pronounced than coal dust, it has on the contrary the privilege of determining on the surface of the affected parts a more marked sclerous reaction. To remain in the domain of practical statements we must say that if the dense, fibrous phthises of coal miners sometimes are identical in appearance with those of rock miners they are, at the same time, infinitely rarer.
>
> To summarize the determining causes of these pulmonary fibroses of miners appears to us to be:
>
> 1. Less severe tubercular infection favored by the inhaled dusts which reduce the efficacy of the natural means of defense of the organ.
>
> 2. An exaggerated sclerous reaction of the lung connected with the presence of mineral particles endowed with special qualities.

PREVENTION OF DUST DISEASES

As no cure is known for silicosis after it has developed, prevention is the only effective remedy. There are two main lines of approach to the problem of dust-disease prevention in industry—the engineering, through control of dust production and dissemination, and the medical, through selection and control of health of workers by examination; success probably depends upon a combination of the two.

Hatch (*233*) considered the following measures essential to a complete, well-balanced program of control of the dust hazard in industry:

1. Selection of workers by means of physical examinations in order especially to eliminate those with tuberculosis and other disorders of the respiratory system.
2. Reduction of the dust concentration below the standard of permissible dustiness through adequate means of dust control and good housekeeping.
3. Limiting the frequency and duration of employment in the dusty occupations, thus reducing the rate of deposition of dust in the lungs.
4. Routine measurement of the effectiveness of the dust-control program by means of periodic medical examinations and dust surveys.

Of these, reduction in dustiness is the most direct in its effect and is fundamental to any program of control. Hatch (*233*) emphasized, however, that present-day standards of safety are only estimated values and reduction in dust exposure below the estimated threshold value does not give absolute assurance of complete control of the disease. Moreover, a potential hazard always exists in basically dusty industries, and a constant guard therefore must be maintained. Workers should be selected by physical examination to insure maximum resistance against the action of dust in the lungs. The employment of workers already injured by previous dust exposure thus would be largely eliminated. Routine determination of dust concentrations provides a measure of operation of the dust-control system, but the efficacy of the control program must be measured by periodic medical examination, as only by this means can the presence or absence of silicosis be determined. An inadequate control program thus can be strengthened in time to arrest the widespread development of the disease.

PRINCIPAL DUST FACTORS PRODUCING PULMONARY PATHOLOGY

The principal factors now thought to determine whether exposure to dust will produce pulmonary pathology are nature of the dust, particle size, quantity of the dust dispersed in the atmosphere, and length of exposure.

Gardner (*234*) concluded from his field studies in some of the dusty industries that the evidence makes it seem improbable that a silica hazard is defined solely by the number and size of the silica particles in an industrial atmosphere. The other components of a dust modify its action. Some may inhibit, others retard, and perhaps some will be found to prevent its injurious effects.

Every mineral has certain characteristic properties that separate it from other minerals of similar appearance when viewed under the polarizing microscope. These diagnostic characteristics are supposed to persist even to the smallest grains; with patience, therefore, the percentage of the various mineral constituents of a dust can be determined. (*235*).

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BIBLIOGRAPHY *

1. Richardson, B. W. Unhealthy Trades. (Quoted by Hoffman, *see* ref. 4.)
2. Drinker, Philip. Dust, Fumes, and Smoke. Internat. Labor Office, Occupation and Health, vol 1, 1930, p. 603.
3. Baskerville, Charles. Air Impurities: Dust, Fumes, and Gases. (Quoted by Hoffman, *see* ref. 4.)
4. Hoffman, F. L. Mortality from Respiratory Diseases in Dusty Trades. U. S. Dept. of Labor, Bur. of Labor Statistics Bull. 231, 1918, 458 pp.
5. Thompson, W. Gilman. The Occupational Diseases: Their Causation, Symptoms, Treatment, and Prevention. New York and London, 1914, p. 71.
6. De Balsac, F. H., and Agasse-La Font, E. (Quoted by Drinker, *see* ref. 2.)
7. Schürmann, S. [Industrial Dust: Its Control with Special Reference to Legal Measures.] Zentralb. Gewerbehyg. u. Unfallverh., supp. 15, 1929, pp. 1–21.
8. Sayers, R. R. Harmful Dusts. Repr. 1906, from Pub. Health Repts., vol. 53, No. 6, Feb. 11, 1938, 12 pp.
9. Lyell, Sir Charles. The Geological Evidence of the Antiquity of Man. 1873, 577 pp.
10. Collis, Edgar L. Phthisis Among Flint Knappers. Roy. Commission on Metalliferous Mines and Quarries, vol. 2, app. J., 1914, p. 202.
11. Carozzi, Luigi. Contributo bibliografico alla storia della pneumoconiosi "silicose" (dal XXVII secolo A. C. al 1871). [Bibliographic Contribution to the History of the Pneumoconiosis "Silicosis" (from the XXVII Century B. C. to 1871).] Repr. from Rassegna di medicina industriale, vol. 12–13, 1941–42.
12. Kettle, E. H. Relation of Dust to Infection. Proc. Roy. Soc. Med., vol. 24, 1930, pp. 79–94.
13. Landsberg, H. Dust—a Hazard and a Problem in Mineral Industries. Mineral Ind., vol. 5, no. 6, 1936, p. 4.
14. Pfaff, I. [Concerning Dust in City and Room Air.] Gesundheits-Ingenieur, vol. 57, 1934, pp. 57–58. Abs. Jour. Ind. Hyg., vol. 16, 1934, p. 84.
15. Soper, G. A. Air and Dust in the New York Subway. (Quoted by Thomas Oliver in Diseases of Occupation, New York, 1916, pp. 244–245.)
16. van Sicklen, M. Health Hazard from Dust in the Mines and Allied Industries of the United States. Initial Survey of the Extent and Severity. Am. Inst. Min. and Met. Eng., Contr. 45, 1933, 13 pp.
17. Economic Commission For Europe. Monthly Bulletin of Coal Statistics of the United Nations. Coal Div., No. 9, 1947, p. 4.
18. Jeppe, C. Biccard. Gold Mining on the Witwatersrand. Vol 2, 1946, 1821 pp. Pub. by Transvaal Chamber of Mines, Cape Town, S. A.
19. Böhme, A. Present State of the Silicosis Problem in Germany. Silicosis Rec. Internat. Conf., Johannesburg, Aug. 13–27, 1930. Internat. Labor Office, ser. F, No. 13, 1930, p. 339–368.
20. Bax, K. Die Bekämpfung der Gesteinstaublungenkrankheit in Ruhrbezirk. [Control of Rock-Dust Lung Disease in the Ruhr District.] Glückauf berg-u. hüttenmännische Ztsch., vol. 71, 1935, pp. 1241–1248.
21. Lanza, A. J., and Vane R. J. The Prevalence of Silicosis in the General Population and Its Effects Upon the Incidence of Tuberculosis. Am. Rev. Tuberculosis, vol. 29, 1934, pp. 8–16.
22. Plinius Secundus, Caius. Naturalis Historiae. II. Trans. by Kenneth C. Bailey under the title "The Elder Pliny's Chapters on Chemical Subjects," pt. I, 1929, p. 125.
23. Sayers, R. R. Silicosis Among Miners. Bureau of Mines Tech. Paper 372, 1925, 24 pp.
24. Mavrogordato, A. The Etiology of Silicosis Rept. 4th Meeting Permanent Internat. Commission for the Study of Occupational Diseases, Lyons, Apr. 3–6, 1929, pp. 1–22.

* Titles in brackets are translations from the original language in which the citation was published.

25. Agricola, Georgius. De Re Metallica. I. Trans. from 1st Latin ed., 1556, by Herbert Clark Hoover and Lou Henry Hoover, 1912, p. 6.
26. Koelsch, Franz. [Theophrastus von Hohenheim, Called Paracelsus. Of Miners' Consumption and Other Miners' Diseases.] Deut. Gesell. Gewerbehyg., N. F. 12, 1925, 69 pp.
27. Pancoast, Henry K., and Pendergrass, Eugene P. A Review of Pneumoconiosis: Further Roentgenological and Pathological Studies. Am. Jour. Roentgenol. Radium Therapy, vol. 26, pp. 556–614.
28. van Diemerbroek, Isbrand. (Quoted by Schürmann, *see* ref. 7.)
29. Ramazzini, B. (Quoted by Collis, *see* ref. 34.)
30. Ursinus, —. (Quoted by Koelsch, *see* ref. 26.)
31. Stochausen, —. (Quoted by Koelsch, *see* ref. 26.)
32. Mining Journal. Miners' Asthma or Silicosis. Vol. 228, 1947, p. 309.
33. Löhneiss, G. E. (Quoted by Collis, *see* ref. 34.)
34. Collis, Edgar L. Industrial Pneumoconioses with Special Reference to Dust Phthisis. Milroy Lectures, 1915, 42 pp.
35. Scheffler, —. (Quoted by Koelsch, *see* ref. 26.)
36. Bruce, Torsten. Die Silicose als Berufskrankheit in Schweden. Eine klinische und gewerbemedizinsche Studie. [Silicosis as Ossupational Disease in Sweden. A Clinical and Industrial Medical Study.] Acta Medica Scandinavica, suppl. 129, 1942, 383 pp.
37. Ackerman, —. (Quoted by Koelsch, *see* ref. 26.)
38. Allison, —. (Quoted by Collis, *see* ref. 10.)
39. Miller, Hugh. My Schools and My Schoolmasters. Edinburgh, 1809. (Quoted by Collis, *see* ref. 10.)
40. Hoffman, Friedr. (Quoted by Koelsch, *see* ref. 26.)
41. Henkel, —. (Quoted by Koelsch, *see* ref. 26.)
42. Thackrah, C. T. (Quoted by Collis, *see* ref. 10.)
43. Holland, G. C. (Quoted by Collis, *see* ref. 10.)
44. Greenburg, Leonard. Studies on the Industrial Dust Problem. I. Dust Inhalation and Its Relation to Industrial Tuberculosis. U. S. Pub. Health Repts., Repr. 990, vol. 40, 1925, pp. 291–309.
45. Rosen, George. The History of Miners' Diseases. 1943, 490 pp.
46. Middleton, E. L. Silicosis in Great Britain. (*See* ref. 19, pp. 384–480.)
47. Britten, Rollo H. Occupational Mortality Among Males in England and Wales, 1921–23, U. S. Pub. Health Repts., Repr. 1230, vol. 43, 1928, pp. 1595–1616.
48. British Medical Journal. Silicosis and Fibrosis. Mar. 13, 1926, p. 512.
49. His Majesty's Stationery Office. Memorandum on the Industrial Diseases of Silicosis and Asbestosis. July 1932, 18 pp.
50. Medical Research Council. Chronic Pulmonary Diseases in South Wales Coal Miners. I. Medical Studies. Spec. Rept. Ser. 243, London, 1942, 222 pp.
51. Stewart, J. Logan. Silicosis and Tuberculosis. British Jour. Tuberculosis, vol. 23, 1929, pp. 6–11.
52. Colliery Guardian. Dust Prevention in the Coal Industry. Vol. 144, 1932, pp. 97–99.
53. Chief Inspector of Workshops and Factories. Industrial Diseases and Poisoning in British Factories, 1930. *Safety Eng.*, December 1931, p. 354.
54. Wood, W. B., and Gloyne, S. R. Pulmonary Asbestosis Complicated by Pulmonary Tuberculosis. Lancet, vol. 221, 1931, p. 954.
55. Gloyne, S. R. Pulmonary Asbestosis Complicated by Pulmonary Tuberculosis. Lancet, vol. 22, 1931, p. 1103.
56. Sparks, J. V. Diagnosis of Lung Disease. British Med. Jour., Aug. 20, 1932, pp. 303–304.
57. Sutherland, C. L., and Bryson, S. Report on the Occurrence of Silicosis Among Sandstone Workers. London, 1929, 41 pp.
58. Ferguson, T. Silicosis Among Sandstone Workers in Scotland and the North of England. Jour. Ind. Hyg., vol. 16, 1934, pp. 203–211.
59. Health and Industrial Hygiene. Industrial Diseases and Poisoning in British Factories, 1932. Monthly Labor Rev., vol. 37, No. 4, 1933, pp. 884–890.
60. Colliery Guardian. Parliamentary Intelligence: Industrial Diseases. Aug. 4, 1933, p. 214.
61. ——— —. Feb. 16, 1934, p. 309.
62. Haldane, J. S. Silicosis in South Wales. Iron and Coal Trades Rev. vol. 128, 1934, p. 475.

63. HARPER, ARCHIBALD. Silicosis. Coll. Guard., vol. 147, 1934, pp. 982–983.
64. FISHER, S. W. Dust Diseases Amongst the British Coal Miners. Colliery Guardian, vol. 174, 1947, pp. 481–482.
65. COLLIERY GUARDIAN. Parliamentary Intelligence—Silicosis Medical Board. Vol. 174, 1947, p. 554.
66. MEIKLEJOHN, A. Health Hazards in the North Staffordshire Pottery Industry, 1688–1945. Jour. Roy. Sanitary Inst., 1946, pp. 516–525.
67. TISSOT, —. (Quoted by Lochtkemper and Teleky, *see* ref. 80.)
68. GOETZINGER, —. (Quoted by Lochtkemper and Teleky, *see* ref. 80.)
69. HAUER, —. (Quoted by Schürmann, *see* ref. 7.)
70. NASSE, —. (Quoted by Schürmann, *see* ref. 7.)
71. PAPPENHEIM, —. (Quoted by Schürmann, *see* ref. 7.)
72. ZENKER, F. A. (Quoted by Schürmann, *see* ref. 7.)
73. SCHLOCKOW, —. (Quoted by Lochtkemper and Teleky, *see* ref. 80.)
74. LOCHTKEMPER, I. (Quoted by Komissaruk, *see* ref. 79.)
75. BERGERHOFF, W. Untersuchen über Sandstrahlersilicosen. [Investigations on Sandblasters Silicosis.] Archiv. Gewerbepath. und Gewerbehyg., vol. 7, 1936–37, pp. 156–181.
76. THIELE, A. (Quoted by Komissaruk, *see* ref. 79.)
77. BÖHME, A. Tuberkulose und Silikose. Haufigkeit der Tuberkulose in Steinstaubgefährdeten Berufen. [Tuberculosis and Silicosis. Frequency of Tuberculosis in Rock-Dust Hazardous Occupations.] Zentralb. Gewerbehyg. u. Unfallverh., vol. 22, N. S. 12, 1935, pp. 101–104.
78. ———. (Quoted by Komissaruk, *see* ref. 79.)
79. KOMISSARUK, BELA. [Pneumoconiosis, Tuberculosis, and Social Conditions Among Foundry Workers in Wien.] Arch. Gewerbepath. Gewerbehyg., vol. 2, 1931, pp. 123–139.
80. LOCHTKEMPER, I., AND TELEKY, L. Studien über Staublunge. [Studies on the Dusted Lung.] I, II, and III. Arch. Gewerbepath. Gewerbehyg., vol. 3, 1934, pp. 418–470, 600–672, 673–761.
81. KOELSCH, F., AND KARSTEN, K. (Quoted by Lochtkemper and Teleky, *see* ref. 80.)
82. LANDAU, W. [Pneumoconiosis Observed Among Workers Engaged in Cleaning Castings.] Arch. Gewerbepath., vol. 4, No. 3, 1933, pp. 515–523; abs. Chim. et Ind., vol. 30, No. 4, 1933, p. 825.
83. NEIDERHAUMER, M. H. Lutte contre la silicose dans le bassin Rhenan-Westphalien. [The Fight Against Silicosis in the Rhenish-Westphalian Basin.] Berghau, 1936 (Sept. 3). Abs. Rév. Ind. Minérale, 1937, pp. 306–307.
84. HOLLMANN, RICHARD. Die Anthrakose und ihre Differentialdiagnose zur Silikose und andern Pneumokoniosen. [Anthracosis and Its Differential Diagnosis from Silicosis and Other Pneumoconioses.] Aerztliche Sachverständigen-Ztg., vol. 43, 1937, pp. 3–13.
85. MAASZEN, R., AND BÜTTNER, H. E. Über die Staubgefährdung in der Schmotte- und Dinasindustrie der schlesischen Oberlausitz. [The Dust Hazard at the Ceramic- and Silica-Brick Factory in Silesian Oberlausitz.] Arch. Gewerbepath. Gewerbehyg., vol. 9, 1939, pp. 179–197.
86. PAYNE, A. E., PIROW, HANS, AND ROBERTS, F. G. Historical Review of Mining Conditions on the Witwatersrand and the Changes Which Have Taken Place Since the Early Days of the Fields. (*See* ref. 19, pp. 107–128.)
87. IRVINE, L. G., MAVROGORDATO, A., AND PIROW, HANS. A Review of the History of Silicosis on the Witwatersrand Goldfield. (*See* ref. 19, pp. 178–208.)
88. UNION OF SOUTH AFRICA. Report upon the Work of the Miners' Phthisis Medical Bureau for the 3 Years Ended July 31, 1932. U. G. 22, 1933, 62 pp.
89. SOUTH AFRICAN MINING AND ENGINEERING JOURNAL. Miners' Phthisis. Mar 10, 1934, p. 32.
90. UNION OF SOUTH AFRICA. Report Upon the Work of the Miners' Phthisis Medical Bureau for the Three Years Ended 31st July, 1941. 1944, 46 pp.
———. Report Upon the Work of the Miners' Phthisis Medical Bureau for the Three Years Ended 31st July, 1944. 1946, 52 pp.
91. MURRAY, A. J., AND CROCKET, J. A. Interim Report on the Prevalence of Silicosis and Tuberculosis Among Mine Workers in the Gold Coast. Bull. Hyg., vol. 17, 1942, pp. 523–525.
92. ———. Report on Silicosis and Tuberculosis Among Mine-Workers in the Gold Coast. Abs. Bull. Hyg., vol. 21, 1946, pp. 744–746.

93. SMITH, ADELAIDE R. Review of Silicosis. New York State Dept. of Labor, Ind. Bull., vol. 12, No. 4, 1933, p. 90–92.
94. LANZA, A. J., AND HIGGINS, EDWIN. Pulmonary Disease Among Miners of the Joplin District, Missouri, and Its Relation to Rock Dust in the Mines. Bureau of Mines Tech. Paper 105, 1915, 47 pp.
95. HIGGINS, E., LANZA, A. J., LANEY, F. B., AND RICE, G. S. Siliceous Dust in Relation to Pulmonary Disease Among Miners in the Joplin District, Missouri. Bureau of Mines Bull. 132, 1917, 116 pp.
96. LANZA, A. J., AND CHILDS, SAMUEL B. I. Miners' Consumption: A Study of the Disease Among Zinc Miners in Southwestern Missouri. II. Roentgen-Ray Findings in Miners' Consumption. U. S. Public Health Bull. 85, 1917, 40 pp.
97. HARRINGTON, DANIEL, AND LANZA, A. J. Miners' Consumption in the Mines of Butte, Mont. Bureau of Mines Tech. Paper 260, 1921, 19. pp.
98. KINDALL, CLEVE E. Dust-Ventilation Investigation of the Mines at Tonopah, Nev. Unpublished rept. of Bureau of Mines, U. S. Dept. of the Interior, 1921.
99. SAYERS, R. R., MERIWETHER, F. V., LANZA, A. J., AND ADAMS, W. W. Silicosis and Tuberculosis Among Miners of the Tri-State District of Oklahoma, Kansas, and Missouri. I. For the Year Ended June 30, 1928. Bureau of Mines Tech. Paper 545, 1933, 30 pp.
100. MERIWETHER, F. V., SAYERS, R. R., AND LANZA, A. J. Silicosis and Tuberculosis Among Miners of the Tri-State District of Oklahoma, Kansas, and Missouri. II. For the Year Ended June 30, 1929. Bureau of Mines Tech. Paper, 552, 1933, 28 pp.
101. OFFICE OF INDUSTRIAL HYGIENE AND SANITATION. The Health of Workers in the Dusty Trades. III. Exposure to Dust in Coal Mining. U. S. Pub. Health Bull. 208, 1933, pp. 7–19.
102. BRUNDAGE, DEAN K. Mortality of Coal Miners. U. S. Pub. Health Bull. 210, 1933. 17 pp.
103. SAYERS, R. R., BLOOMFIELD, J. J., DALLAVALLE, J. M., JONES, R. R., DREESSEN, W. C., AND BRITTEN, R. H. Anthraco-Silicosis (Miners' Asthma). Pennsylvania Dept. of Labor and Industry, Spec. Bull. 41, 1934, 81 pp.
104. SAYERS, R. R., BLOOMFIELD, J. J., DALLAVALLE, J. M., JONES, R. R. DREESSEN, W. C., BRUNDAGE, D. K., AND BRITTEN, R. H. Anthraco-Silicosis Among Hard-Coal Miners. U. S. Pub. Health Bull. 221, 1935, 114 pp.
105. KIBBEY, C. H. Relative Pneumonia Fatality Among Surface Workers and Miners. Am. Jour. Pub. Health, vol. 22, No. 4, 1932, pp. 300–306.
106. BLOOMFIELD, J. J. Preliminary Surveys of the Industrial Environment. U. S. Pub. Health Repts., vol. 48, No. 44, 1934, pp. 1343–1351.
107. DIVISION OF INDUSTRIAL HYGIENE, NATIONAL INSTITUTE OF HEALTH, U. S. PUBLIC HEALTH SERVICE AND THE UTAH STATE BOARD OF HEALTH. The Working Environment and the Health of Workers in Bituminous Coal Mines, Nonferrous Metal Mines, and Nonferrous Metal Smelters in Utah, 1940, 310 pp.
108. JONES, R. HAROLD. Pneumoconiosis Encountered in Bituminous-Coal Miners. Jour. Am. Med. Assoc., vol. 119, 1942, pp. 611–615.
109. CONNECTICUT HEALTH BULLETIN. Rock-Drilling Dust Hazard. Vol. 50, 1936, pp. 37–39.
110. PORRO, F. W., PATTON, J. R., AND HORNS, A. A. Pneumoconiosis in the Talc Industry. Am. Jour. Roentgenol. Rad. Ther., vol. 47, 1942, pp. 508–524.
111. GREENBURG, L. Dust Hazard in Tremolite Talc Mining. Yale Jour. Biol. and Med., vol. 19, 1947, p. 481.
112. WINSLOW, C.-E. A., AND GREENBURG, LEONARD. A Study of the Dust Hazard in the Wet and Dry-Grinding Shops of an Ax Factory. U. S. Pub. Health Repts., vol. 35, No. 41, 1920, pp. 2393, 2401.
113. RIDDELL, A. R. Silicosis: Its Relation to Tuberculosis. Pub. Health Jour. (Toronto), vol. 17, 1926, pp. 1–8.
114. CLARK, W. I. The Dust Hazard in the Abrasive Industry: Third Study. Jour. Ind., vol. 13, 1931, pp. 343–346.
115. KESSLER, H. H. Silicosis in the Abrasive-Powder Industry. Am. Jour. Pub. Health, vol. 21, 1931, pp. 1390–1392.
116. CLARK, IRVING W. Health Hazards in the Use of Artificial Abrasive Grinding Wheels. Industrial Hygiene Foundation of America, Inc., Proc. 7th Ann. Meeting, Nov. 10–11, 1942, pp. 26–31.
117. JOURNAL OF AMERICAN MEDICAL ASSOCIATION. Notes and Queries. Vol. 103, 1934, p. 1472. (Quoted by Clark, *see* ref. 114.)

118. Hoffman, F. L. The Problem of Dust Phthisis in the Granite-Stone Industry. U. S. Dept. of Labor, Bur. of Labor Statistics Bull. 293, 1922. 178 pp.
119. Russell, A. E., Britten, R. H., Thompson, L. R., and Bloomfield, J. J. The Health of Workers in Dusty Trades. II. Exposure to Siliceous Dust (Granite Industry). U. S. Pub. Health Bull. 187, 1929, 206 pp.
120. Goodrich, Howard B. Silicosis. Jour. Missouri State Med. Soc., vol 31, 1934, pp. 193–198.
121. Commonwealth of Massachusetts. Report to the General Court of the Special Disease Commission. 1934, 215 pp.
122. McConnell, W. J., and Fehnel, J. W. Health Hazards in the Foundry Industry. Jour. Ind. Hyg., vol. 16, 1934, pp. 227–251.
123. Sander, O. A. Lung Findings in Foundry Workers. A Four-Year Survey. Am. Jour. Pub. Health, vol. 28, 1938, pp. 601–608.
124. Goldberg, Jacob A. Industrial Hygiene, Dusty Foundries. Safety Eng., vol. 77, 1939, pp. 8–10.
125. Lanza, A. J. Silicosis. Wisconsin Med. Jour., October 1941, 3 pp.
126. Merritt, L. M. Ceramic Whiteware Industry Requires Rigid Dust-Control Standards. Occupational Hazards, vol. 3, 1941, pp. 6–9, 30–31.
127. Williams, Charles R. Composition of Industrial Dusts. I. Foundry Dusts. Jour. Ind. Hyg. Toxicol., vol. 27, 1945, pp. 110–114.
128. Thompson, L. R., Brundage, D. K., Russell, A. E., and Bloomfield, J. J. The Health of Workers in Dusty Trades. I. Health of Workers in a Portland-Cement Plant. U. S. Pub. Health Bull. 176, 1928, 138 pp.
129. Russell, A. E. The Effect of Cement Dust Upon Workers. Am. Jour. Med. Sci., vol. 185, 1933, p. 330.
130. Garner, Leroy U., Durkin, Thomas M., Brumfiel, Daniel M., and Sampson, Homer, L. Survey in Seventeen Cement Plants of Atmospheric Dusts and Their Effects Upon the Lungs of Twenty-two Hundred Employees. Jour. Ind. Hyg. Toxicol., vol. 21, 1939, pp. 279–318.
131. Flinn, R. H. Dreessen, W. C., Edwards, T. I., Riley, E. C Bloomfield, J. J., Sayers, R. R., Cadden, John F., and Rothman, S. C. Silicosis and Lead Poisoning Among Pottery Workers. Pub. Health Bull. 244, 1939, 178 pp.
132. Fulton, Wm. B., Butters, F. E., Dooley, A. E., Koppenhaver, F. B., Matthews, J. L., and Kirk, R. M. A Study of Silicosis in the Silica Brick Industry. Bureau of Ind. Hyg., Pennsylvania State Department of Health, 1941, 60 pp.
133. Stalker, William W. Silicosis Exposure in the Fire Brick Industry. I. Engineering Study. Jour. Ind. Hyg. Toxicol., vol. 27, 1945, pp. 275–282.
134. Ritter, Wayne L., and Bovard, Paul G. Silicosis Exposures in the Fire Brick Industry. Jour. Ind. Hyg. Toxicol., vol. 27, 1945, p. 283.
135. Moore, Keith R. Silicosis in Australia. (*See* ref. 19, pp. 296–312.)
136. ———. Fibrosis of the Lungs in South Coast Coal Miners, New South Wales. Health, Commonwealth Dept. of Health, vol. 9, No. 5, 1931, 10 pp.
137. Workers' Compensation Commission of New South Wales. Third Annual Report and Statistics of the Workers' Compensation Commission of New South Wales for the Year July 1, 1928, to June 30, 1929, Dec. 18, 1929, 78 pp.
138. Nelson, W. T. Report on an Investigation of the Pulmonary Conditions of Mine Employees, Western Australia, During the Years 1925–26. Commonwealth of Australia, Dept. of Health, Ser. Pub. 5 (Div. Ind. Hygiene), 1927, 99 pp.
139. South African Mining and Engineering Journal. Silicosis in Western Australia, Vol. 11, part 11, No. 2507, p. 647.
140. Elliott, J. H. Silicosis in Ontario Gold Mines. (Quoted by J. G. Cunningham in Silicosis in Canada, *see* ref. 19, pp. 317–337.)
141. Hague, O. G., and McBain, R. W. Silicosis as an Industrial Hazard in Ontario Gold Mines. Am. Jour. Roentgenol., vol. 18, 1927, pp. 315–322.
142. Canadian Mining Journal. Silicosis in Ontario. Vol. 62, 1941, pp. 316–317.
143. Kranenburg, W. R. H. Silicosis in the Netherlands. (*See* ref. 19, pp. 512–534.)
144. Vossenaar (or Heeslen) and Dounrow (or Paris). Premiers resultats d'une enquete sur les affections des voies respiratoires chez les mineurs des charbonnages des Pays-Bas. [First Results of an Inquiry Regarding the Affections of the Respiratory Passages of Coal Miners of the Low Countries.] Bull. Acad. Med., vol. 115, 1936, pp. 300–307.

145. Langelez, A. Belgian Miners' Silicosis. Proc. Internat. Conf. Aug. 29–Sept. 9, 1938. Studies and Repts. Series F (Industrial Hygiene), No. 17, Geneva, 1940, pp. 140–150.
146. Mey, A. Periodiek röntgenologisch Longonderzoek bij Steenarbeiders van de Staatsmijnen. Een Overzicht Van Het Tijdvak Februari 1936 Tot Februari 1946. [Periodic Röntgenologic Examination of the Lungs of Rock Workers in the State Mines. A Survey of the Period February 1936 to February 1946.] Communication 1 of Medical Service of the Netherlands Collieries, 1947, 118 pp. (p. 31 for summary).
147. Møller, P. F. [On Silicosis in Porcelain Workers, from the Roentgenological and Some Clinical Points of View.] Acta Radiologica, vol. 15, 1934. Abs. in Bull. Hyg., vol. 10, 1935, p. 83.
148. Stub-Christensen, V. Silicose dans l'industrie danoise. [Silicosis in Danish Industry.] Med. travail, vol. 12, 1940, pp. 12–44.
149. Riembault, A. Hygiene des ouvriers mineurs dans les exploitations houillères. [Hygiene of Coal-Mine Workers.] Paris, 1861, 316 pp.
150. Martin, Edmé, and Roche, Louis. Resultats d'une enquete effectuée sur des mineurs au rocher du bassin de la Loire. [Results of an Inquiry of the Rock Miners of the Loire Basin.] Arch. malad. prof. med. trav. hyg. tox. sec. indust., vol. 6, 1944–1945, pp. 65–69.
151. Policard, A., Hanaut, A., and Leroy (Lyon).—La question des pneumoconioses par lignite. Enquete dans une exploitation de la Fuveau (Bouches-du-Rhone) Basin. [The Question of Pneumoconioses from Lignite. Inquiry into an Operation in the Fuveau Basin (Bouches-du-Rhone).] Arch. Malad. Prof. Med. Trav. Hyg. Tox. Sec. Indust., vol. 6, 1944–1945, pp. 61–66.
152. Mining Journal. Silicosis in the Kolar. Vol. 226, 1946, p. 506.
153. Iron and Coal Trades Review. Silicosis, Pneumoconiosis and Dust Suppression in Mines. Résumé of Papers Read at London Conference, Apr. 25, 1947, pp. 729–730.
154. Barenbrug, A. W. T. Mining at Depth on the Kolar Gold Fields, South India. Jour. Chem., Met., and Min. Soc. of South Africa, vol. 48, 1948, pp. 5–27.
155. Loriga, Giovanni. [Pneumoconiosis in Italy.] (*See* ref. 19, pp. 481–511.)
156. Biondi, C. (Quoted by Loriga, *see* ref. 155.)
157. Frongia, —. (Quoted by Loriga, *see* ref. 155.)
158. Pesenti, Rota, and Finzi. (Quoted by Loriga, *see* ref. 155.)
159. Bianchi, G. (Quoted by Loriga, *see* ref. 155.)
160. Turano, L. (Quoted by Loriga, *see* ref. 155.)
161. Parmeggiani, Luigi. Considerazioni sui casi di silicosi oggetto di controversie guidiziarie nella provincia di Milano prima dell assicurazione obbligatoria. [Considerations on Cases of Silicosis Subject of Judicial Controversy in the Province of Milan Before Compulsory Insurance.] Med. del Lavoro, vol. 37, 1946, 249–264.
162. Moshkovsky, I. I. Tuberculosis and Pneumokoniosis Among Underground Workers, 1941. Abs. Ind. Hyg., vol. 17, 1942, pp. 74–76.
163. Teixeira, Carlos Martins, Curty, Manoel, Maredo, Ephrem, and Barbosa, Octavio. Higiene das Minas de Ouro, Silicosa Morro Velho, Minas Gerais [Hygiene of the Gold Mines, Silicosis Moro Velho, Minas Gerais.] Bull. 44, Ministry of Agriculture, Rio de Janeiro, 1940, 158 pp.
164. Labbé, V. V., Reyes, A. P., Zuniga, M., Schuller, H. P., and Garcia, C. Victoria. Revision de 200 casos de silicosis pulmonar confirmada. [Review of 200 Cases of Verified Pulmonary Silicosis.] Bol. Medico Social, vol. 13, 1946, pp. 160–165.
165. Bloomfield, J. J. Industrial Hygiene Problems in Bolivia, Peru, and Chile. Pub. Health Bull. 301, 1948, 139 pp.
166. Oiler, A. Die Silicosis in Spanien. [Silicosis in Spain.] Arch. e. Gewerbepath., vol. 7, 1936, pp. 383–387.
167. Greinacher-Christofari, Vera. Die Mineur-Silikose in der Schweiz. [Silicosis of Miners in Switzerland.] Inaug. Dissert. Medical Dept. of SUVA, Zurich, 1945, 28 pp.
168. Lang, Fritz. Prophylaxie medicale de la silicose. [Medical Prophylaxis of Silicosis.] Communications of Medical Service, No. 19, Lucerne, 1946, 100 pp.
169. van Mechelen, Victor. Enquete sur le probleme de la silicose en suisse. [Investigation on the Problem of Silicosis in Switzerland.] Communication 17, Institute d' Hygiene des Mines, 1946, 13 pp.

170. HARRINGTON, DANIEL. Dust and the Health of Miners. Presented before Am. Inst. Min. and Met. Eng., New York meeting, February 1924, 5 pp.
171. BALLANTYNE, HORATIO. Silicosis and the Industrialist. Jour. State Med. (London), vol. 40, 1932, pp. 324–349.
172. SILICOSIS (MEDICAL-ARRANGEMENTS) COMMITTEE. Report of the Departmental Committee Appointed by the Secretary of State to Advise as to the Medical Arrangements Which Could be Made for the Diagnosis of Silicosis. Home Office, London, 1929, 19 pp.
173. LEVEY, SIMON A. Roentgenological Consideration of Silicosis. Jour. Missouri Med. Assoc., vol. 31, 1934, pp. 189–193.
174. BADHAM, CHARLES. Notes on a Fine Type of Fibrous Pneumoconiosis Produced by Silicates and Other Minerals. Rept. of Director General of Public Health, New South Wales, for year ended Dec. 31, 1927, Studies in Ind. Hyg., No. 13, Sydney, 1929, pp. 102–110.
175. COLLIS, E. L. Occupational Dust Diseases. Bull. Hyg., vol. 6, 1931, pp. 663–670.
176. COLLIS, E. L., AND GILCHRIST, J. C. Effects of Dust Upon Coal Trimmers. Jour. Ind. Hyg., vol. 12, 1930, pp. 266–280.
177. GOODING, C. G. Pneumoconiosis in South Wales Anthracite Miners. Jour. Am. Med. Assoc., vol. 133, 1947, p. 894.
178. IRON AND COAL TRADES REVIEW. Silicosis: Discussion on a Paper in Which Sericite Was Suggested as a Causative Factor. Vol. 128, 1934, p. 331.
179. GARDNER, L. U. The Effects of Inhaled Mineral Dusts. Nat. Safety News, vol. 27, 1933, pp. 34–36.
180. PENDERGRASS, EUGENE P., AND LEOPOLD, SIMON S. Benign Pneumoconiosis. Jour. Am. Med. Assoc., vol. 127, 1945, pp. 701–705.
181. DUNNER, L., AND BAGNALL, J. T. Graphite Pneumoconiosis Complicated by Cavitation Due to Necrosis. British Jour. Radiol., vol. 19, 1946, pp. 133–172. Abs. in Jour. Am. Med. Assoc., vol. 131, 1946, p. 796.
182. ROVIDA, L. [A Case of Silicosis of the Lungs, with Chemical Analysis.] Ann. di chim. appl. a med. Milani, vol. 53, 3 s., 1871.
183. KETTLE, E. H. The Action of Harmful Dusts: Some Pathological Aspects of Silicosis. Coll. Guard. vol. 148. 1934, pp. 1096–1098.
184. GARDNER, L. U., MIDDLETON, E. L., AND ORENSTEIN, A. J. Report upon the Medical Aspects of Silicosis, Including Etiology, Pathology, and Diagnostics. (See ref. 19, pp. 80–89.)
185. SAYERS, R. R. The Clinical Manifestations of Silicosis. Jour. Am. Med. Assoc., vol. 101, 1933, pp. 580–583.
186. GARDNER, L. U. The Pathology of Various Mineral-Dust Diseases. Safety Eng., vol. 67, 1934, pp. 109–112.
187. BELT, THOMAS H., AND KING, EARL J. Tissue Reactions Produced Experimentally by Selected Dusts from South Wales Coal Mines. Chronic Pulmonary Disease in South Wales Coal Miners. III. Experimental Studies. Spec. Rept. Ser. 250, 1945, pp. 29–68.
188. KASTEN, W. [Phagocytosis of Coal and Stone Dust in Vitro.] Arch. Gewerbepath., vol. 9, 1939, pp. 337–345. (Abs. by Air Hygiene Foundation of America, Inc., Oct. 17, 1939, p. 8.)
189. SIMSON, F. W., STRACHAN, A. S., AND IRVINE, L. G. Silicosis in South Africa: A Symposium on the Histopathology, Pathological Anatomy, and Radiology of the Disease. Proc. Transvaal Mine Med. Off. Assoc., spec. supp., 1930, 44 pp.
190. POLICARD, A. Mécanismes et consequences de l' accumulation des poussières dans les gaines bronchiques et certerielles au cours des pneumoconioses. [Mechanisms and Consequences of the Accumulations of Dusts in the Bronchial and Arterial Tissues on the Course of the Pneumoconioses.] Presse Medicale, vol. 55, 1947, pp. 213–214.
191. PROSKE, H. O., AND SAYERS, R. R. Pulmonary Infection in Pneumoconiosis. I. A Bacteriologic and Experimental Study. U. S. Public Health Repts. vol. 49, 1934, pp. 839–858.
192. TILLSON, B. F. Silicosis: Its Causation. Eng. and Min. Jour., vol. 135, 1934, pp. 121–124.
193. RIDDELL, A. R. The Clinical Aspects of Simple Silicosis and Silicosis With Tuberculosis. Am. Rev. Tuberculosis, vol. 29, 1934, pp. 36–42.
194. JOURNAL OF AMERICAN MEDICAL ASSOCIATION. Silicosis. For. Letters, vol. 102, 1934, p. 1861.

195. Croizier, Louis, Martin, Edmé, and Policard, Albert. La fibrose pulmonaire des mineurs. [The Pulmonary Fibrosis of Miners.] Pub. by Masson et Cie, editors, Libraire de l'Academie de Medecine, Paris, 1938, 214 pp.
196. Gardner, Leroy U. Pathological Anatomy, Fourth Saranac Laboratory Symposium on Silicosis. Saranac Lake, N. Y., June 19–23, 1939, 379 pp.
197. Gudjonsson, Sk. V. An Examination of 300 Workers in Granite and Sandstone Quarries. Jour. Ind. Hyg., vol 18, 1936, pp. 215–221.
198. Fisher, S. W. Dust Diseases Amongst the British Coal Miners. Colliery Guardian, vol. 174, 1947, pp. 481–482.
199. International Labour Office. Silicosis. Records of the Int. Conf. held at Johannesburg 13–27 August 1930. Studies and Reports, Series F (Industrial Hygiene), No. 13, Geneva, 1930, 692 pp.
200. Lanza, A. J. (Ed.). Silicosis and Asbestosis. Oxford University Press, New York, 1938, 439 pp.
201. Sayers, R. R., and Lanza, A. J. Pneumoconiosis. Am. Public Health Assoc. Yearbook, 1932–33., supp. to Am. Jour. Public Health, vol. 23, 1933, pp. 100–102.
202. Pancoast, H. K., and Pendergrass, E. P. The Roentgenological Aspects of Pneumoconiosis and Its Medicolegal Importance. Jour. Ind. Hyg., vol. 15, 1933, pp. 117–135.
203. Irvine, L. G. The Clinical Pathology of Silicosis. (*See* ref. 19, pp. 240–269.)
204. Pancoast, H. K., and Pendergrass, E. P. Pneumoconiosis: The Importance of Accuracy in Roentgenological Interpretation. New England Jour. Med., vol. 209, 1933, pp. 425–437.
205. Haldane, J. S. Silicosis: Further Contribution to Discussion. Iron and Coal Trades Rev., vol. 128, 1934, p. 640.
206. Smith, Adelaide R. Silicosis Among Rock Drillers, Blasters, and Excavators in New York City, Based on a Study of 208 Examinations. Jour. Ind. Hyg., vol. 11, 1929, pp. 37–81.
207. ———. The Unreliability of the Physical Examination in the Diagnosis of Silicosis. Ind. Med., vol. 3, 1934, p. 277.
208. McNally, W. D. Silicon Dioxide Content of the Lungs in Health and Disease. Jour. Am. Med. Assoc., vol. 101, 1933, pp. 584–587.
209. Sayers, R. R. Pneumoconiosis. Am. Public Health Assoc. Yearbook, 1934–36, pp. 65–70.
210. Koppenhöfer, G. F. Untersuchungen zur Pathogenese silikotischer Gewebeveränderungen. Silikotischen Gewebe. [Studies on the Pathogenesis of Silicotic Tissue Changes. II. Morphological Investigations on the Content of Quartz Dust in Silicotic Tissue.] Arch. Gewerbepath., vol. 6, 1935, pp. 18–37.
211. Peacock, T. B (Quoted by McNally, *see* ref. 208.)
212. Schmidt, —. (Quoted by McNally, *see* ref. 208.)
213. Woskressensky, —. (Quoted by McNally, *see* ref. 208.)
214. Gardner, Leroy U. Employability of Silicotic Subjects. Occupational Medicine, vol. 4, 1947, pp. 17–21.
215. Myers, W. M. Solubility of Finely Divided Rock Dusts in Water, Kerosine, and Alcohol. Bureau of Mines Rept. of Investigations 2548, 1932, 6 pp.
216. Heffernan, P. Some Notes on the Biophysics of Silica and the Etiology of Silicosis. British Med. Jour., no. 3584, 1929, pp. 489–492.
217. Kraut, H. Über den Kieselsäuregehalt des menschlichen Blutes und seine Veränderungen durch Kieselsäurezufuhr. [Silicic Acid Content of Human Blood and Its Change Through the Administration of Silicic Acid.] Hoppe-Seyler's Ztschr. physiol. Chem., vol. 194, 1931, pp. 81–97.
218. Lieb, H., and Schadendorff, E. [Silicic Acid Content of Lungs of Silicon Workers in Steiermark.] Arch. Gewerbepath. Gewerbehyg., vol. 4, 1933, pp. 576–615. Chem. Abs., vol. 28, 1934, p. 2068.
219. Smith, C. S., and Wikoff, Helen L. The Silica Contents of the Lungs of a Group of Tunnel Workers. Am. Jour. Pub. Health, vol. 23, 1933, pp. 1250–1254.
220. Coal Age. Notes from Across the Sea. Vol. 39, 1934, p. 142.
221. Travers, M. W. The Effect of Grinding on the Solubility of Mineral Substances, and on the Toxic Character of the Resulting Dusts. Chem. and Ind., vol. 58, London, 1939, p. 226. Abs. Am. Air. Hyg. Found., Inc., 1939, p. 5.

222. Ulrich, Hans. A. Originalabhandlung Enstehung der Silikose vom Standpunkt des Mineralogen. [A. Original Treatise. Origin of Silicosis from the Standpoint of Mineralogy.] Zentralb. Gewerbehyg. u. Unfallverh., vol. 22, 1935, pp. 81–87.
223. King, E. J. Solubility Theory of Silicosis. Occupational Medicine, vol. 4, 1947, pp. 26–49.
224. Gardner, L. U. Studies on the Relation of Mineral Dusts to Tuberculosis. III. Am. Rev. Tuberc. 1923, vol. 7, p. 344.
225. Gye, W. E., and Purdy, W. J. The Poisonous Properties of Colloidal Silica. I. The Effects of the Parenteral Administration of Large Doses. British Jour. Exper. Path. 1922, vol. 3, p. 75.
Gye, W. E., and Kettle, E. H. Silicosis and Miners' Phthisis, p. 241.
226. Gye, W. E., and Purdy, W. J. The Poisonous Properties of Colloidal Silica; II. Effects of Repeated Intravenous Injections on Rabbits. British Jour. Exper. Path. 1924, vol. 5, p. 238.
227. Gardner, L. U., and Cummings, D. E. Reaction to Fine and Medium-Sized Quartz and Aluminum Oxide Particles. Silicotic Cirrhosis of Liver. Am. Jour. Path. 1933, vol. 9, p. 751.
228. Policard, A. Sur la pathogenie de la silicose pulmonaire: Mode de formation du nodule silicotique. [Pathogenesis of Pulmonary Silicosis: Manner of Formation of Silicotic Nodules.] Presse med., 1933, vol. 41, p. 89.
229. Kettle, E. H. Interstitial Reactions Caused by Various Dusts and their Influence on Tuberculosis Infections. Jour. Path. and Bact., 1932, vol. 35, p. 395.
230. Loriga, G., Badham, C., and Roberts, F. G. A. Report of Subcommittee on Preventive Measures. (*See* ref. 19, p. 94.)
231. Colliery Guardian. Silicosis: The Sericite Theory. Vol. 149, 1934, pp. 662–664.
232. Cummings, Donald E. The Etiology of Silicosis. Proc. 4th Saranac Laboratory Symposium on Silicosis, 1939, pp. 20–34.
233. Hatch, Theodore. Industrial Dusts and Their Control. Safety Eng., vol. 68, 1934, pp. 261–262, 264.
234. Gardner, Leroy U. "Mixed" Dusts and "Protector" Dusts. Air Hygiene Foundation of America, Inc., Proc. Special Meeting of Members, Nov. 30, 1937, pp. 25–27.
235. Hurlbut, C. S. Petrography in the Study of Occupational Diseases. Safety Eng., vol. 68, 1934, pp. 73–74.
236. Smith, Adelaide R. Review of Silicosis. New York State Dept. of Labor, Ind. Bull., vol. 12, 1933, pp. 32–33.
237. Jones, W. R. Silicotic Lungs: The Minerals They Contain. Jour. Hyg. vol. 33, 1933, pp. 307–320; Min. Mag., vol. 49, 1933, pp. 67–68.
238. ———. Silicosis. Jour. Chem., Met. and Min. Soc. South Africa, September 1933, pp. 99–123.
239. Irvine, L. G. (Discussion of paper by Jones, *see* ref. 237.)
240. Mavrogordato, A. (Discussion of paper by Jones, *see* ref. 237.)
241. Iron and Coal Trades Review. Silicosis: Sericite Suggested as the Causative Factor. Vol. 128, 1934, pp. 204–205.
242. Mitchell, A. R. (Discussion of paper by Jones, *see* ref. 237, pp. 130–161.)
243. Trotter, R. M. (Discussion of paper by Jones, *see* ref. 237.)
244. Industrial Medicine. Sericite as a Cause of Silicosis. Vol. 3, 1934, pp. 107–108.
245. Lemon, W. S., and Higgins, G. M. The Tissue Reactions of the Lungs to the Intratracheal Injection of Particulate Sericite. An Experimental Study. Am. Rev. Tuber., vol. 32, 1935, pp. 243–250.
246. Faillon, J. T., and Banting, F. G. Tissue Reaction to Sericite. Canadian Med. Assoc. Jour., vol. 33, 1935, pp. 407–411.
247. Russell, A. E. Occupation and Respiratory Disease. Southern Med. Jour., vol. 25, 1932, pp. 919–927.
248. Rao, S. S. Silicosis in India. British Med. Jour., June 16, 1934, p. 1094. Jour. Ind. Hygiene, vol. 16, 1934, pp. 101–102.
249. King, Earl J. Chronic Pulmonary Disease in South Wales Coal Miners. III. Experimental Studies: E. The Solubility of Dusts From South Wales Coal Mines. Med. Res. Coun. Spec. Report Ser. 250, London, 1945, pp. 69–94.
250 Canadian Mining Journal. Liquids in Quartz. Vol. 55, 1934, p. 406.
251. Iron and Coal Trades Review. Causes of Silicosis. Vol. 129, 1934, p. 890.

252. KNOPF, ADOLPH The Quantitative Determination of Quartz ("Free Silica") in Dusts. Public Health Repts., Repr. 1560, vol. 48, 1933, pp. 183–190.
253. METROPOLITAN LIFE INSURANCE CO. (INDUSTRIAL HEALTH SECTION). Silicosis. 32 pp. (Undated pamphlet.)
254. POLICARD, A. The Action of Mica Dust on Pulmonary Tissue. Jour. Ind. Hyg., vol. 16, 1934, pp. 160–164.
255. COLLIS, E. L. (Comment on Policard's article, ref. 254.) Bull. Ind. Hyg., vol. 9, 1934, p. 513.
256. LECLERQ, J. [Pulmonary Silicosis Among the Coal Miners of the North and Pas-de-Calais.] Med. du Trav., vol. 5, 1933, pp. 222–260. Abs. Jour. Ind. Hyg., vol. 16, 1934, p. 32.
257. POPE, A. S. Workers in Dust. Safety Eng., vol. 58, 1934, p. 80.
258. PIT AND QUARRY. Nuisance Dust May Be Source of Profit. Most Danger in Fine Particles. Vol. 28, 1936, p. 45.
259. BEDFORD, T., AND WARNER, C. G. Chronic Pulmonary Disease in South Wales Coalminers. II. Environmental Studies. B. Physical Studies of the Dust Hazard and of the Thermal Environment in Certain Coal Mines. Med. Res. Coun. Spec. Rept. Ser. No. 244, London, 1943, 222 pp.
260. PIROW, HANS. (*See* ref. 19, pp. 26–44.)
261. MAVROGORDATO, A. (*See* ref. 19.)
262. FINDEISEN, W. [The Deposit in the Human Lungs of Small Particles Suspended in Air.] Arch. ges. Physiol. (Pflüger's), vol. 236, 1935, pp. 367–379. Abs. Jour. Ind. Hyg. and Toxicol., vol. 18, 1936, pp. 39–40.
263. BADHAM, C., RAYNER, H. E. G., AND BROOKE, H. D. (*See* ref. 19.)
264. GREENBURG, L. Studies on the Industrial Dust Problem. II. A Review of the Methods Used for Sampling Aerial Dust. Public Health Repts., Repr. 1004, vol. 40, 1925, pp. 765–786.
265. HATCH, THEODORE T., AND KINDSVATTER, VICTOR H. Lung Retention of Quartz Dust Smaller Than One-half Micron. Jour. Ind. Hyg. and Toxicol., vol. 29, 1947, pp. 342–346.
266. GUDJONSSON, SK. V. A Study of 78 Workers Exposed to Inhalation of Cryolite Dust. Jour. Ind. Hyg., vol. 15, 1933, pp. 27–33.
267. COLLIERY GUARDIAN. Law Intelligence. Workmen's Compensation: Silicosis. Vol. 151, 1935, p. 1156.
268. BADHAM, C. (*See* ref. 19.)
269. BLOOMFIELD, J. J. A Study of the Efficiency of Dust-Removal Systems in Granite-Cutting Plants. Public Health Repts., Repr. 1324, vol. 44, 1929, pp. 2505–2522.
270. INDUSTRIAL COMMISSION OF WISCONSIN. General Orders on Dusts, Fumes, Vapors, and Gases, Effective Mar. 18, 1932. Madison, 22 pp.
271. MINING AND METALLURGY. Miners' Health. Vol. 16, 1935, pp. 290–291.
272. BRUNDAGE, D. K., AND FRASER, ELIZABETH S. The Health of Workers in Dusty Trades. III. Exposure to Dust in Coal Mining. U. S. Public Health Bull. 208, 1933, pp. 7–19.
273. MOIR, J. (Quoted by Tillson, *see* ref. 192.)
274. BLOOMFIELD, J. J. (Quoted by Tillson, *see* ref. 192.)
275. BADHAM, C. (Quoted by Tillson, *see* ref. 192.)
276. MCCRAE, J. (Quoted by Tillson, *see* ref. 192.)
277. JONES, W. R. (Quoted by Tillson, *see* ref. 192.)
278. WATKINS-PITCHFORD, W. The Silicosis of the South African Gold Mines and Changes Produced in It by Legislative and Administrative Efforts. Jour. Ind. Hyg., vol. 9, 1927, pp. 109–139.
279. JEPHCOTT, C. M. Some Aspects of the Chemistry of Silicosis. Canadian Public Health Jour., vol. 26, 1935, pp. 606–610. Abs. Jour. Ind. Hyg. and Toxicol., vol. 18, 1936, p. 39.
280. HAMLIN, I. E. Rocky Mt. Med. Jour., vol. 41, 1944, pp. 301–309.
281. IRON AND COAL TRADES REVIEW. Pneumoconiosis of Coal Workers, Relation of Silica Particle Size and Concentrations to Development of Fibrosis of the Lungs. Vol. 147, Sept. 10, 1943, pp. 401–403.
282. OFFICE OF INDUSTRIAL HYGIENE AND SANITATION. Anthraco-Silicosis Among Hard-Coal Miners. U. S. Pub. Health Bull. 221, 1935, 14 pp.
283. DIVISION OF INDUSTRIAL HYGIENE, NATIONAL INSTITUTE OF HEALTH AND DIVISION OF INDUSTRIAL HYGIENE, NORTH CAROLINA STATE BOARD OF HEALTH. Pneumoconiosis Among Mica and Pegmatite Workers. U. S. Public Health Bull. 280, 1940, 74 pp.

284. TELEKY, L. Die Silicose. [Silicosis.] Wien. Med. Wochenschr., vol. 86, 1936, pp. 1351–1354, 1382–1387.
285. COMMITTEE ON THRESHOLD LIMITS. Maximum Allowable Concentration Values. Am. Conf. Govt. Ind. Hyg., 1947, 6 pp.
286. DIVISION OF INDUSTRIAL HYGIENE. Silicosis and Its Prevention. New York State Dept. of Labor, Spec. Bull. 198, 1938, p. 198.
287. ——— Operations to Protect Workers' Health During the Year 1939. New York State Dept. of Labor, Ind. Bull. vol. 19, 1940, pp. 63–66.
288. BOARD OF STANDARDS AND APPEALS. Rules Relating to the Control of Silica Dust in Stone Crushing Operations. New York State Dept. of Labor Code Rule, Effective July 1, 1942, No. 34, 1941, 18 pp.
289. GARDNER, LEROY U. Elements of Diagnosis and Prognosis in Pneumoconiosis. Rocky Mt. Med. Jour., vol. 41, 1944, pp. 385–391.
290. SUTHERLAND, C. L., AND BRYSON, S. Report of the Incidence of Silicosis in the Pottery Industry. Home Office, London, 1926, 52 pp.
291. SAPPINGTON, C. O. Silicosis and Other Dust Disease with Which Industry Must Cope. Pit and Quarry, vol. 24, 1932, p. 81.
292. MOORE, K. R. (*See* ref. 19.)
293. BÖHME, A. (*See* ref. 19.)
294. KOELSCH, F. (*See* ref. 19.)
295. CUNNINGHAM, J. G. (*See* ref. 19.)
296. MIDDLETON, E. L. (*See* ref. 19.)
297. MCFARLAND, W. Silicosis and Tuberculosis as Seen in the Granite Workers in Barre, Vt. Jour. Ind. Hyg., vol. 9, 1927, pp. 315–330.
298. HAYHURST, E. R., KINDEL, D. J., NEISWANDER, B. E., AND BARRETT, C. D. Silicosis with Low Incidence of Tuberculosis. Jour. Ind. Hyg., vol. 11, 1920, pp. 228–244.
299. CHAPMAN, E. M. Acute Silicosis. Jour. Am. Med. Assoc., vol. 98, 1932, pp. 1430–1440.
300. MACDONALD, G., PIGGOT, A. P., AND GILDER, F. W. Two Cases of Acute Silicosis. Lancet, vol. 2, 1930, p. 846. (Quoted by Chapman, *see* ref. 299.)
301. RUSSELL, A. E. (Quoted by Chapman, *see* ref. 299.)
302. BRITTON, J. A., AND HEAD, J. R. Pneumoconiosis: The Delayed Development of Symptoms. Jour. Am. Med. Assoc., vol. 96, 1931, pp. 1038–1039.
303. CHIEF INSPECTOR OF FACTORIES. Annual Report for the Year 1945. 1946, pp. 78–79.
304. KILGORE, E. S. Pneumoconiosis, an Unusually Acute Form. Jour. Am. Med. Assoc., vol. 99, 1932, pp. 1914–1916.
305. QUAINTANCE, P. A., AND MORRIS, F. J. Pneumoconiosis. California and West. Med., vol. 40, 1934, pp. 337–340. Abs. Ind. Med., vol. 3, 1934, pp. 349–350.
306. DREESSEN, W. C. Effects of Certain Silicate Dusts on the Lungs. Jour. Ind. Hyg., vol. 15, 1933, pp. 66–78.
307. SMITH, ADELAIDE R. A Study of Granite Cutting and Granite Cutters in the Vicinity of New York City. Am. Jour. Pub. Health, vol. 24, 1934, pp. 821–834.
308. COLLIERY GUARDIAN. Factory Inspection in 1933. Vol. 149, 1934, pp. 267–268.
309. BLOOMFIELD, J. J., AND DREESSEN, WALDEMAR C. Silicosis Among Granite Quarriers. Safety Eng., vol. 68, 1934, pp. 77–78.
310. SUBCOMMITTEE OF THE COMMITTEE ON LABOR, HOUSE OF REPRESENTATIVES. An Investigation Relating to Health Conditions of Workers Employed in the Construction and Maintenance of Public Utilities. 74th Cong., H. J. Res. 449, 1936, 2003 pp.
311. KURODA, S. Hygienic and Clinical-Roentgenological Investigations of Pulmonary Silicosis in a Japanese Foundry. Japan, 1935. Abs. Jour. Ind. Hyg., vol. 18, 1936, pp. 27–28.
312. MARTIN, EDMÉ, AND ROCHE, LOUIS. Resultats d'une enquete effectuée sur des mineurs au rocher du bassin de la Loire. [Results of a Study of the Rock Miners of the Loire Basin.] Med. Trav. Hyg., Toxicol. et Sec. Ind., vol. 6, 1944–1945, pp. 65–69.
313. MAVROGORDATO, A. The Value of the Konimeter—Being an Investigation Into the Methods and Results of Dust Sampling as at Present Practiced in the Mines of the Witwatersrand, South African Inst. Med. Res., 1923, 71 pp.
314. GREEN, H. L. Recent Developments in Methods of Sampling Dusts. Inst. Min. and Met. Bull. 362 (London), November 1934, 21 pp.
315. BROWN, CARLTON E., AND SCHRENK, H. H. Standard Methods for Measuring Extent of Atmospheric Pollution. Bureau of Mines Inf. Circ. 7210, 1942, 19 pp.

316. YALE JOURNAL OF BIOLOGY AND MEDICINE. Dust Hazard in Talc Mining. Vol. 19, 1947, pp. 493, 499–500.
317. COULIER, —. [On A New Property of the Air.] Jour. Pharm., vol. 22, 1875, pp. 165–254. (Quoted by Greenburg, *see* ref. 264.)
318. AITKEN, JOHN. On the Number of Dust Particles in the Atmosphere. Trans. Roy. Soc. Edinburgh, vol. 35, 1889, pp. 1–20. (Quoted by Greenburg, *see* ref. 264.)
319. ———. On the Number of Dust Particles in the Air of Certain Places in Great Britain and on the Continent. Nature, vol. 41, no. 1061, p. 394. (Quoted by Greenburg, *see* ref. 264.)
320. COHEN, J. B. The Air of Towns, Smithsonian Misc. Coll., No. 1073, 1896. (Quoted by Greenburg, *see* ref. 264.)
321. HILL, E. V. Quantitative Determination of Air Dust. Heat. and Vent. Mag., vol. 14, 1917, pp 23–33. (Quoted by Greenburg, *see* ref. 264.)
322. OWENS, J. S. Suspended Impurity in the Air. Proc. Roy. Soc. (London), S. O. 101, 1922, p. 18. Atmospheric Dust. Jour. Soc. Chem. Ind., vol. 41, 1922, p. 436R. Jet Dust-Counting Apparatus. Jour. Ind. Hyg., vol. 4, 1923, p. 522. (Quoted by Greenburg, *see* ref. 264.)
323. FEHNEL, J. W. Methods of Determining Dust Concentrations. Safety Eng., vol. 47, 1934, pp. 217–220. (Quoted by Greenburg, *see* ref. 264.)
324. STACY, —. (Quoted by Greenburg, *see* ref. 264.)
325. HILL, E. V. Ventilation Division of the Health Department, Chicago, Ill. Trans. Am. Soc. Heat. and Vent. Eng., vol. 19, 1913, pp. 412–434. (Quoted by Greenburg, *see* ref. 264.)
326. KNOWLES, R. R Dust Determinations in Air and Gases. Trans. Am. Soc. Heat. and Vent. Eng., vol. 25, 1919, pp. 101–132. (Quoted by Greenburg, *see* ref. 264.)
327. AMERICAN SOCIETY OF HEATING AND VENTILATING ENGINEERS. Preliminary Report of the Committee for Standardizing Methods for Testing Air Washers. Trans., vol. 20, 1914, pp. 425–441. (Quoted by Greenburg, *see* ref. 264.)
328. TOBB, J. V. A New Method of Determining the Dust in Air and in Fresh-Air Schoolrooms. New York Med. Jour., vol. 99, 1914, pp. 426–429. (Quoted by Greenburg, *see* ref. 264.)
329. SARGENT, C. E. The Testing of Inflammable Gases. Iron Trades Rev., vol. 40, 1907, p. 78. (Quoted by Greenburg, *see* ref. 264.)
330. MÖLLER, K. [Quantitative Determination of the Dust Content of the Air.] Gesundh. Ing., vol. 17, 1894, p. 373. (Quoted by Greenburg, *see* ref. 264.)
331. HUBENDICK, E. [Dust Determination in Blast-Furnace Gas.] Ztschr. angew. Chem., vol. 1, 1900, p. 350. (Quoted by Greenburg, *see* ref. 264.)
332. JOHANNSEN, O. [Regarding Dust Determination in Blast-Furnace Gas.] Stahl u. Eisen, vol. 32, 1912, p. 16. (Quoted by Greenburg, *see* ref. 264.)
333. Nesbitt, C. T. A Simple Apparatus for Estimating Dust in Gas. Chem. News, vol. 106, 1912, p. 235. (Quoted by Greenburg, *see* ref. 264.)
334. BROWN, CARLTON E. Filter-Paper Method for Obtaining Dust-Concentration Results Comparable to Impinger Results. Bureau of Mines Rept. of Investigations, 3788, 1944, p. 20.
335. KERSHAW, J. B. C. Dust, Soot, and Smoke. Cassiers Mag., vol. 39, 1911, pp. 250–257. (Quoted by Greenburg, *see* ref. 264.)
336. FRANKLAND, L. F. A New Method for the Quantitative Estimation of the Micro-organisms Present in the Atmosphere. Phil. Trans. Roy. Soc. (London), vol. 178, 1886, pp. 113–152. (Quoted by Greenburg, *see* ref. 264.)
337. MINERS' PHTHISIS PREVENTION COMMITTEE, UNION OF SOUTH AFRICA. General Report. 1916, p. 20, app., p. 66. (Quoted by Greenburg, *see* ref. 264.)
338. HIGGINS, E., LANZA, A. J., LANEY, F. B., AND RICE, G. S. Siliceous Dust in Relation to Pulmonary Disease Among Miners in the Joplin District, Missouri. Bureau of Mines Bull. 132, 1917, 116 pp.
339. FIELDNER, A. C., KATZ, S. H., AND LONGFELLOW, E. S. The Sugar-Tube Method of Determining Rock Dust in Air. Bureau of Mines Tech. Paper 278, 1921, 42 pp.
340. PALMER, G. T. A New Sampling Apparatus for the Determination of Aerial Dust. Am. Jour. Pub. Health, vol. 6, 1916, pp. 54–55. (Quoted by Greenburg, *see* ref. 264.)
341. MEYER, A. L. A Method for Determining the Finer Dust Particles in Air. Jour. Ind. Hyg., vol. 3, 1921, pp. 51–56. (Quoted by Greenburg, *see* ref. 264.)

342. Drinker, Philip, Thompson, R. M., and Fitchet, S. M. Atmospheric Particulate Matter. I. Dust, with a New Apparatus for Its Determination. Jour. Ind. Hyg., vol. 5, 1923, pp. 19–35, 62–78.
343. Matthiass, —, and Landwehr, —. Neuere Beobachtungen und Maszнahme auf dem Gebiet der Silikosebekämpfung von Bergassessar a D. Dr. Matthiass und Bergingenieur Dr. Landwehr. [Recent Observations and Precautions in the Control of Silicosis, by Mine Assessor Matthiass and Mining Engineer Landwehr.] Ztschr. Berg.- Hütten- und Salinenwesn, January 1935, pp. 421–446.
344. Miquel, P. [Study on the Dusts Formed in the Atmosphere.] Ann. Hyg., vol. 2, 1879, ser. 3, pp. 226, 333. (Quoted by Greenburg, *see* ref. 264.)
345. Tissandier, G. [The Dusts of the Atmosphere.] Rev. Sci., vol. 17, ser. 2, 1880, p. 814. (Quoted by Greenburg, *see* ref. 264.)
346. Irwin, W. The Soot Deposited on Manchester Snow. Jour. Soc. Chem. Ind., vol. 21, 1920, p. 533. (Quoted by Greenburg, *see* ref. 264.)
347. Leifman, K. Habilitationschrift. Degree Thesis. Univ. of Halle, 1907. (Quoted by Greenburg, *see* ref. 264.)
348. Des Voeux, H. A., and Owens, J. S. The Sootfall of London: Its Amount, Quality, and Effects. Lancet, vol. 1, 1912, p. 47. (Quoted by Greenburg, *see* ref. 264.)
349. Mitchell, J. P. Determination of Dust Fall in the Neighborhood of Cement Plants. Jour. Ind. Eng. Chem., vol. 6, 1914, p. 454.
350. Whipple, G. C., and Whipple, M. C. Studies in Air Cleanliness. Trans. Am. Soc. Heat. and Vent. Eng., vol. 21, 1915, pp. 221–224.
351. Green, H. L. Some Accurate Methods of Determining the Number and Size Frequency of Particles in Dusts. Jour. Ind. Hyg., vol. 16, 1934, pp. 29–39.
352. Cottrell, F. G. Problems in Smoke, Fumes, and Dust Control. Smithsonian Rept. for 1913, Pub. 2307, pp. 653–685.
353. Bill, J. P. The Electrostatic Method of Dust Collection as Applied to the Sanitary Analysis of Air. Jour. Ind. Hyg., vol. 1, 1919, pp. 223–342. (Quoted by Greenburg, *see* ref. 264.)
354. Drinker, P., Thompson, R. M., and Fitchet, S. M. Atmospheric Particulate Matter. II. The Use of Electric Precipitation for Quantitative Determinations and Microscopy. Jour. Ind. Hyg., vol. 5, 1923, pp. 162–185. (Quoted by Greenburg, *see* ref. 264.)
355. Drinker, P., and Thompson, R. M. The Determination of Suspensions by Alternating-Current Precipitation. Jour. Ind. Hyg., vol. 7, 1925, pp. 261–272.
356. Stokes, R. S. G. Ventilation, Air Conditioning, and Dust Elimination. South African Min. and Eng. Jour., vol. 46, 1936, pp. 632–635.
357. Ingels, M. New Data on Air Dust Determinations. Jour. Am. Soc. Heat. and Vent. Eng., vol. 29, 1923, pp. 177–198.
358. Miner's Phthisis Prevention Committee, Union of South Africa. Final Report. 1919, p. 110. (Quoted by Greenburg, *see* ref. 264.)
359. Bureau of Mines, Bureau of Chemistry, Research Laboratory American Society of Heating and Ventilating Engineers, and Public Health Service. Comparative Tests of Instruments for Determining Atmospheric Dusts. U. S. Pub. Health Bull. 144, 1925, 69 pp.
360. Littlefield, J. B., Brown, C. E., and Schrenk, H. H. Technique for Routine Use of the Konimeter. Bureau of Mines Inf. Circ. 6993, 1938, 5 pp.
361. Green, H. A. A Photomicrographic Method for the Determination of Particle Size of Paint and Rubber Pigments. Jour. Franklin Inst., vol. 192, 1921, p. 637. (Quoted by Fehnel, *see* ref. 323.)
362. Hatch, Theodore, and Thompson, E. W. A Rapid Method of Dust Sampling and Approximate Quantitation for Routine Plant Operation. Jour. Ind. Hyg., vol. 16, 1934, pp. 92–99.
363. Greenburg, L., and Broomfield, J. J. The Impinger Dust-Sampling Apparatus. U. S. Pub. Health Repts., Repr. 1528, vol. 47, 1932, pp. 654–657.
364. Brown, Carlton E., and Schrenk, H. H. A Technique for Use of the Impinger Method. Bureau of Mines Inf. Circ. 7026, 1938, 20 pp.
365. Brown, Carlton E., and Yant, William P. The Microprojector for Determining Particle-Size Distribution and Number Concentration of Atmospheric Dust. Bureau of Mines Rept. of Investigations 3289, 1935, 8 pp.
366. Brown, Carlton E., Baum, Lester A. H., Yant, William P., and Schrenk, Helmuth H. Microprojection Method for Counting Impinger Dust Samples. Bureau of Mines Rept. of Investigations 3373, 1938, 9 pp.

367. Brown, Carlton E. Midget Microprojector for Dust Determination. Bureau of Mines Rept. of Investigations 3780, 1944, 14 pp.
368. Green, H. L., and Watson, H. H. Physical Methods for the Estimation of Dust Hazard in Industry. Med. Res. Council, His Majesty's Stationery Office, Code 45, 1934, pp. 5–99.
369. Barnes, E. C. Dust Determinations. Am. Jour. Pub. Health, vol. 26, 1936, pp. 274–280.
370. Clayton, George D. Improvement of the MSA Electrostatic Precipitator. U. S. Pub. Health Service, Ind. Hyg. Div., Bureau of State Services, Jour. Ind. Hyg. Toxicol., vol. 29, 1947, No. 6, pp. 400–402.
371. Hicks, Victor. X-Ray Studies of Dusts. Ind. Med., vol. 5, 1936, pp. 173–174.
372. Ballard, James W., and Schrenk, H. H. Routine Quantitative Analysis by X-Ray Diffraction: 1. Photometric Procedure 2. Analytical Method. Bureau of Mines Rept. of Investigations 3888, 1946, 10 pp.
373. Ballard, James W., Ohrby, H. I., and Schrenk, H. H. Quantitative Analysis by X-Ray Diffraction: 1. Determination of Quartz. Bureau of Mines Rept. of Investigations 3520, 1940, 10 pp.
374. Ballard, James W., Ohrby, Howard I., and Schrenk, H. H. Some Photometric Instruments Used in X-Ray Diffraction and Spectrographic Methods of Analysis. Bureau of Mines Rept. of Investigations 3638, 1942, 12 pp.
375. Iron and Coal Trades Review. Selective Dust Sampling: Modified Zeiss Konimeter. Vol. 128, 1934, p. 840.
376. Hay, P. S. The Prevention of Silicosis. Review of Practical Methods. Iron and Coal Trades Rev., vol. 127, 1933, p. 713.
377. Franks, W. R., and Tressider, Leone C. Photometric Estimation of Konimeter Dust Samples. Min. Mag., vol. 51, 1934, pp. 265–271.
378. Church, C. S. Limitations to the Use of the Konimeter. 8th Rept., Monmouthshire and South Wales Coal Owners' Assoc. Coal Dust Research Com., 1943, 27 pp. (Quoted from Bull. Hyg., vol. 18, 1943, pp. 485–486.)
379. Potter, N. M. Micro and Semi-Micro Methods for the Investigation of Airborne Mine Dusts. Bull. Hyg., vol. 22, 1947, p. 44.
380. Foster, Wilder D., and Schrenk, H. H. Petrographic Identification of Atmospheric Dust Particles. Bureau of Mines Rept. of Investigations 3368, 1938, 10 pp.
381. Brown, Carlton E., and Fricht, Florence L. Size of Smallest Dust Particles Revealed by Various Microscopic Systems. Bureau of Mines Rept. of Investigations 3821, 1945, 11 pp.
382. Brown, Carlton E., Beatty, R. L., and Kirby, T. B. Dust-Counting Cells. Bureau of Mines Inf. Circ. 7331, 1945, 9 pp.
383. Kettle, E. H. The Detection of Dangerous Dusts. Lancet, vol. 220, 1934, pp. 889–890.
384. Colliery Guardian. The Differentiation of Noxious Dust. Vol. 149, 1934, p. 64.
385. Stüber, Katharina. The Cellular Response of Lymph Nodes to Various Dust Suspensions Introduced into Lymphatics. Jour. Ind. Hyg., vol. 16, 1934, pp. 282–295.
386. Miller, John W., and Sayers, R. R. The Physiological Response of Peritoneal Tissue to Certain Industrial and Pure Mineral Dusts. Pub. Health Repts., Repr. 1787, vol. 51, No. 49, Dec. 4, 1936, pp. 1677–1688.
———. The Response of Peritoneal Tissue to Industrial Dusts. Pub. Health Repts., Repr. 2234, vol. 56, No. 7, Feb. 14, 1941, pp. 264–272.
387. Hatch, Theodore. Dust Control. Present and Future Design Considerations. Pres. before Am. Soc. Mech. Eng., New York, Dec. 3–7, 1934.
388. Stratton, Reuel C. Industrial Dusts and Their Control. Safety Eng., vol. 68, 1934, pp. 38–40.
389. Harrington, D. Occupational-Disease Hazard of Silicosis in Construction Operations and Its Prevention. Pres. before Nat. Safety Council, Construction Sec., Chicago, Oct. 2, 1933, 13 pp.; Mineral Survey, Mexico City, December 1933, January and February 1934.
390. Colliery Guardian. Dust Prevention in the Coal Industry. Vol. 149, 1932, pp. 97–99.
391. Farrell, Andrew J. Silicosis—in Certain of Its Legal Aspects. Ind. Med., vol. 1, 1932, pp. 35–37.
392. Willson, F. Dust in Industry. Mech. Eng., vol. 55, 1933, pp. 80–82.
393. McCann, W. S. Silicosis in Rochester. Ind. Med., vol. 3, 1934, pp. 386–388

394. South African Mining and Engineering Journal. Rand Dust-Samplers' Progress in Study of Sericite and Phthisis. Vol. 45, 1934, p. 91.
395. Scrutator. The Campaign Against Miners' Phthisis—New Light on the Cause of the Disease—the work of Dr. W. R. Jones—Some Valuable Suggestions. South African Min. and Eng. Jour., 1934, pp. 477–478.
396. Colliery Guardian. Problems of Deep Mining. South African Experience. Vol. 149, 1934, pp. 525–526.
397. Bateman, G. C. Silicosis From the Operator's Standpoint. Canadian Min. Jour., vol. 56, 1935, pp. 21–22.
398. South African Mining and Engineering Journal. The Incidence of Silicosis: A Statistical Examination of the Question—Progressive and Noteworthy Reduction—Mining Industry's Initiative in Investigating the Dust Problem. Vol. 49, 1938, p. 717.
399. Miners' Phthisis Medical Bureau, Union of South Africa. Report Upon Work for the Three Years Ended 31st July 1944, p. 19.
400. Patterson, H. S. Certain Dusts Produced by Mining Processes and in Other Ways. Bull. Inst. Min. Met., No. 417, 1939, pp. 1–14.
401. Forbes, J. J. and Emery, A. H. Sources of Dust in Coal Mines. Bureau of Mines Rept. of Investigations 2793, 1927, 17 pp.
402. Bureau of Mines. Allaying Dust in Bituminous Coal Mines with Water. Tech. Paper 593, 1939, 55 pp.
403. Zeitschrift für Berg-, Hütten-, und Salinenwesen. [Control of Drill Dust in the Mining Industry: Report of "Prize Contest" for Drill-Dust Protection.] Vol. 79, 1931, pp. B367–B410.
404. Nelson, A. Silicosis in Coal Mines. VII. Account of an Investigation in Western Wales. Coll. Guard., vol. 152, 1936, pp. 683–685.
405. Graham, Ivon. Dust in Mechanised Mining. Queensland Govt. Min. Jour., June 15, 1939, vol. 40, pp. 188–189.
406. Harrington, D. Ventilation. Jour. Chem., Met. and Min. Soc. South Africa, vol. 35, 1934, pp. 131–134.
407. Médecine du Travail. [Influence of Ventilation on the Dust Content in the Air of Mines.] Vol. 6, 1934, pp. 254–256. Abs. Jour. Ind. Hygiene, vol. 16, 1934, pp. 141–142.
408. Hay, P. S. A Method of Trapping the Dust Produced by Pneumatic Rock Drills. Safety in Mines Res. Board Paper 23 (London), 1926, 18 pp.
409. Gardner, L. U., Middleton, E. L., and Orenstein, A. J. Silicosis (supplement). Internat. Labor Office, ser. F. (Ind. Hyg.), No. 13, 1930, 24 pp.
410. Safety Engineer. Lung Diseases Among Miners—Method for Combatting Silicosis Explained to Engineers. Vol. 60, 1935, p. 95.
411. Moss, K. N. The Physiological Aspects of Deep Mining. Is Dry Mining the Remedy? Symposium: Problems Arising Out of Temperature and Humidity in Deep Mining on the Witwatersrand. Jour. Chem., Met. and Min. Soc. of South Africa, vol. 36, 1936, pp. 183–185.
412. Brown, Carlton E., and Schrenk, H. H. Relation of Dust Dissemination to Water Flow Through Rock Drills. Bureau of Mines Rept. of Investigations 3393, 1938, 6 pp.
413. Owings, C. W. Some Preliminary Data on Methods for Controlling the Dust Hazards in Mechanical Mining. Bureau of Mines Inf. Circ. 7151, 1941, 12 pp.
414. ———. Suggested Methods for Installing Dust-Allaying Equipment in Bituminous-Coal Mines. Bureau of Mines Rept. of Investigations 3843, 1945, 31 pp.
415. ———. Methods of Allaying Dust in Underground Mining Operations. Bureau of Mines Rept. of Investigations 3631, 1942, 38 pp.
416. Meguin, R. J. Practical Control of Drill Dust in Mines. Pres. before Am. Inst. Min. and Met. Eng., New York City, Feb. 18, 1935, 9 pp.
417. Spencer Turbine Co. The Dust Count (Spencer Drill-Vac). Hartford, 1934.
418. Colliery Guardian. A New System of Dust Allaying in Mines. Vol. 146, 1933, p. 302.
419. Flinn, Frederick B., and Miller, Philip S. Some Dust-Count Data Derived with Wet Drilling and Ventilation. Eng. and Min. Jour. Vol. 139, 1938, pp. 38–43.
420. New York State Department of Labor. Standards for the Testing of "Wet" Drilling Equipment and "Wet Methods" of Dust Control. Ind. Bull., vol. 17, 1938, pp. 63–64.

421. Price, F. H. Dust Suppression (Pneumoconiosis) Experiments at a Kent Colliery. Coll. Guard., vol. 172, 1946, pp. 517–521.
422. Mining Journal. Pneumoconiosis in South Wales Coal Mines, Methods of Dust Suppression. Vol. 226, No. 5777, 1946, pp. 367–368.
423. Sauzéat, M. L'Injection d'eau en veine dans le bassin de St. Etienne. [Injection of Water in the Vein in the St. Etienne Basin.] Institut d'Hygiene des Mines Bull. de Documentation Technique 4, 1947, pp. 4–6.
424. George, K. Z. Steam Infusion As a Means of Dust Suppression. Coll. Guard., vol. 178, No. 4605, Apr. 14, 1949, p. 475.
425. Colliery Guardian. Orders and Announcements, Pneumokoniosis in South Wales. Vol. 167, No. 4331, 1943, p. 790.
426. Tebbins, Bernard D., and Tabershaw, Irving R. Dust Control in Hematite Mining. I. The Effect of Wet Methods. Jour. Ind. Hyg. and Toxicol., vol. 28, 1946, pp. 100–103.
427. ————. Dust Control in Hematite Mining. II. The Effect of Ventilation. Jour. Ind. Hyg. and Toxicol., vol. 28, 1946, pp. 104–106.
428. Sioner, M. I., Yurdakos, S. M., and Sarf, B. M. Control of Dust in Mines. South African Min. and Eng. Jour., 1947, pp. 219–221.
429. Katz, S. H., Smith, G. W., and Meiter, E. G. Dust Respirators: Their Construction and Filtering Efficiency. Bureau of Mines Tech. Paper 394, 1926, 52 pp.
430. Bureau of Mines. Procedure for Testing Filter-Type Dust, Fume, and Mist Respirators for Permissibility. Sched. 21, Aug. 20, 1934, 14 pp.
431. Schrenk, H. H. List of Respiratory Protective Devices Approved by the Bureau of Mines. Bureau of Mines Inf. Circ. 7444, 1948, 21 pp. Berger, L. B. Supplemental List of Respiratory Protective Devices Approved by the Bureau of Mines. Bureau of Mines Inf. Circ. 7513, 1949, 3 pp.
432. Schrenk, H. H., and Pearce, S. J. Selection, Use, and Maintenance of Respiratory Protective Devices. Bureau of Mines Inf. Circ. 7230, 1943, 12 pp.
433. Drinker, Philip. Uses and Limitations for Respiratory Protective Equipment. Mech. Eng., vol. 58, 1936, pp. 171–175.
434. Colliery Guardian. Silicosis Conference at Swansea. Vol. 148, 1934, pp. 1149–1152.
435. Cowles, E. P., and Fluxor-De Smidt, R. A. H. Discussion of Paper on Mine-Air Filtration. Jour. Chem., Met. and Min. Soc. South Africa, vol. 35, 1934, pp. 50–67.
436. Smith, Adelaide R. Review of Silicosis. New York State Dept. of Labor, Ind. Bull., vol. 12, 1933, pp. 224–226.
437. Winslow, C.-E. A., Greenburg, L., and Greenburg, D. The Dust Hazard in the Abrasive Industry. U. S. Pub. Health Repts., Repr. 530, vol. 34, 1918, pp. 1171–1187.
438. Jütten, K. W. [Significance of the Abrasive on the Occurrence of Silicosis and Silico-Tuberculosis in an Ax-Grinding Plant.] Zentralb. Gewerbehyg. u. Unfallverh., vol. 10, 1933, pp. 173–176. Abs. Jour. Ind. Hyg., vol. 16, 1934, pp. 33–34.
439. Bloomfield, J. J., and Greenburg, L. Sand and Metallic Abrasive Blasting as an Industrial Health Hazard. Jour. Ind. Hyg., vol. 15, 1933, pp. 184–204.
440. Meiter, E. G. The Dust Problem in the Foundry Industry. Pres. before Am. Foundrymen's Assoc., June 22, 1933. Abs. Ind. Med., vol. 2, 1933, pp. 82–83.
441. Policard, A. Sur Certains mécanismes pathogéniques de la silicose chez les travailleurs de la métallurgie. (On Certain Pathogenic Mechanisms of Silicosis in Metallurgical Workers.) Le Médecine d'usine, vol. 8, 1946, p. 65. Quoted in Archives des Malad. Prof. Med. Trav. et Secur. Social., vol. 7, 1946, p. 400.
442. Hatch, T., Drinker, P., and Choate, Sarah P. Control of the Silicosis Hazard in the Hard-Rock Industries. I. A Laboratory Study of the Design of Dust-Control Systems for Use with Pneumatic Granite-Cutting Tools. Jour. Ind. Hyg., vol. 12, 1930, pp. 75–91.
443. Beyer, D. S. The Mechanical Control of Occupational Diseases. Nat. Safety News, vol. 28, 1933, pp. 21–22, 54–55.
444. Russell, Albert E. The Health of Workers in Dusty Trades. VII. Restudy of a Group of Granite Workers. U. S. Pub. Health Service Bull. 269, 1941, 71 pp.

445. U. S. Department of Labor, Division of Labor Standards. National Silicosis Conference Report on Engineering Control. Bull. 21, Part 2, 1938, 61 pp.
446. Whipple, G. C. Human Health and the American Engineer. Jour. Ind. Hyg., vol. 1, 1919, pp. 75–84.
447. Drinker, Philip. Protecting the Worker Against Dust Inhalation. Nat. Safety News, vol. 27, 1933, pp. 25–26.
448. Iron and Coal Trades Review. Dust Collector for Grinding Machines. Vol. 128, 1934, p. 453.
449. Dallavalle, J. M., and Hatch, T. Studies in the Design of Local Exhaust Hoods. Trans. Am. Soc. Mech. Eng., vol. 54, 1932, pp. 31–37, Abs. Jour. Ind. Hyg., vol. 15, 1933, pp. 10–11.
450. Hatch, T., Kelley, G. S. and Fehnel, J. W. Control of the Silicosis Hazard in the Hard-Rock Industries. II. An Investigation of the Kelley Dust Trap for Use with Pneumatic Rock Drills of the "Jack-Hammer" Type. Jour. Ind. Hyg., vol. 14, 1932, pp. 69–79.
451. Hatch, T., Fehnel, W. G., Warren, H., and Kelley, G. S. Control of the Silicosis Hazard in the Hard-Rock Industries. IV. Application of the Kelley Trap to Underground Drilling Operations. Jour. Ind. Hyg., vol. 15, 1933, pp. 41–56.
452. Brackett, R. A. Dust Collection in Percussion Drilling. Pres. before Am. Inst. Min. and Met. Eng. Feb. 19–22, 1934. Abs. Rock Products under Symposium on Silicosis Adds Much Helpful Literature, vol. 37, 1934 p. 56–58.
453. Ollett, E. T. Prevention of Silicosis. Crushing, Grinding, and Quarrying Jour., vol. 11, 1934, pp. 355–356.
454. Zangger, H. [Aim, Significance, and Necessity of Medical Factory Inspection.] Schweiz. Med. Wochenschr., 1934, pp. 761–765, 781–787, 801–805. Abs. Bull. Hyg vol. 10, 1935, pp. 14–15.
455. Davis, H. G. (Quoted by J. E. M. Thompson in Review of Recent Advances in Industrial Medicine and Surgery.) Jour. Ind. Hyg., vol. 2, 1920–21, p. 219.
456. Colliery Guardian. Silicosis in British Coal Mines. Vol. 150, 1935, pp. 246–248.
457. Iron and Coal Trades Review. Pneumoconiosis. London, Sept. 8, 1944, vol. 149, No. 3993, pp. 351–352.
458. Lauffer, C. A. Industrial Health Hazards. Jour. Ind. Hyg., vol. 1, 1919, pp. 373–379.
459. Carozzi, L. Considerations et reflexions sur les sources du "De Morbis Artificum." [Consideration and Reflections on the Sources of "De Morbis Artificum".] La Med. Trav., vol. 10, 1938, pp. 275–296.
460. Selby, C. D. Scope of the Physical Examination in Industry. Jour. Ind. Hyg., vol. 1, 1919, pp. 380–384.
461. Burlingame, C. C. The Art, Not the Science of Industrial Medicine. Jour. Ind. Hyg., vol. 2, 1920–21, pp. 368–373.
462. Armstrong, D. B. The Framingham Health Demonstration and Industrial Medicine. Jour. Ind. Hyg., vol. 3, 1921–22, pp. 183–186.
463. Journal of American Insurance. Medical Aspects of Silicosis. Vol. 11, 1934, pp. 21, 24.
464. Cameron, W. J. How Doctors Help Industry. Am. Jour. Pub. Health, vol. 25, 1935, p. 107.
465. Britton, J. A. Industrial Medical Service. Ind. Med., vol. 4, 1935, pp. 53-54.
466. Safety Engineering. Industrial Health and the Physician. Vol. 68, 1934, p. 167.
467. Lehmann, Gunther. The Dust-Filtering Efficiency of the Human Nose and Its Significance in the Causation of Silicosis. Jour. Ind. Hyg. vol. 17, 1935, pp. 37–40.
468. Safety Engineering. Mouth Breathers. Vol. 58, 1934, p. 80.
469. Coal Age. Bronchitis and Sequelae. February 1936, p. 42.
470. Safety Engineering. The Industrial-Disease Problem. Vol. 68, 1934, p. 182.
471. McCord, C. P. Health Hazards in the Foundry. Nat. Safety News, vol. 29, 1934, pp. 34–36.
472. Flinn, F. G. Problems and Improvements of Dust, Fumes, and Gases. Safety Eng., vol. 40, 1935, pp. 89–94.
473. Miners' Phthisis Prevention Committee, Union of South Africa General Report. 1916, 189 pp.
474. Rock Products. Safety Congress Draws Devotees. Vol. 36, 1933, p. 46.

475. McConnell, W. J. Medical Supervision of Workers Exposed to Dust. Nat. Safety News, vol. 30, 1934, p. 34.
476. U. S. Department of Labor Division of Labor Standards. Proceedings of the 7th National Conference on Labor Legislation. Bull. 45, 1940, p. 57.
477. International Association of Industrial Accident Boards and Commissions. Discussion of Industrial Accidents and Diseases. (1944 Convention of the International Association of Industrial Accident Boards and Commissions, Boston, Mass.) Bull. 1, 1944, pp. 195–197.
478. McNally, W. D., and Bergman, W. L. Silicosis—An Old Disease Revived. Ind., Med., vol. 4, 1935, pp. 61–65.
479. Iron and Coal Trades Review. Silicosis in British Coal Mines. Views of H. M. Medical Inspector of Mines. Vol. 130, 1935, pp. 252–253.
480. Cook, C. E. Evaluation of Chest Pathology. Ind. Med., vol. 4, 1935, pp. 55–56.
481. Gardner, L. U. Silicosis and Its Relationship to Tuberculosis. Am. Rev. Tuberculosis, vol. 29, 1934, pp. 1–7.
482. Landis, H. R. M. Tuberculosis and Dust. Ind. Med., vol. 2, 1933, pp. 302–306.
483. Pancoast, H. K., and Pendergrass, E. P. The Roentgenological Aspects of Pneumoconiosis and Its Medicolegal Aspect. Jour. Ind. Hyg., vol. 15, 1933, p. 117. Roentgen Technic with Especial Reference to the Examination to Diagnose or Exclude Silicosis. Jour. Ind. Hyg., vol. 16, 1934, pp. 165–168.
484. Colliery Guardian. Silica-Dust Danger. Vol. 130, 1925, p. 471.
485. Cole, Lewis Gregory. Pneumoconiosis, the Story of Dusty Lungs. Am. Jour. Roentgenol., vol. 51, 1944, pp. 127–128.
486. Gardner, Leroy U. Elements of Diagnosis and Prognosis in Pneumoconiosis, Industry, Tuberculosis, Silicosis, and Compensation. Symposium Pub. by Nat. Tuberculosis Assoc., 1945, pp. 71–84.
487. Bruce, T., and Jönsson, G. [The Roentgen Picture of Silicosis in Different Industries.] Acta Radiologica, Apr. 30, 1943, vol. 24, No. 2 (No. 138), pp. 89–112. Abs. Bull. Hyg., vol. 19, 1944, pp. 620–622.
488. Smith, J. M. A Review of the Work of the Silicosis Medical Board. Conf. on Silicosis, Pneumoconiosis, and Dust Suppression, Paper 7, 1947, 8 pp. Abs. Jour. Ind Hyg. Toxicol., vol. 30, 1948, pp. 27–28.
489. Industrial Medicine. A Meeting on Silicosis. Vol. 2, 1933, p. 84.
490. Lawson, G. B., Jackson, W. P., and Gardner, J. E. Pneumoconiosis in Iron Miners. Jour. Am. Med. Assoc., vol. 16, 1931, pp. 1129–1130.
491. McCord, C. P. The Emergence of Occupational Diseases from Their Dark Ages. Ind. Med., vol. 2, 1933, pp. 349–359.
492. Eck, Marcel, and Hanaut, André. Un projet de terminologie radiologique pour le classement des pneumokoniosis silicotiques. [A Project of Radiologic Terminology for the Classification of Silicotic Pneumoconiosis.] Arch. Malad. Prof., Med. du Trav. Hyg., Toxicol. Secur. Indust., vol. 6, 1944–45, pp. 74–78.
493. Grimault, Jaques. A Propos de la silicose evolutive apres cessation du travail dangereux. [Regarding Evolutive Silicosis After Cessation of Dangerous Work.] Thesis for Dr. of Medicine, Paris, 1945, 62 pp.
494. Middleton, E. L. A Study of Pulmonary Silicosis. Jour. Ind. Hyg., vol. 2, 1920–21, pp. 433–449.
495. Oliver, Thomas. Phthisis and Occupation. Jour. Ind. Hyg., vol. 2, 1920–21, pp. 115–119.
496. Collis, E. L. A Study of the Mortality of Coal Miners, England and Wales. Jour. Ind. Hyg., vol. 4, 1922–23, pp. 256–272.
497. ———. The Statistical Characteristics of Dust Phthisis. Jour. Ind. Hyg., vol. 8, 1926, pp. 456–465.
498. U. S. Public Health Service, Office of Industrial Hygiene and Sanitation. Anthraco-Silicosis (Miners' Asthma): A Preliminary Report of a Study Made in the Anthracite Region of Pennsylvania. Pennsylvania Dept. Labor and Ind., Spec. Bull. 41, 1934, 81 pp.
499. Haldane, J. S. Physiological Problems in Mining. Proc. Empire Min. and Met. Cong. (London), 1923, p. 179 (Quoted by Kettle, *see* ref. 12.)
500. Böhme, A. [Pneumoconiosis and Tuberculosis Among Ruhr Coal Miners.] Beitr. Klin. Tuberk. (Berlin), vol. 61, 1925, p. 364. Abs. Jour. Ind. Hyg., vol. 9, 1927, p. 39.

558. Proceedings Australasian Institute of Mining and Metallurgy, Inc. Aluminum Therapy in Silicosis. New Ser., No. 138, 1945, pp. 19–22.
559. Gye, W. E., and Kettle, E. H. Silicosis and Miners' Phthisis, British Jour. Exp. Path., vol. 3, 1922, p. 241.
560. Gye, W. E., and Purdy, W. J. (*See* ref. 225.)
561. Heffernan, P. The Method of Action of Silica Dust in the Lungs. Jour. Ind. Hyg., vol. 10, 1928, p. 272.
562. Hepplestone, A. G. A Review of Pneumokoniosis and Dust Suppression In Mines. Part I—Medical Aspects of Pneumokoniosis. Bull. Inst. Min. and Met. June 1949, vol. 511, pp. 9–19.
563. Denny, J. J., Robson, W. D., and Irwin, D. A. Prevention of Silicosis by Metallic Aluminum. No. 1. Canadian Med. Assoc. Jour., vol. 37, 1937, pp. 1–11.
564. ————————. Prevention of Silicosis by Metallic Aluminum. No. 2. Canadian Med. Assoc. Jour., vol. 40, 1939, pp. 213–228.
565. Crombie, D. W., Blaisdell, J. L., and McPherson, G. The Treatment of Silicosis by Aluminum Powder. Canadian Med. Assoc. Jour., vol. 50, 1944, pp. 318–328.
566. Gardner, L. U., Dworski, M., and Delahant, A. B. Aluminum in Silicosis. Jour. Ind. Hyg. Toxicol, vol. 26, 1944, pp. 211–223.
567. Hannon, J. W. G. The Utilization of Aluminum. Ind. Medicine, vol. 15, 1946, pp. 527–528.
568. George, W. E. A Report of a Visit to Canada and the United States of America (April-July 1946) to Enquire Into the Use of Different Types of Aluminum Powder in the Prevention and Treatment of Silicosis, together with a General Review of the Subject. Australasian Inst. Min. and Met., Inc., New Ser., No. 143–144, Sept. 30,–Dec. 31, 1946, pp. 1–140.
569. Brull, Lucien. Enquete sur le problème de la silicose. [Inquiry into the Problem of Silicosis.] Inst. Hyg. Mines, Hasselt, Belgium Communication 9, 1947, 17 pp.
570. Shaver, Cecil Gordon, and Riddell, Andrew Rutherford. Lung Changes Associated with the Manufacture of Aluminum Abrasives. Jour. Ind. Hyg. Toxicol., vol. 29, 1947, pp. 145–157.
571. Iron and Coal Trades Review. Aluminum Therapy and Prophylaxis For Silicosis (Use Recommended with Proper Safeguards). Vol. 154, 1947, pp. 1083–1084.
572. Robson, W. D. Medical Facts Revealed by Aluminum Therapy Tests. Eng. and Min. Jour. (summary of address before Canadian Institute of Mining and Metallurgy, Ottawa, January 1947), vol. 148, 1947, pp. 64–66.
573. Journal of the American Medical Association. Aluminum in the Prevention and Treatment of Silicosis, Apr. 27, 1946, vol. 130, no. 17, p. 1223.
574. Riddell, A. R. Present Status of Aluminum Therapy in Silicosis. U. S. Dept. of Labor Standards, Bull. 94, 1948, pp. 25–28.
575. Rauschenbach, C. W., Johns, D. R., Larrabee, J. F., Hamman, L. M., and Porucky, R. F. Silicosis Study and Management in the Calumet Industrial Area. Ind. Medicine, vol. 17, 1948, pp. 1–6.
576. Bloomfield, J. J., and Johnson, W. Scott. Potential Problems of Industrial Hygiene in a Typical Industrial Area. Am. Jour. Pub. Health, vol. 25, 1935, pp. 415–424.
577. U. S. Department of Labor, Division of Labor Standards. National Silicosis Conference Report on Medical Control. Final Rept. of Committee on Prevention of Silicosis Through Medical Control. Bull. 21, pt. 1, 1938, pp. 68–69.
578. Dick, P. G. Role of X-Ray in Industrial Hygiene. Ind. Med., vol. 4, 1935, pp. 156–159.
579. Kreuscher, P. H. Discussion of paper by P. G. Dick, ref. 578, p. 160.
580. Sappington, C. O. Discussion of paper by P. G. Dick, ref. 578, p. 161.
581. Given, William B., Jr. A Better Place to Work. 12th Ann. Meeting, Ind. Hyg. Foundation of America. Ind. Hyg. Foundation, Inc., 1947, pp. 1–24.
582. Tillson, B. F. Silicosis Legislation. Eng. and Min. Jour., vol. 135, 1934, pp. 68–70.
583. Leland, R. G. The Insurance Principle in the Practice of Medicine. U. S. Dept. of Labor, Bur. of Labor Statistics Bull. 602, 1934, p. 58.
584 Acee, Alfred. State Workmen's Compensation Legislation in 1947. Monthly Labor Review, vol. 65, 1947, pp. 415–418.

585. AINSWORTH, CYRIL. Experts Urge Engineering Methods to Prevent Factory-Disease Hazards. Ind. Standard. and Commer. Standards Monthly, vol. 6, 1935, pp. 31–33.
586. COMMITTEE OF RESEARCH AND STANDARDS, AMERICAN PUBLIC HEALTH ASSOCIATION. Report of Committee on Standard Practices in the Problem of Compensation of Occupational Diseases. Am. Pub. Health Assoc., Ind. Hyg. Sec., 1931, 124 pp.
587. KELLOGG, F. S. Silicosis Claims—A New Problem. Safety Eng., vol. 49, 1935, pp. 47–48, 51.
588. WILCOX, F. M. The "Schedule" Fraud in Occupational-Disease Compensation. Am. Labor Leg. Rev., vol. 24, 1934, pp. 119–123.
589. NATIONAL RESOURCES BOARD. Report of the Planning Committee for Mineral Policy, 1935, p. 435.
590. SAYER, HENRY D. Occupational Disease—Real and Supposed. Ind. Med., vol. 4, 1935, pp. 151–153.
591. CRANCH, A. G. Occupational Diseases and Compensation. Safety Eng., vol. 48, 1934, p. 206.
592. KRISER, H. D. Silicosis Compensation: Its Import for the Industry. Eng. and Min. Jour., vol. 135, 1934, pp. 34–36.
593. ROCK PRODUCTS. Illinois Supreme-Court Decision Has Important Bearing on Silicosis. Vol. 37, 1935, p. 53.
594. MONTHLY LABOR REVIEW. Occupational-Disease Legislation in the United States. Vol. 38, 1934, pp. 1348–1363.
595. INDUSTRIAL MEDICINE. West Virginia Silicosis Act. Vol. 4, 1934, pp. 280–283.
596. ———. North Carolina's New Law. Vol. 4, 1935, pp. 149–151.
597. SAYER, H. D. Realities of Workmen's Compensation. Ind. Med., vol. 4, 1935, pp. 331–336.
598. INDUSTRIAL BULLETIN. Silica Hazards in Foundry Dust. New York State Dept. of Labor, vol. 14, 1935, p. 96.
599. INDUSTRIAL MEDICINE. New York Silicosis Bill Vetoed. Vol. 4, 1935, pp. 342–343.
600. ———. Report of Pennsylvania Commission on Compensation for Occupational Diseases. Vol. 4, 1935, pp. 82–87.
601. AIR HYGIENE FOUNDATION OF AMERICA, INC. Fourth Fall Meeting of Members, Pittsburgh, Pa., Nov. 14–15, 1939, pp. 5–9.
602. U. S. DEPARTMENT OF LABOR, DIVISION OF LABOR STANDARDS. Discussion of *Industrial Accidents and Diseases, Convention of the International Assoc.* of Industrial Accident Boards and Commissions, Toronto, Ontario, Canada, Bull. 94, 1947, pp. 76–79.
603. WEST VIRGINIA COAL ASSOCIATION. Silicosis Cases Dismissed. Bull. 609, 1935, pp. 2–3.
604. MINING WORLD. Idaho Mine Legislature Action Is All Negative in Character. Vol. 3, 1941, p. 20.
605. U. S. DEPARTMENT OF LABOR, DIVISION OF LABOR STANDARDS. Principal Features of Workmen's Compensation Laws as of July 1944. Bull. 62 Rev., 1944, 21 pp.
606. INDUSTRIAL HYGIENE FOUNDATION. Transactions, Legal Section, 10th Ann. Meeting. Trans. Ser. Bull. 7, pt. IV of Proc., Nov. 14–15, 1945, p. 4.
607. COFFINBERRY, GEORGE L. The New Occupational Disease Merit Rating Plan Explained. *Ohio Ind. Commission Monitor*, vol. 19, 1946, pp. 115–116.
608. LABOR STANDARDS. Montana Law Provides Welfare Instead of Workmen's Compensation Benefits for Disabled Silicotics, Under Safety, Health, and Workmen's Compensation. Vol. 4, 1941, p. 4.
609. WATERS, THEODORE C. Analysis of Occupational Disease Compensation Statutes and Developments in 1947 Sessions of State Legislatures Prepared for Foundation Members. Indus. Hyg. Found., 1947, p. 10.
610. GIEVER, PAUL M. Silicosis Costs Rise in Montana. Ind. Hyg. Newsletter, vol. 7, 1947, p. 3.
611. STATE OF NEW JERSEY. Workmen's Compensation Law—Revised Statutes, 1945, 78 pp.
612. DAVIS, C. G., SALMONSEN, ELLA M., AND EARLYWINE, J. L. The Pneumoconioses (Silicosis) Bibliography and Laws. Chicago, 1934, pp. 349–475.
613. CUNNINGHAM, J. G. Silicosis in Canada. Records of Internat. Conf. held at Johannesburg, Aug. 13–27, 1930. Internat. Labor Office, Geneva, 1930, pp. 316–337.

614. Bateman, G. C. Silicosis from the Operator's Standpoint. Canadian Min. Jour., vol. 56, pp. 21–22.
615. Workmen's Compensation Board. The Workmen's Compensation Act of Ontario with Amendments to 1934 and Regulations of Board Synopsis. Toronto, Canada, 1935, pp. 50–53.
616. Safety Engineering. Some Recent Changes in Workmen's Compensation Laws. Vol. 47, 1934, p. 272.
617. American Public Health Association. Occupational Disease Legislation. Rept. of Committee on Standard Practices in the Problem of Occupational Diseases of the Ind. Hyg. Sec., Am. Pub. Health. Assoc., 1931, 124 pp.
618. Sahourin, Ivin. Canada and Compensation. Trans., Legal Sec., 10th Ann. Meeting, Trans. Series, Bull. 7, Ind. Hyg. Foundation, pt. IV, 1934, pp. 8–13.
619. Collis, E. L. Legislation in Various Countries Regarding Silicosis with Special Reference to Compensation. Rept. of 4th Meeting, Permanent Internat. Commission for Study of Occupational Diseases. Lyon, Apr. 3–6, 1929, pp. 133–169.
620. Crushing–Grinding–Mining–Quarrying. This Silicosis Question. Vol. 3, 1935, pp. 119–120.
621. Home Office. Workmen's Compensation Act, 1925. The Sandstone-Industry (Silicosis) Scheme, 1929. Worker's Register (London), 1929, 24 pp.
622. Colliery Guardian. Parliamentary Intelligence. Silicosis Amongst Coal Miners. Vol. 148, 1934, p. 149; vol. 149, 1934, p. 205.
623. ———. Law Intelligence. Workmen's Compensation: Silicosis Claim Dismissed. Vol. 149, 1934, p. 322.
624 Iron and Coal Trades Review. Legal Intelligence. Silicosis Appeals in the House of Lords. Vol. 131, 1935, p. 58.
625 Colliery Guardian. Law Intelligence. Workmen's Compensation: Silicosis. Vol. 151, 1935, pp. 203–204.
626. ———. Orders and Announcements. Silicosis (Medical Arrangements). Vol. 149, 1934, p. 348.
627. Davis, G. G., Salmonsen, Ella M., and Earlywine, J. L. The Pneumoconioses (Silicosis). Literature and Laws of 1934, 1935, p. 419.
628. International Labor Office. Compensation for Silicosis in Great Britain Beginning March 1, 1935. Ind. and Labor Inf., vol. 53, 1935, p. 202. Abs. Jour. Ind. Hyg., vol. 17, 1935, p. 114.
629 Southerland, C. L. Workmen's Compensation in Pulmonary Disease. Jour. State Med., vol. 40, 1932, pp. 709–716.
630 Lancet. South African Mine Dust. Jan. 31, 1925, p. 243.
631. Workmen's Compensation Commission of New South Wales. Third Annual Report and Statistics for the Year July 1, 1928, to June 30, 1929. 78 pp.
632. Mining and Metallurgy. Miners' Health. Vol. 16, 1935, pp. 290–291.
633. George, W. E. Medical Examination and Compensation of Miners for Industrial Injury. Occupational Medicine, vol. 4, 1947, pp. 68–85.
634. Magnin, Jean. Sur la prophylaxie de la silicose dans les mines. [Prophylaxis of Silicosis in Mines.] Arch. Malad. Prof. Med. Travail et Sec. Soc., vol. 7, 1946, 475–488.
635. Sherman, P. Tecumseh. History of Workmen's Compensation for Occupational Disease. Bull. Assoc. Casualty and Surety Exec., July 1935. (Quoted by Ind. Med., vol. 4, 1935, pp. 453–457.)
636. Swiss Federal Council. Prevention de la silicose dans le construction de tunnels, de galeries et dans les mines. I. Arrete du conseil federal du 4 decembre 1944. II. Ordonnance 1 du department federal de l'economie publique of 23 decembre 1944. [Prevention of Silicosis in the Construction of Tunnels, Galleries, and in Mines. I. Decree of the Federal Council of Dec. 4, 1944. II. Order of the Federal Department of Public Economy of Dec. 23, 1944.]
637. Lang, Fritz. Silicose et assurance. [Silicosis and Assurance.] Jour. Medical de Leysin, no. 2, March–April 1945, 10 pp.
638 Pennsylvania Commission on Compensation for Occupational Disease. Occupational Disease Compensation. Commonwealth of Pennsylvania, 1933, pp. 58–59.
639. Safety Engineering. The Cost of Industrial Disease. Vol. 69, 1935, pp. 281–282.

640. Wrabetz, V. Workmen's Compensation Legislation in Relation to Occupational Diseases. U. S. Dept. of Labor, Bull. 4, Div. of Labor Standards, 1936, p. 40.
641. Industrial Commission of Wisconsin. Diseases of Occupation and Other Nonaccidental Injury Cases Settled in 1945. Stat. Release 3214 (Mim.), Dec. 19, 1946, 24 pp.
642. Bartlett, T. N. (*See* ref. 640, p. 325).
643. Jones, F. R. Occupational-Disease Compensation. Ind. Med., vol. 5, 1936, pp. 179–183.
644. Rock Products. California Compensation Rates Increased to Cover Silicosis. Vol. 39, 1936, p. 70.
645. Engineering and Mining Journal. Silicosis Surcharge in California. Vol. 137, 1936, p. 442.
646. Graff, W. M. The Responsibility of the Health Department for Occupational-Disease Control. Safety Eng., vol. 68, 1934, pp. 123–126.
647. West Virginia Coal Association. Silicosis-Compensation Fund. Bull. 612 1935, pp. 1–2.
648. South African Mining and Engineering Journal. Employees' Provident Fund. Vol. 46, 1935, p. 787.
649. ———. Miners' Phthisis Board. Vol. 46, 1935, p. 787.
650. ———. Phthisis Liability. Vol. 58, pt. I, 1947, p. 301.
651. Colliery Guardian. Workmen's Compensation in 1933—Industrial Diseases. Vol. 150, 1935, p. 212.
652. Rock Products. Various Rock-Products Subjects Discussed by Mining Engineers. Symposium on Silicosis Adds Much Helpful Literature. Vol. 37, 1934, pp. 56–58.
653. American Journal of Public Health. Compensation for Silicosis. Abs. vol. 23, 1933, p. 1084.
654. Wright, George W. Industrial Pulmonary Diseases and Capacity for Work. Occupational Medicine, vol. 2, 1946, pp. 546–550.
655. George, W. E. Proceedings of the International Conference Held in Geneva, Aug. 29 to Sept. 9, 1938. Item 7, Determination of Disability and Assessment of the Degree of Such Disability in Pneumoconiosis, 1940, p. 65.
656. Coal and Colliery News. New Factories for Disabled Miners. 1945, p. 573.
657. Colliery Guardian. Employment of Silicotic and Pneumoconiotic Persons. Vol. 172, 1946, p. 69.

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