The American Ceramic Society

February 15, 1993

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Christine Schnitzer

Christine Schnitzer
Product Manager
Ceramic Information Center

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735 Ceramic Place
Westerville, Ohio 43081-8720
614 • 890 • 4700
TWX: 7101109409

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CERAMIC ABSTRACTS

Compiled by

The American Ceramic Society

Ross C. Purdy, *Editor*

Mary J. Radcliffe
Emily C. Van Schoick
Dorothy J. Turner
} *Assistants*

Committee on Publications: J. D. Sullivan, *Chairman*, E. de F. Curtis, R. F. Sherwood, W. W. Wenrich, R. C. Purdy

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Volume 16, 1937

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Editorial Office: 2525 North High Street, Columbus, Ohio.
Publication Office: 20th and Northampton Streets, Easton, Pa.

have been plotted in a ternary diagram. These results show that the pigments of the best hiding powers were prepared from solutions with a titanium dioxide concentration falling between 13 and 19% and ratios ranging from 1/1 to 1/0.65. Seeding opalescence occurs at different hydrogen-ion concentrations with different concentrations of solutions. Illustrated. F.G.H.

Viscosity and plasticity of disperse systems: VI, Effect of temperature and electrolytes on the plastic properties of kaolin. M. P. Wolarowitsch and D. M. Tolstoi. *Kolloid-Z.*, 73 [1] 92–96 (1935); see *Ceram. Abs.*, 16 [3] 101 (1937). VII, Plastic flow in an apparatus with cylinders displaceable lengthwise. D. M. Tolstoi. *Kolloid-Z.*, 73 [1] 96–101 (1935).—A method for determining the flow resistance and shearing modulus of high concentration mineral suspensions is described. The apparatus was designed particularly to eliminate surface gliding of the cylinders. Results agreed well with those obtained by the rotating cylinder method of Wolarowitsch. At very small velocities, Bingham's equation for plastic flow does not hold for high concentration mineral suspensions. For Parts I–V see *Ceram. Abs.*, 15 [7] 222 (1936). F.P.P.

PATENTS

Alkali silicate. Anders E. A. Cornelius (Helios Kemisk-Tekniska Fabriker A.-B.). Swed. 80,000, April 4, 1934. *Chem. Abs.*, 29, 6000 (1935).—Steam is blown into an electrically heated bath of alkali silicate formed by the reaction of alkali chloride and SiO_2. During the process the bath is kept covered by a layer of the unmelted charge of alkali chloride and SiO_2, with or without another substance, *e.g.*, lime, for the formation of double silicates.

Method of manufacture of precipitated zinc oxide. G. Antonoff. Brit. 459,238, Jan. 20, 1937 (June 5, 1935).

Production of a calcium aluminate suitable for the manufacture of sodium aluminate by treatment with soda lye. Aktieselskapet Norsk Aluminium Co. Brit. 457,676, Dec. 16, 1936 (Aug. 30, 1934).

General

Animal silicosis. H. Siegmund. *Verhandl. Deut. Path. Ges.*, pp. 262–71 (1934); *Chem. Abs.*, 29, 8169 (1935).

Causation of pneumonoconioses. Philip Drinker. *Jour. Ind. Hyg. & Toxicol.*, 18 [8] 324–36 (1936).—The various pneumonoconioses and their causative agents are discussed. Silicosis and asbestosis are definite diseases; in anthraco-silicosis and various silicatoses, modifications of normal lung conditions are seen. Effects of carbon or carbonates, gypsum, and iron oxides are also discussed. Methods of estimating dust exposures of a workman include the taking of dust samples from the work place and interpreting the results during the man's total period of exposure. Errors made from estimating the composition of dust from samples which have settled upon rafters instead of using samples taken from the air are emphasized. Permissible dust concentrations need not exceed 50 million particles per cu. ft. for dust with very low quartz content or 5 million for pure quartz. In defense of the use of standards of dustiness, the experience in S. Africa where dust standards have been applied successfully for many years is cited. E.J.V.

Charles N. Brady. Axon. *Bull. Amer. Ceram. Soc.*, 16 [2] 73–74 (1937).

Chemical considerations on some pneumoconioses (anthracosis, silicosis): I, Nature of the problem. René Fabre. *Congr. Chim. Ind., 14th Congress, Paris*, 5 pp. (1934); *Chem. Abs.*, 29, 5774 (1935).

Clinical aspects, diagnosis, and treatment of pneumoconiosis. W. Irving Clark. *Jour. Ind. Hyg. & Toxicol.*, 18 [8] 537–49 (1936).—Pneumonoconiosis is a disease resulting from the inhalation of inorganic dust. The two pneumonoconioses which produce disability are silicosis and asbestosis. The pathology of silicosis is characterized by the presence of fibrotic nodules scattered through both lungs, and that of asbestosis is characterized by an interstitial fibrosis involving the lower half of both lungs. The symptoms of silicosis and asbestosis are similar, the most important being dyspnea. The most common complication is pulmonary tuberculosis which, in silicosis, is a frequent cause of death. The diagnosis of both silicosis and asbestosis is made largely by a correlation of history and symptoms with the X-ray examination. The treatment of pneumonoconiosis is preventive and symptomatic. The course of silicosis and of asbestosis is slow but eventually leads to incapacity due either to the disease itself or to a complication, frequently chronic pulmonary tuberculosis. The number of workers exposed to harmful mineral dusts in mining and in industry in the U.S. has been estimated as 500,000. E.J.V.

Clinical hematological studies for the differential diagnosis of industrial silicosis. C. Schlomka and F. A. Nolte. *Klin. Wochschr.*, 14, 987–92 (1935).—The authors have experimented with the pathological-anatomical actions of the changes of the silicotic lungs in connection with the heart and the complete bloodstream. With the assistance of electrocardiograms, they have been able to investigate the condition of the lungs of silicotic patients. At the policlinic of Bonn University, Röntgen observations of the last 190 silicosis patients were taken, and these were analyzed according to pure radiological points of view. Various tables showing the bloodcounts of several silicotic and tubercular patients were compiled. After certain peculiarities were established in the blood stream of the silicosis patients, it was found necessary to make further investigations of the connections between the studies of the blood and the clinical Röntgenological status. To obtain a most secure foundation, it was decided to separate those silicosis patients from the others who showed an important degree of tubercular complications. A characteristic blood status was shown in stone dust patients. This shows clearly excessive increase of white blood corpuscles with increased sinking at approximate normal counts of red corpuscles and distinct small counts of lymphocytes at relative transitional forms of lemkocytes in a streak preparation. The same symptoms, even if less impressive, are shown in a second group from the total of silicosis patients as positive cases with tuberculosis complications.

W.F.F.

Colored ceramic aggregate for decorative concrete.

C. W. PLANJE. *Jour. Amer. Ceram. Soc.*, 20 [3] 90–96 (1937).

Diagnosis of silicosis. EMERY R. HAYHURST. *Amer. Labor Leg. Rev.*, 26, 61–68 (1936); abstracted in *Jour. Ind. Hyg. & Toxicol.*, 18 [3] 131–32 (1936).—Factors necessary to diagnose chronic silicosis are: (1) exposure to silica dust *in sufficient quantity for 3 to 20 years*; (2) determination by intraperitoneal injection of the nodule-forming character of the dust; (3) nature of the work; (4) personal factors such as mouth breathing, previous respiratory diseases, etc.; (5) X-ray findings. Serial pictures should be taken over a period of time. The location of the nodules is characteristic, avoiding the periphery, symmetrical distribution, shape, and size. In this phase chronic silicosis is without disability. (6) Physical examination; no characteristic but some suggestive findings. (7) The patient's complaints: these are negligible in the first and second stages, but later cough, dyspnea, pains in chest, etc., may occur. E.J.V.

Economizers and air preheaters. V. WALKER. *Elec. Rev.*, 119 [3082] 353 (1936).—W. discusses the conditions which govern the design of economizers and air preheaters for use with boilers. J.L.G.

Epidemiological aspects of silicosis and tuberculosis. ALTON S. POPE AND DAVID ZACKS. *Amer. Rev. Tuberculosis*, 32, 229–42 (1935); *Chem. Abs.*, 29, 7534 (1935).—In representative groups of both granite and foundry workers in Massachusetts, the frequency of silicosis and of silicosis and tuberculosis was correlated with the duration of exposure to dusts containing free silica and to the concentration of such dusts in the occupational environment. Among granite workers silicosis was found in 15.2% and with tuberculosis in 7.6%. Tuberculosis is the cause of death in over one-third of all granite workers, a proportionate mortality 3 times that in foundry workers and 4 times that in all males of 20 years and over. Among the employees of 10 Mass. foundries, silicosis was found in 8.8% and with tuberculosis in 2.8%, a total frequency of one-half that in granite workers. The silicosis in foundrymen was not only less frequent but also less advanced in degree than in the granite workers. Pneumonia, causing one-fourth of all the deaths of foundrymen, is the greatest occupational hazard in the industry.

Industrial health hazards and employer responsibility. W. J. MCCONNELL. *Trans. Amer. Foundrymen's Assn.*, 7 [1] 161–65 (1936).—The importance of greater attention to the problem of overcoming industrial health hazards for foundry employees is recognized. Medical aspects are discussed, foundry hazards are outlined, and suggestions for a control program are given. D. E. CUMMINGS. *Ibid.*, pp. 166–79.—With the invention of the machine, dust became an industrial hazard. C. discusses the action of dust in the lungs and points out that all dusts are not dangerous. Silica dust is one of the dangerous dusts but only when present in certain concentrations of certain particle sizes. The silicosis problem is complicated by the fact that in many cases there is also decided evidence of the presence of tuberculosis. In foundries many men with normal lungs work for years and do not contract silicosis although there may be evidence of fibrosis. C. explains the action of dust particles inhaled on previous pulmonary injuries and states that workmen who are disabled by occupational fibrosis contracted in the foundry generally are those who exhibit evidence of concurrent pulmonary infection or injury. It is the executive's responsibility to inform himself on dust hazards, conduct surveys of the plant, and inaugurate a program of control. Prospective employees are grouped into three general groups; those which are fair risks and those which should not be employed are indicated. C. recommends that an educational program similar to that used in "Safety First" campaigns be inaugurated to combat the dust hazard. Discussion, AVERY *et al. Ibid.*, pp. 180–90.—The various ways of preventing illness are pointed out, and precautionary measures taken in the foundry are outlined. H.E.S.

Industrial pulmonary disease due to inhalation of dust, with special reference to silicosis. E. L. MIDDLETON. Milroy Lectures, 1936. *Lancet*, pp. 1–9 (July 4); pp. 63–64 (July 11, 1936); abstracted in *Jour. Ind. Hyg. & Toxicol.*, 18 [3] 129–30 (1936).—The first lecture is devoted to silicosis, as being the most important of the pneumonoconioses. Particular attention is directed to the occurrence of the disease according to occupation. During 1930 to 1934, at least 1521 deaths occurred in England and Wales, of these, 372 were caused in the sandstone industry, and 326 were ascribed to coal mining; the manufacture of pottery claimed 270, metal grinding 142, gold miners, returned from S. Africa, 104, and tin miners 91. The disease is always originated by the dust of fine particles of free silica; the way in which this risk is present in a number of industries is explained. Sandblasting and the manufacture of abrasive soaps receive special note, as in both the hazard is so intensive that the onset of the disease is unusually quick and its course rapid; thus only 3 or 4 years of employment may have preceded the onset, instead of 15 to 20 years as in most other occupations. The occurrence of cases in coal mining is unexpected, since coal itself is not a dangerous dust; apparently the disease is originated by dust arising from the rocks among which silica is found or from impurities in the coal. Out of 169 deaths in all coal fields in Great Britain, 147 came from S. Wales; this undue proportion has not yet been explained. The picture, both clinical and histological, becomes modified when silica is mixed with other forms of dust; when coal is associated in the dust, the condition is known as anthracosis; here the fibrous tissue entangles the coal dust and retains it to help in blocking up the lymphatics. In the second lecture the influence of the dust of silicates, *i.e.* of chemically combined silica, is considered. Of these asbestosis stands out as most toxic; the particles consist of fragments of fibers, some 3 microns long and 1/2 micron in diameter. They originate pulmonary fibrosis which is more generalized and less nodular than that of silicosis; the condition develops quicker than silicosis, but the tendency to succumb to superimposed tuberculosis is not so pronounced. Other dusts considered are those of sillimanite, china clay, talc, mica, and sericite; none of these approach asbestos and silica in their dangerous qualities. The harmful nature of any dust may be tested by noting the reactions which follow when it has been either injected subcutaneously or administered by the tracheal route suspended in fluid; injection into the peritoneal cavity may also be employed. The theory adopted is that dusts exert their harmful influence by reacting chemically with

the tissues of the lungs, the life and structure of which are thereby modified. Each form of dust stimulates a special reaction of its own, while mixed dusts set up a mixed reaction. No evidence is put forward that dusts other than those of silica or silicates are harmful, or that any dust is capable of reducing the toxic influence of any other.

E.J.V.

Investigations on quartz and sericite analysis of industrial dusts. F. SARTORIUS AND K. W. JÖTTEN. *Arch. Hyg. & Bakteriol.*, 115, 135–51 (1935); abstracted in *Jour. Ind. Hyg. & Toxicol.*, 18 [4] 54 (1936).—The separation of various mineral constituents of dusts by the use of liquids having suitable specific gravities is discussed. The standard methods of mineralogical and chemical analysis of dusts are mentioned, with a description of the X-ray examination. Some of the common minerals are shown. Mention is made of the mineralogy of sericite. E.J.V.

Methods of dust control. J. D. LEITCH. *Trans. Amer. Foundrymen's Assn.*, 7 [1] 209–21 (1936).—The point to begin elimination of the dust hazard is at the source. Methods used for controlling the dust hazards originating at different types of sources are described. L. discusses the general methods used in dust-suppression problems and the estimation of the hazard, contending that the efficiency of a dust-collection system is best determined by dust counts. Methods used for testing ventilating equipment and the selection of fans are presented. Dust collectors and respirators, such as helmets, filter-type respirators, and positive-pressure masks, are discussed. Maintenance of equipment is necessary to the successful operation of such equipment and to the efficiency of any installation. A discussion is included.

H.E.S.

Molding of clay after heating at various temperatures. R. MORETTI. *Corriere Ceram.*, 15, 221–23 (1934); *Chem. Zentr.*, 1934, II, 3296; *Brit. Chem. Abs.-B*, 54 [27] 591 (1935).—Heating at 100 to 300° before molding increases the apparent porosity of the product fired at 900° and decreases the resistance to breaking of a specimen dried at 120°.

Nomograph for ceramic calculations. A. A. GUSHYAKH. *Khimprom*, 3 [5] 374–78 (1935).—The nomograph facilitates the chemical analyses and the calculation of the amount of oxides in the fired product and of the raw material required for a given quantity of the latter.

P.B. & E.S.

Personnel management. J. E. WALTERS. *Factory Management & Maintenance*, 94 [12] S333–44 (1936).—The topics considered are (1) personnel management costs, (2) selection of employees, (3) personnel maintenance, (4) training and education, (5) medical and health service, (6) safety and accident prevention, and (7) personnel service work. Figures, tables, and photographs are included.

J.L.G.

Significance of free crystalline silica in the etiology of silicosis and silicotuberculosis. K. W. JÖTTEN AND H. [illegible]. *Med. Welt.*, 10, 545–50 (1936); abstracted in *Jour. Ind. Hyg. & Toxicol.*, 18 [8] 130–31 (1936).—The authors dusted rabbits over a period of four years, first 2, then 3, then 6 hr. daily, with porcelain dust having 30% free silica and 1 to 2% sericite. After this period the animals showed no typical silicosis although there could be seen sharp, pinpoint-sized, subpleural foci and a slight increase of fibrosis. During the dusting, tubercle bacilli were administered intravenously. These grew slowly, and with indurating processes, into a pulmonary tuberculosis while the controls showed no noteworthy disposition to contract tuberculosis. It is concluded that over a 3 to 4 year period of dusting with tubercle bacilli injected, it was possible to produce a tuberculosilicosis, although with the dust alone it was not possible to show silicosis, in spite of the presence of sericite. By dusting with pure quartz (without tuberculosis injection and without sericite) for 1 hr. daily for 2 years, typical silicotic nodules were produced.

E.J.V.

Silicate analysis in industrial dusts. F. SARTORIUS AND K. W. JÖTTEN. *Zentr. Gewerbehyg. & Unfallverhüt.*, 21, 65–69 (1934).—Materials studied were from the Rhine Steel Works, the Thyssen, and the U. S. Steel Co. Tables show in detail the procedure and results, accompanied by ample comment and description of methods involved. If dusty occupations are to be graded on a danger scale over long or short periods, the sickness percentages and dust analyses must be carefully compared. These new research methods are concerned with the size of grains in dust raised and the accurate preparation of undissolved acid particles, thus serving the combined purposes of mechanical phase analysis and chemical analysis. Important insight into the structure of different kinds of dust has been gained, and this has been increased by using definite purity tests, *e.g.*, the tetralin method.

K.R.

Silicosis: harmful dusts which cause it. W. R. JONES. *Trans. Ceram. Soc.*, 34, 303–43 (1935); see *Ceram. Abs.*, 14 [3] 84 (1935).

Silicosis in the porcelain industry from the standpoint of the physician. SCHILFFARTH. *Ber. Deut. Keram. Ges.*, 17 [6] 322–30 (1936).—It is pointed out that a silicotic lung rarely occurs by itself, being usually associated with tuberculosis. A description of a silicotic lung is given. S. outlines the two theories of silicosis, mechanical and chemical, and discusses the chemical aspect of the problem.

E.J.V.

Training your own instrument men. ALFRED G. MOOR. *Instruments*, 10 [1] 15–18 (1937).—M. reviews basic considerations, qualifications of candidates, training of instrument men, division of time between various types of repair jobs, supplementary educational work, progress reports, and rating guide to be used in semiannual check-up.

R.W.R.

BOOKS AND BULLETINS

Determination and Control of Industrial Dust. J. J. BLOOMFIELD AND J. M. DALLAVALLE. *U. S. Pub. Health Bull.*, No. 217, 167 pp. (April, 1935).—This brief but comprehensive work presents "the methods and instruments used in conducting dust studies in industry, the interpretation of results thus obtained, and their practical application to industrial problems, especially those phases relating to control of the dust hazard." Preliminary procedure involves the sanitary and occupational survey of a plant; final emphasis is placed on personal respiratory protection of workers. Between these occurs the dust technique.

increasingly large verdicts. The Workmen's Compensation Act first applied only to accidental injuries. Occupational diseases should be compensated under an arrangement similar to workmen's compensation but separately, because there is so much difference between them. Partial disability should be compensated. It is difficult to obtain unbiased and expert medical diagnosis of silicosis and the degree of disability. There should be a special medical board in each state for such a diagnosis. Prevention is more important than compensation and much cheaper. W.D.F.

Mineral materials which produce silicosis. B. Gossner. *Sprechsaal*, 69 [41] 601–602; [42] 618–17 (1936).—Lung diseases occurring in the porcelain industry are not produced by sericite or other minerals containing mica, as it is not present in the dust and mica is not contained by the siliceous materials used. This fact shows that lung diseases are not connected with the presence of mica. The appearance of silicosis is chiefly due to the presence of free silica in the siliceous rocks used. M.V.C.

Protection of health of glass and ceramic workers. T. Thomas. *Glashütte*, 66 [49] 823–26 (1936).—Measures to be taken in the case of various injuries sustained by workmen are discussed. M.V.C.

Rating and performance of stationary steam boilers. W. S. Patterson. *Combustion*, 8 [2] 23–30 (1936).—The absurdity of the terms "boiler horse power" and "per cent of rating" as applied to modern steam generating units is discussed, and more rational expressions are suggested. The effect of variable entering gas temperature, operating pressure, and superheat on the boiler performance is analyzed and illustrated by curves and equations. Gas-temperature gradient and draft loss through boilers are also discussed. H.E.S.

Report of the Committee on Ceramic Education for 1936. Harold E. White. *Bull. Amer. Ceram. Soc.*, 16 [3] 111–20 (1937).—This report includes tabular data compiled by E. E. Marbaker and E. H. Fritz.

Suggestions for starting and maintaining a foundry apprenticeship system. J. E. Goss. *Trans. Amer. Foundrymen's Assn.*, 7 [2] 323–50 (1936).—G. outlines points to be taken into consideration in apprentice training, such as methods of selection, testing prospects, age limits, rates of pay, piece-work, requirements of a program, necessity of centralized activities, trial apprenticeship periods, agreements, kind of instruction, related work, etc. A discussion is included. H.E.S.

BOOKS AND BULLETINS

Bureau of Mines activities in the field of building materials. D. M. Banes. *U. S. Bur. Mines Information Circ.*, No. 6924, 31 pp. Free. B. collates all publications by members of the Bureau personnel on the subject of building materials and includes reports published by the Government Printing Office and mimeographed by the Bureau and articles in technical journals and cooperative reports. R. A. Heindl.

Chemist's Year Book. Edited by E. Hope. Sherratt & Hughes, Manchester; Chemical Publishing Co., New York, 1936. 13th ed. Price $6.00. 1257 pp. The handbook has complete information concerning the various chemical constants and characteristics and properties of most organic and inorganic elements and compounds. In addition, various qualitative and quantitative determination procedures are given both for organic and inorganic substances. M.C.Shaw

National Physical Laboratory Collected Researches: Vol. XXV, 1935. H. M. Stationery Office, London. 432 pp. Price £1 5s. The volume contains twenty-three reports of researches devoted chiefly to the study of metallurgical problems. The reports cover studies of the various iron alloys, alloys of Au-Cu, Cd-Zn, Ag-Sn, and Ag-Hg, metallic cadmium, and the crystal structure of α- and β-manganese. One paper is devoted to special refractories for metallurgical research. A brief résumé is given of the properties and methods of producing refractory crucibles, muffles, and tubes from silica, alumina, magnesia, thoria, alumina-clay, and silicon carbide-clay. M.C.Shaw

Official Year-Book of the Scientific and Learned Societies of Great Britain and Ireland; Record of Publications Issued during the Session 1935–1936. Compiled from Official Sources. 33rd annual issue. Charles Griffin and Co., Ltd., London, 1936. vii + 170 pp. Price 10s net. Noted in *Nature*, 139 [3508] 51 (1937). J.L.G.

Third Ordinance Increasing Compensation of Occupational Diseases in Germany. M. Bauer. Reprint from *Reichsarbeitsblatt*, No. 36, Part IV (1936). 47 pp. Berlin, 1937. The new ordinance relating to occupational diseases in Germany went into effect April 1, 1936, superseding the Second Ordinance of Feb. 11, 1929. Several industries are added to the list of compensable diseases, including all the ceramic industries (previously only the porcelain industry was insured), sandblasting, and operations for cleaning castings and molds. Asbestosis in the asbestos trades and lung cancer in the chromium industries become compensable. Severe silicosis, compensable under the Second Ordinance, is still the only form of silicosis insured against, but what constitutes the disease for clinical, röntgenological, and judicial estimation is definitely specified, thus doing away with a hitherto vexatious miscellany of complications. Severe silicosis accompanied by pulmonary tuberculosis requires only the establishment of the severe silicosis in diagnosis. Among damage claims reported for occupational diseases since 1929, silicosis has stood at the peak; in 1934, of 1043 cases, 322 were for silicosis; in 1935, of 1161 cases, 627 were for silicosis, with 146 deaths in 1935 from this cause. K.R.

NEW JOURNAL

Staub (Dust). Edited by C. Lümgert. Vol. 1, No. 1, April, 1936. Published quarterly by Wilhelm Knapp, Halle. Price 5 Rm. The Association of German Industrial Insurance Societies reports on the current status of the dust-control campaign and surveys the literature. Two or three original research papers comprise the first part of each issue, followed by reviews of pertinent literature arranged systematically under (1) dust research into properties, estimation, and measurement, (2) dust sources, (3) dust diseases, (4) other dust injuries, (5) preventive methods, and (6) laws and decisions. K.R.

ADDRESSES AND ORIGINAL ARTICLES

INDUSTRIAL PULMONARY DISEASE DUE TO THE INHALATION OF DUST

WITH SPECIAL REFERENCE TO SILICOSIS*

BY E. L. MIDDLETON, M.D. Edin., D.P.H.
H.M. MEDICAL INSPECTOR OF FACTORIES

THE aim in these lectures is to reach some conclusions regarding the ætiology of silicosis from a study of the occupations in which it occurs. In order to save confusion as to the condition to which the term silicosis is applied, the typical form of the disease is briefly described. The spread of the disease over the industries in this country is referred to and some of the occupations are more closely studied. It will be found that in all these occupations in which silicosis occurred there had been exposure to the inhalation of dust of free silica.

Besides the industries in which silicosis occurs, certain occupations are referred to in which the workers are exposed to dust of silicates without free silica, and the changes following such exposure are described. These form a contrast with the characteristics of silicosis and, in general, they conform to the description of a condition which has been called "silicatosis."

Silicosis

Silicosis is the most important of that group of diseases which have been termed collectively the pneumoconioses. In this country it is always the result of prolonged exposure to certain kinds of dust in the course of occupation. The onset of symptoms is marked by dyspnœa. Slight at first, noticeable only after prolonged or unusual exertion, it remains the most important symptom and is progressive throughout the course of the disease. In the early stage physical signs are characteristic, but they must be sought for with care. In this first stage the radiograph shows the presence of discrete shadows indicative of nodulation. In the second stage dyspnœa and cough become established and greater changes are noted in the physical signs. The radiograph shows the whole of both lung fields occupied by shadows indicative of nodulation, and there is some coalescence to form more or less dense opacities. There is always some degree of impairment of working capacity. The third stage is reached with total incapacity, through increasing severity of symptoms of which dyspnœa is the most distressing. The radiograph indicates areas of massive consolidation. Pulmonary tuberculosis may be present in any stage of silicosis. It may alter the symptoms, physical signs, radiological appearances, and the whole course of the disease. It is the most frequent accompaniment of silicosis.

Post-mortem appearances.—In fatal cases of uncomplicated silicosis the lungs are generally large, of increased density, and retain their shape on removal from the chest. Pleural adhesions are nearly always present and may be extensive and of long standing. On parts of the lung not covered by adhesions the pleura is typically mottled a grey colour and studied over with whitish mammillated nodules, each of which can be felt above the general surface and to be part of a nodule which extends into the lung.

The cut surface of the lung shows excess of pigmentation throughout its extent, but the striking and distinctive feature is the presence of numerous rounded nodules. These are dense, tough, or hard, and the cut surface is grey or black. Each nodule is from 2 to 5 mm. in diameter, but several may be aggregated together to form large composite nodules, or many may be united in a massive fibrosis. In old-standing cases individual nodules may be sharply defined from the surrounding lung tissue, which may show emphysema. The centre of the nodule may have undergone calcareous change. In fatal cases, where exposure to dust has been intense and the course of the disease relatively rapid, the nodules may be so crowded that practically no lung tissue can be seen remaining. This condition is found in some sand-blasters and it appears to follow exposure to highly siliceous dust—i.e., dust containing a very high proportion of free silica.

Gardner [1] described the pathological changes of the lungs in 15 cases of so-called "acute" silicosis. There were masses of small nodules embedded in broad sheets of fibrous tissue, a generalised fibrous thickening of the alveolar walls, and slight involvement of the glands.

The distribution of the fibrosis is not always uniform and discrete, but in certain cases it is condensed in large masses, of rounded or oval shape situated centrally or peripherally in the lung and usually about the middle zones, but the masses may be multiple. This form of distribution is met with typically amongst coal-miners and is described by observers in France and Belgium, as well as in this country.[2,3]

The diagnosis of silicosis is made post mortem on the macroscopic evidence of nodules which are palpable. The fibrous nodule is the essential and characteristic lesion of simple silicosis. Recourse to histological examination is usually necessary for diagnosis only in the presence of complications—notably tuberculosis and malignant disease. The presence of tuberculosis may render the diagnosis very difficult. According to Kettle [4] the differential diagnosis depends on the amount and distribution of the collagen in the tissue reaction; it is a matter of degree and there are no definite standards on which to base an opinion. Policard and Morel [4a] applied a spectrographic method for the detection of mineral particles in pulmonary tissues, and Irwin [4b] has developed a method for the demonstration of siliceous material in histological sections, as an aid to diagnosis, by incineration and treatment with acid.

Clinical and radiological examinations at earlier stages in the progress of the disease can be of immense importance in the diagnosis of complicated cases. Changes in the lymphatic glands usually occur early in the course of silicosis. At first enlarged, they are found in more advanced cases hard and on section dark grey or black, often showing several concentrically arranged systems of nodular fibrosis. The lymphatic glands may show foci of tuberculosis when tuberculous lesions are not apparent in extensively silicotic lungs.

Examination of the Dust-cloud

The International Conference on Silicosis held at Johannesburg in 1930 [5] adopted the view that in order to produce the pathological condition known

* The Milroy lectures for 1936 delivered before the Royal College of Physicians of London on Feb. 27th and March 3rd. The second lecture will appear in the next issue of THE LANCET.

as silicosis, silica must reach the lungs: (*a*) in a chemically uncombined condition, although it might be mixed with other dusts; (*b*) in fine particles of the order of less than 10 microns in diameter; (*c*) in sufficient amount, and over a certain period of time. It is generally agreed that the majority of dust particles found in the lungs are under 5μ and very few exceed 10μ. There are exceptions, for example, in the asbestos industry and in coal-mining, where the length of some particles found in the lungs may greatly exceed 10μ.

Special methods are required for determining the concentration, particle-size distribution, and composition of the dust-clouds to which workers are exposed. For the *numerical method* of dust estimation it is necessary to collect the sample in such a way that it can be examined under a high power of the microscope and the particles counted, measured, and their size-distribution ascertained.

The instruments used for this purpose are of two kinds. With one the sample is taken in a fraction of a second; with the other, the process of sampling can be run for over an hour or more. To the first class belongs the original konimeter introduced by Sir Robert Kotzé in 1916 and used as the standard instrument in the South African gold-mines.[6] The Owens Jet Dust-Counter, which also takes momentary samples, was designed by Dr. J. S. Owens,[7] and has been used in this country for estimating industrial dust-clouds since 1922.[8]

The Greenburg-Smith Impinger dust sampling apparatus was first described by Greenburg and Smith in 1922.[9] The standard instrument as now used in the United States Public Health Service was described by Greenburg and Bloomfield in 1932.[10]

The latest method of collecting samples of atmospheric dust for enumeration is by means of the Thermal Precipitator. This instrument was originally developed by Prof. R. Whytlaw-Gray and Dr. Lomax at Leeds and has been adapted by Green and Watson for estimating industrial dust-clouds as a result of work undertaken for the Medical Research Council.[11] The results of the counts obtained by the numerical method are usually expressed as the number of particles in 1 c.cm. of air. In America they are stated as millions of particles in a cubic foot of air. (One million particles in a cubic foot is approximately equivalent to 35 particles in 1 c.cm.) Dust-counts recorded in these lectures are of samples taken at breathing level and are stated as a number indicating particles in 1 c.cm. and, in parentheses, the approximately equivalent number of millions of particles in a cubic foot, thus: wet, hand grinding table-blades, 280 (8). To save the time of counting a comparative method of quantitation may be used.[12]

By the *gravimetric method* the sample of dust is collected from a large volume of air and is then weighed and the result expressed as the weight of dust in a given volume—e.g., mg. per cubic metre. When used alone the method is open to the criticism that particles of large size are included, which, though they have no pathological importance since they cannot reach the alveoli, yet represent a very high proportion of the weight of the sample. It is useful, however, to combine the numerical and gravimetric methods, especially in initial surveys.

The identification of mineral particles of dust is possible by means of the petrological microscope, but even under favourable conditions it is not possible to identify minerals below about 2 microns in size. This is important from the pathological aspect, for it is just below 2μ that the average size of all mineral particles found in the lungs lies, and the majority, over 80 per cent., of particles in an average industrial dust cloud, are under 2μ in diameter. Chemical analysis may be of value, but it does not give all the information necessary for assessing the pathogenic importance of a sample of dust, because the chemical composition may be less important than the mineralogical character.

A method for employing X ray diffraction for analysis has been introduced.[13]

The Occurrence of Silicosis

Silicosis is not reportable as are certain other occupational diseases, and, until a few years ago, the term had rarely been used for the purpose of certifying the cause of death.

Since the beginning of 1930, by arrangement with the Registrar-General, the Home Office have obtained copies of the certificates of all deaths due to fibrosis of the lungs, silicosis, asbestosis, or pulmonary disease due to dust. It is thus possible, for the first time, to present these data and for the present purpose the five-year period 1930 to 1934 has been taken.

The number of deaths included in the group during the period is 4038. Of these, silicosis was mentioned on the certificate as a cause of death in 1521 cases. The distribution by industries is shown in Table I.

TABLE I.—*Deaths from Silicosis (England and Wales)*

—	Industry.	1930	1931	1932	1933	1934	Total.
1	Sandblasting	10	17	5	10	3	45
2	Steel dressing and cleaning of castings	1	4	1	1	..	7
3	Flint and pebble crushing	1	1	2	4	1	9
4	Refractories industries	11	19	5	7	9	51
5	Scouring powders and abrasive soaps	..	1	3	..	1	5
6	Enamel maker	..	..	..	..	1	1
7	Metal grinding	35	37	31	21	18	142
8	Glaziers' diamond setter	..	..	..	..	1	1
9	Sandstone quarrying and dressing	17	25	13	29	33	117
10	Sandstone masons	35	48	44	75	53	255
11	Grave diggers	1	..	..	..	2	3
12	Tunnel mining (sewage works)	..	1	..	3	1	5
13	Granite quarrying and dressing	2	1	..	3	..	6
14	Slate quarrying and dressing	1	2	5	3	3	14
15	Slate dressing, with sand	..	..	1	..	..	1
16	Gold mining (South Africa)	24	28	16	18	18	104
17	Tin mining	10	18	28	17	18	91
18	Lead mining	2	4	6	1	..	13
19	Copper mining	..	2	1	..	..	3
20	Iron ore (hæmatite) mining	..	10	7	6	13	36
21	Barytes mining	..	1	..	1	1	3
22	Fluorspar mining	..	1	..	..	..	1
23	Mining engineers	2	2	1	2	1	8
24	Diamond mine manager	..	..	..	..	1	1
25	Coal mining	41	50	76	74	85	326
26	Fireclay mining	..	..	..	1	..	1
27	Pottery, manufacture of	53	57	63	53	44	270
28	Leather dressing	1	..	..	..	..	1
29	Metallurgist	..	1	..	..	..	1
	Total silicosis	247	330	308	329	307	1521

It is not claimed that these figures represent, accurately, the occurrence of silicosis. They are almost certainly an understatement.

Occupations in which Silicosis Occurs

It will be convenient in reviewing some of these occupations to pay special attention to the materials and the kind of dust to which workers are exposed during their employment.

The simplest process, from this point of view, is perhaps *sandblasting*. It is the process of projecting sand or other grit, by means of compressed air, against a surface.

It can be carried out in various ways. For large articles the operator works in a specially constructed room, and he is protected by special clothing, and a helmet which is supplied with pure air under pressure. Small articles are treated in a closed cabinet, the worker passing his arms through guarded holes to direct the abrasive while watching the process through a glass panel.

The dust hazard is from the abrasive, usually quartzose sand or crushed flint. Metal grit or shot has now replaced sand as the abrasive to a considerable extent, and where

it is used on clean metal no siliceous dust is produced. When it is used for cleaning metal castings, dust is produced from the adherent moulding sand, which is highly siliceous.

In the 5-year period under review there were 45 deaths from silicosis amongst sandblasters. Of these, 38 were fully investigated and the ages at death and the period of employment on sandblasting were determined. The striking fact which emerges is the short periods of employment which caused death from silicosis, compared with other occupations. All had been employed less than 20 years; 8 less than 5 years; 15 between 6 and 10 years; 11 between 11 and 15 years; and only 4 over 16 years. In the census of England and Wales, 1931, the total numbers employed do not exceed 1395. This gives with 45 deaths in 5 years a mortality-rate of 6·4 per 1000 living. It is actually higher than this because the census figures include shotblasters working on clean metal, who are not exposed to siliceous dust.

Silicosis is found amongst sandblasters in various parts of the world. The same features, a short period of employment and rapid course of the disease, characterise the cases in this occupation.

Flint and pebble crushing.—Flint, a chalcedonic form of silica, is crushed for making chicken grit, sandblasting grit, abrasive papers, and brake-sand for tramcars. The flints may be first calcined, in order to make the material more friable and whiter before crushing. The persons employed are few in number and often the employment is casual or intermittent, so that it is impossible to estimate the actual damage to the health of the men employed. Uncomplicated silicosis, with total disablement, was found in one man who had been employed on flint-grinding during a total period of eleven months, in three months of which there was intermittent exposure to concentrations of dust ranging from 5200 (148) to 10,225 (292). During the five-year period, 1930 to 1934, there were 9 deaths from silicosis.

Refractories industry.—Some 3000 workers are employed. The processes include the quarrying or mining of the raw material, usually a sandstone of the coal measures called ganister, which occurs in open quarries and in mines, or, in some localities, sands or "pocket-clays" are used.

The raw material, which contains usually from 92 to 98 per cent. of silica, is crushed, mixed with suitable bonding substances, and made into silica bricks, silica cement, steel moulders' composition, and similar products for use as refractories in the manufacture of metals, especially steel and steel castings, and in the construction of gas-retorts and flues.

During the five-year period, 51 deaths were certified due to silicosis in persons employed in this industry, a rate of about 3 per 1000 employed. There was some diminution in the number in the later years of the period.

Manufacture of abrasive soaps.—The raw material may be a highly siliceous quartzite rock containing 98 per cent. of silica, or crystalline quartz or quartz-sand. It is used in the manufacture of abrasive soaps by mixing it with powdered soap, anhydrous sodium carbonate, and sometimes other substances.

About ten years ago numerous firms became engaged in this manufacture, who had no knowledge of the dangers of exposure to the dust and were inadequately provided with protective measures. This resulted in the occurrence at one factory of 3 or 4 cases of respiratory disease which was regarded as an acute form of silicosis.[14]

The manufacture in this factory was commenced in 1910, but was not important in quantity until 1921, and in August, 1928, it was given up owing to the death of workers attributed to the inhalation of the dust. Of a total of 81 persons employed on the process, 22 left during the month in which they joined, or in the month following. One of these, who left in September, 1919, died in 1927 of tuberculosis after ten months' illness. Thirteen deaths are known to have occurred amongst the remaining 59 employed, the cause of death being tuberculosis or silicosis. The Chart shows the total number of persons engaged in the manufacture at this factory, the period of employment, and the date of death of those who are known to have died.

A summary of the history of the fatal cases and of one (No. 31) who developed silicosis and is under treatment is as follows:—

CASES OF RESPIRATORY DISEASE INDICATED ON THE CHART

No.

3. Joined July, 1919, aged 14 (female). Left Nov. 20th, 1928. T.B. in sputum December, 1928. Died Jan. 3rd, 1929. At p.m. the tuberculous lung-lesions appeared to be of about four months' duration. It is not probable that this person was a source of infection in the factory. Symptoms dated only from August, sputum was very scanty and positive on the third examination (December, 1928).
7. Joined August, 1919, aged 14 (female). Left September, 1919. Attended hospital March 26th, 1926. Sputum positive; no X ray. Died Jan. 24th, 1927.
17. Joined January, 1923, aged 14 (female). Left March, 1924; rejoined January, 1926, left April, 1928. Sanatorium March, 1928. Sputum negative three times. Died April 26th, 1928.
19. Joined May, 1923, aged 44 (male). Left 1926. Died March 21st, 1926, after illness of one month (T.B. in sputum). No p.m.
24. Joined March, 1924, aged 14 (female). Died Jan. 10th, 1927, after illness of a few months. Certified P.T. No p.m.
26. Joined August, 1924, aged 15 (female), left May, 1928. Died June, 1928, after an illness of five months. P.m. silicosis. T.B. in glands.
35. Joined September, 1925, aged 14 (female), left 1928. Cervical glands removed 1928. Died Oct. 10th, 1935. P.m. massive silicosis of lungs and glands and miliary silicosis of lungs, glands, and spleen.
36. Joined October, 1925, aged 14 (female), left March, 1928. Died June 4th, 1928, after an illness of three months' duration. Sputum repeatedly negative. P.m. chronic fibrosis of lungs and glands. No evidence of tuberculosis, no T.B. found.
39. Joined October, 1925, aged 14 (female), left June, 1926, rejoined February, 1927, left July, 1927. Died Oct. 21st, 1927, after an illness of five months' duration. No p.m. X ray in August; diagnosis miliary T. No sputum.
42. Joined February, 1926, aged 16 (male), off five weeks August–September, 1927, with cough; returned September, 1927, to April, 1928; then to sanatorium until November, 1928. Admitted hospital January, 1929, T.B. in sputum. Died Dec. 27th, 1929. P.m. very advanced P.T. and silicosis.
44. Joined February, 1926, aged 15 (female), to August, 1928. Died P.T. Sept. 8th, 1933.
48. Joined August, 1926, aged 15 (male), left May, 1927. On Nov. 23rd, 1928, X ray negative, clinical examination indefinite. T.B. in sputum June 18th, 1932. Died April 24th, 1933.
49. Joined September, 1926, aged 15 (male), left August, 1928. T.B. in sputum Feb. 7th, 1934. Died P.T. July 7th, 1935.
63. Joined October, 1927, aged 14 (female), left August 1928. T.B. in sputum on July 23rd, 1930. Died P.T. Sept. 9th, 1930.

31. Joined January, 1925, aged 17 (male), left August, 1926. X ray on Sept. 17th, 1932, diagnosis "miliary tuberculosis," later changed to "silicosis." No sputum. Later abscess in neck. Sputum negative on March 7th, 1935 (under treatment).

Cases of silicosis developing rapidly in workers in the manufacture of abrasive polishes have been described by Giese,[15] Teleky,[16] and Kilgore.[17]

Polishing compositions and parting powders containing silica.—The most important forms of silica used for these products are Tripoli and Neuburg chalk. Tripoli is regarded as a chalcedonic variety of silica,[18] and it contains from 96–99 per cent. silica. The substance generally known by the name is imported from Seneca, Missouri. The name comes from Tripoli in North Africa, where the product is a true diatomite.

Two fatal cases of silicosis with tuberculosis have occurred in this country from exposure to dust of Tripoli. The two men had been employed for periods of 10 and 11 years respectively, mixing the ingredients in the manufacture of parting-sand in one case, and of polishing-composition in the other case.

Neuburg chalk is a natural deposit of siliceous material at Neuburg on the Danube. The silica content is given as from 65–97 per cent. At one time this material was believed to be harmless, but recently there have been definite cases of pulmonary disease following exposure to the dust in this country. Koelsch and Kaestle [19] examined 30 workers employed with this material and found 8 who showed signs of silicosis.

Diatomite (kieselguhr, diatomaceous earth) is composed of minute, siliceous skeletons of aquatic plants. When pure it contains up to 96 per cent. of silica. It is used as a mild abrasive or polishing medium, as filtering material, and for insulating properties.

Radiological examination of six workers exposed to dust of kieselguhr for ten years, in this country, showed slight evidence of fibroses in two, and none in the others.

Legge and Rosencrantz [20] made an investigation at a diatomaceous deposit in Santa Barbara County in California, and by clinical and radiological examinations of 108 men, silicosis was found in 81, very early in 15, early in 45, moderately advanced in 15, and advanced in 6. Gudjonsson [21] tells of several workers exposed to high concentrations of fine dust of diatomaceous earth, digging and grinding it for many years. It contained over 90 per cent. free silica. No silicosis in an important degree was found.

Grinding of metals.—The processes which cause silicosis in the grinding of metals are confined to those in which grindstones composed of natural sandstone are used. These are obtained chiefly from the millstone-grit sandstones of Derbyshire and the Sheffield and Newcastle districts. Grinding may be done wet or dry, and the metal being ground may be moved by hand, or by mechanical power on a machine. The processes of dressing the surface of the grindstone are also very important from the health point of view.

The concentration of dust produced varies with the amount of attrition of the grindstone in these processes. Examples of dust concentrations in wet hand-grinding are: table-blades, 280 (8); chisels, 1150 (33); scythes, 660 (19). At wet machine-grinding: paper-knives, 130 (3·7); files, 270 (7·7); edge-tools, 120 (3·4). Dry hand-grinding (with exhaust); sheep-shears, 240 (7).

During the five years, 1930 to 1934, the total number of deaths certified as due to silicosis was 142. The numbers for the consecutive years show a decline, 36, 36, 31, 20, and 19, and it may be that this decline is due to two causes: (1) the change from sandstone to artificial abrasive wheels, and (2) the effect of stringent preventive measures in the grinding rooms.

It was estimated that in 1926 about 1600 natural grindstones were in use in the Sheffield district for hand-grinding. This number was reduced to about 200 in 1933 and many of these grindstones were not in regular use. In the Birmingham district there were 480 natural sandstone wheels in use in 1926 and 452 persons employed. To-day there are only 22 sandstone wheels in use and 18 persons employed. An interesting survey of the Sheffield experience in tuberculosis amongst grinders is contained in the annual report of Dr. John Rennie, the medical officer of health, for 1934 (Table H, p. 21). In this Table the mortality among grinders is compared with that of all persons over 15, in quinquennia from 1886 to 1934, shown as percentage of deaths from tuberculosis of the lungs to all causes. While this has shown amongst all persons over 15 a steady decline from 14·4 per cent. in 1886–90 to 6·7 per cent. in 1931–34, amongst grinders it has shown a variation between 35·6 per cent. reaching a maximum of 53·5 per cent. in 1911–15; it returns to 38·2 per cent. in 1926–30, and in the last quinquennium falls below the starting point to 27·8 per cent. This may be the first sign of a progressive decline. In Table I. of Dr. Rennie's report the mortality-rates for tuberculosis of the lungs amongst grinders and amongst cutlers are shown in comparison with that for all persons over 15 as a rate per 1000 of the population for the years 1925 to 1934. The figures show a reduction from 6·3 to 4·1 amongst grinders, but no reduction amongst cutlers. The rate for all persons over 15 over the period shows a fall from 1·1 to 0·8 per 1000.

Glaziers'-diamond setter.—This unique case was that of a man aged 55, who died of massive and nodular silicosis with tuberculosis. He had been employed for 14 years on a process in which a small abrasive wheel composed of a siliceous stone gave off a fine cloud of dust, which, though scarcely visible, was found to have a concentration of 350 (10). In a petrographic examination of the stone no mineral other than silica was detected.

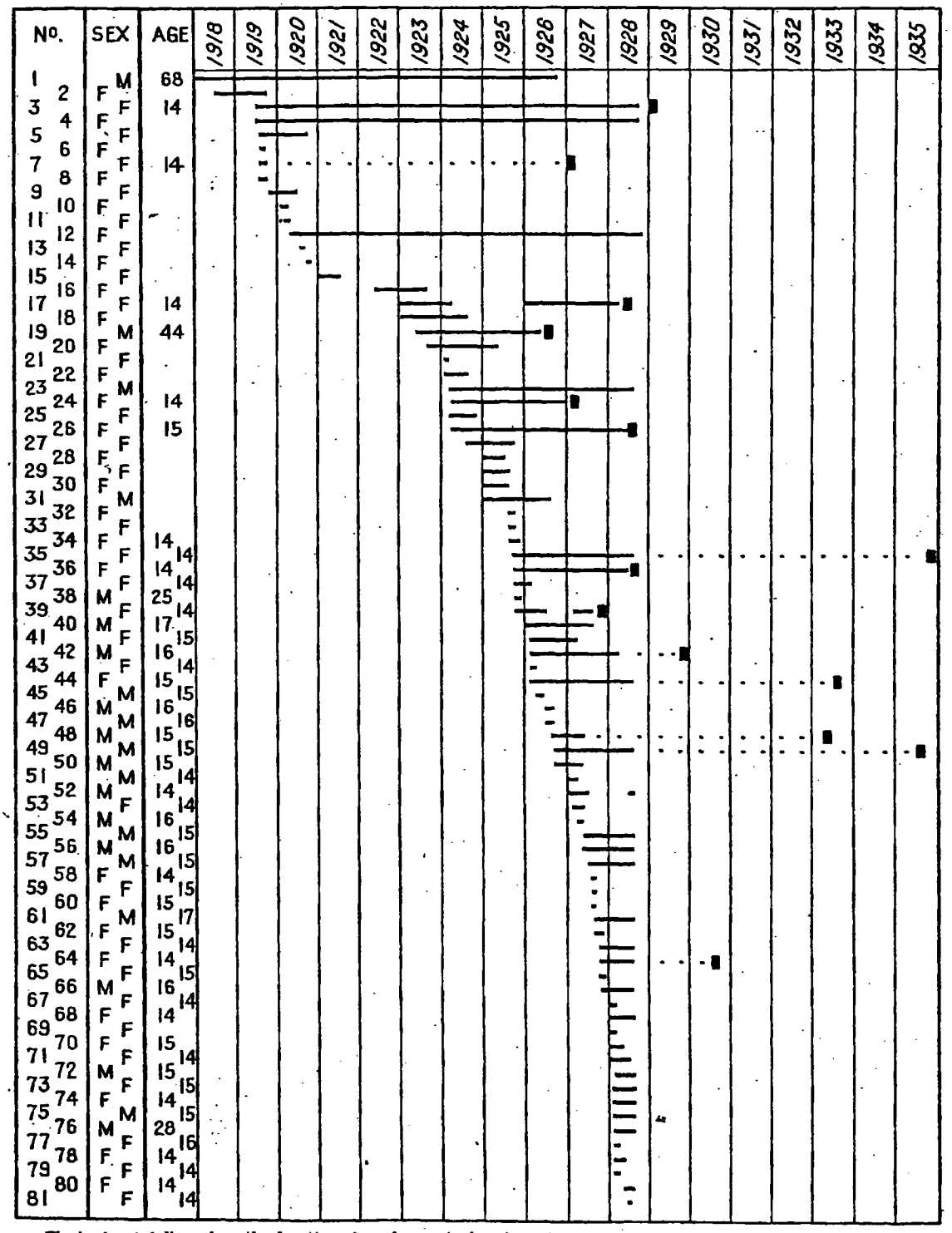
Sandstone industry.—As a cause of silicosis this is the most important of the group of industries in which stone is got and prepared for use or sale. It includes the quarrying, or mining, and dressing of sandstones which are sedimentary siliceous rocks, consisting more or less of quartz-grains, mixed with other minerals, and held together by a cement of varying amount and composition. About 12,000 men are employed on the process in quarries and, in addition, about an equal number of masons are employed in builders' and sculptors' yards throughout the country. The workers who are most affected by silicosis are the stonemasons, and after them the quarrymen and rockgetters.

During the five-year period, 1930 to 1934, the number of fatal cases of silicosis amongst sandstone quarrymen and stonemasons was 372. About 300 of these deaths were amongst stonemasons, and as there are probably about 20,000 sandstone masons in England and Wales, the annual mortality-rate for silicosis is about 3 per 1000 employed.

The following are average concentrations of dust found at various classes of work in the sandstone industry: masons, 545 (16); rockgetters, 319 (9); drillers, 939 (27); quarrymen, 1956 (56); crushermen, 1132 (32). An investigation into the occurrence of silicosis in the sandstone industry was carried out for the Home Office by Sutherland and Bryson [22] in 1929. Of 454 examined clinically, 268 had fibrosis; radiological examination of 266 showed silicosis in 112. In a clinical study of silicosis in 1000 consecutive examinations of workers in quarrying and dressing sandstone, Ferguson [23] found clinical evidence of pulmonary fibrosis in 433. Radiological examination of 173 of these showed silicosis in 109.

Silicosis has been described by Kranenburg [24] in Holland, and by Thiele and Saupe [25] amongst quarry workers in the highly quartzose sandstone of the Elbe district. Koelsch and Kaestle [26] examined 173 sandstone workers and found that 76 per cent. were severely affected after employment for from 20 to 40 years. Teleky and Lochtkemper [27] describe cases of silicosis amongst workers in "grey-wacke." In the sandstone industry of Ohio and Indiana [28] examination of 919 workers showed that the incidence of silicosis and the number of cases of

CHART SHOWING RECORD OF PERSONS EMPLOYED AT ONE FACTORY IN THE MANUFACTURE OF ABRASIVE SOAP-POWDER WITH INCIDENCE OF FATAL RESPIRATORY DISEASE



The horizontal lines show the duration of employment of each worker and the black rectangles the time of death in the fatal cases that are known.

tuberculosis increased with age and length of exposure. An investigation was made by Wulff [28] amongst workers in Sweden on quartzite sandstone containing 94 per cent. of silica.

Silicosis in a grave-digger.—A case was described by Edwards [30] of a man, aged 32, who died of silicosis with tuberculosis after working as a grave-digger for 17 years. The graves were in red sandstone and pneumatic drills were often used for excavating. The work was very dusty and in dry weather the men worked in spells of 20 minutes.

Tunnel mining.—Silicosis was certified as the cause of death in five men employed as tunnel-miners, during the period 1930–34, in which post-mortem examination showed extensive silicosis. In three cases the men had been engaged in tunnelling for water-supply or sewer construction work in sandstone in the North of England. Cases of severe silicosis in tunnel-miners have been described as occurring in Sydney, New York, and Johannesburg.

Granite industry.—The granites are crystalline rocks in which the amount of free silica as quartz rarely exceeds one-third of the weight, and in some granitic igneous rocks frequently included in the commercial term, the proportion of free silica is much less than this. The other constituents are silicates. The processes in getting and dressing granite are similar to those used in the sandstone industry, but pneumatic tools are much more frequently used.

The following are average concentrations of dust found at various classes of work in the granite industry: masons, 412 (12); rockgetters, 343 (10); drillers, 1935 (55); crushermen, 1834 (52).

In the radiograms of granite workers the prevalent type of shadows indicating fibrosis is somewhat different from that found in workers who are exposed to dust containing a high proportion of free silica. It is a finer type of mottling with more linear striation and reticulation, but nodulation of silicotic type is also found in some cases.

An investigation was carried out by Sutherland, Bryson, and Keating [31] in which 494 workers were clinically examined and 211 of these were examined radiologically. Fibrosis was found in 260 (52·6 per cent.) and silicosis in 36 (17 per cent.).

Extensive investigations have been made into the incidence of silicosis amongst granite workers at Vermont.[32] An investigation was made in a representative group of 961 granite workers from four towns in Massachusetts [33] with clinical and radiological examinations. Silicosis was found in 15·2 per cent. and silicosis with tuberculosis in 7·6 per cent. of those examined. Teleky and Lochtkemper [34] found only the slightest evidences of pneumoconiosis in workers on a quartz-free basalt after 20 to 40 years' employment. Gutzeit [35] points out the less severe lung changes which follow exposure to dust of granite compared with those following exposure to dust of quartz-schist with a silica content of 91 to 95 per cent.

Slate.—The industry is concerned with quarrying or mining the rock and making and shaping slates for roofing, and other structural work. It is widely distributed in this country, but about 9000 of the 11,000 wage-earners in the industry are employed in North Wales. Slate is the typical cleaved rock. The most important constituents quantitatively are silica, free as quartz, and combined as silicates of aluminium, iron, alkalis, and other bases. In Penrhyn (Welsh) slates quartz exists to the extent of 34·66 to 43·26 per cent., while the total silica (free and combined) is from 57·75 to 63·01 per cent.[36]

Wade [37] made an investigation into the mortality-rate for tuberculosis amongst slate workers in a district of North Wales. His report shows that from 35 years of age onwards, and, particularly in the later ages of life, those engaged in the slate industry are very unfavourably placed.

A medical inquiry was undertaken for the Mines' Department by Sutherland and Bryson [38] amongst slate workers in North Wales. Clinical examinations of 120 workers showed that severe fibrosis of the lungs existed amongst the older millmen. Fourteen out of 52 millmen examined radiologically showed signs of silicosis.

Dreessen [39] described a survey carried out at two slate quarries and their associated mills. The radiographic shadows in these cases were of the fine reticulated type of fibrosis, with, in advanced cases, dappled areas showing a tendency to conglomerate. They had not the nodular appearance of silicosis.

Gold mining.—Gold mining is of importance to the incidence of silicosis in this country, because of the cases which arise amongst miners who have gone to work for a time in the South African gold-mines and have returned home suffering from the disease. During the five-year period, 1930–34 there were 104 fatal cases of silicosis amongst these miners certified in this country, an annual average of just over 20. Most of them had worked in the tin-mines of Cornwall or the hæmatite-mines of Cumberland, and went to South Africa as skilled miners.

Tin mining.—The mining of tin ore in this country is almost entirely confined to Cornwall. The ore occurs in lodes which traverse granite or "killas," an argillaceous schist. In 1934 there were 1645 wage-earners.

Many tin-miners went from Cornwall to work in the gold-mines of South Africa and elsewhere and, in the early days of the Rand, when conditions were much worse than they are to-day, they contracted pulmonary disease. There is no doubt, however, that silicosis has been caused by working in the tin-mines of Cornwall, for 38 of the rock-drillers referred to in the report on the health of Cornish miners [40] had worked in Cornwall alone. During the five-year period, 1930–34, silicosis was certified as the cause of death in 91 tin-miners, equal to an annual mortality-rate of 11 per 1000 wage-earners employed in 1934.

Hæmatite iron-ore mining.—During the five-year period 1930–34, 36 hæmatite-miners were certified as having died from silicosis. No case was certified during the first year, 1930; the numbers for the subsequent years were 10, 7, 6, and 13 respectively.

Until the last few years silicosis had not been generally regarded as an occupational risk in this industry. In a study of the Registrar-General's statistics for the period 1910–12, Collis [41] pointed out that the Cumberland and Lancashire group of iron-ore miners had a higher mortality-rate than the rest of the ironstone group for phthisis and other respiratory diseases. Since that time a form of pulmonary fibrosis frequently accompanied by tuberculosis has been proved to exist in this industry.

Watson found the following dust concentrations in a hæmatite-mine at a distance of 50 yards from the working face without dust suppression; after an average of six shots with 9 lb. explosive: 222,000 (6343); after an average of four shots with 6 lb. explosive: 105,000 (3000). With the use of a dust-suppressing device the air was apparently free from dust, but actually the thermal precipitator showed a concentration of 30,000 (867). At wet-drilling apparently no dust was produced, but there were actually over 2000 (57) very small particles.

Cronin [42] examined 100 drillers and 16 non-drillers, and found 64 of the former and all the latter to be clinically normal. Thirty drillers showed well-defined and constant features which included dyspnœa on exertion, flattening and diminished expansion of the chest, diminished resonance on percussion, and a change in the quality of the breath sounds.

Radiological examinations of hæmatite-miners show changes in the lungs which resemble those of silicosis as it occurs in other industries, and the changes progress like those of silicosis. Stewart and Faulds [43] have described 15 fatal cases of pulmonary fibrosis in hæmatite-miners. Fatal cases of silicosis have occurred in a hæmatite-mine in the South of England in which the country rock is granite, which contains 27 per cent. of free silica and 64·5 per cent. of total silica.

Coal mining.—During recent years evidence has been accumulating which shows that certain workers employed underground in coal-mines contract a disabling and even fatal fibrosis of the lungs.

In 1909 the Royal Commission on Mines [44] realising the possible danger of drilling in siliceous rock in coal-mines recommended that "water or some equally effective means" should be used when the process is being carried out. Cases of silicosis were found in rock-drillers employed in Somerset coal-mines [45] in 1925, and in 1926, Tattersall [46] reported cases of silicosis among hard-ground workers in coal-mines in South Wales.

The processes underground in a coal-mine which involve direct exposure to siliceous dust are: (1) driving a hard-heading, cross-measure drift, staple pit or sinking pit; and (2) ripping or brushing, i.e., cutting the roof, floor, or sides of a roadway to increase height or width. These processes involve boring the shotholes, shot-firing, and loading the débris.[47]

The following dust-counts are of samples taken by Watson with the thermal precipitator at various distances from the working point in a coal-mine, while means were not taken to suppress the dust. As usual the samples were freed of carbonaceous matter by incineration. Drilling, usually in sandstone, at 15 yards, 10,860 (310); at 65 yards, 860 (25); at 160 yards, 650 (18). Shot-firing (with 24 oz. explosive), at 65 yards distance, 2000 (57); (with 40 oz. explosive), at 160 yards distance, 2300 (65); near the bottoms of the upcast shaft, 550 (15).

In the period June 1st, 1931, to Dec. 31st, 1935, the Silicosis Medical Board issued a total of 987 certificates on account of silicosis in coal-mines. These included 237 suspensions, 581 for total disablement, and 169 for death. The distribution of these cases in the different coal-fields is shown in Table II.

The numbers of certificates issued in respect of fatal cases, if calculated on the number per 100,000 wage-earners employed in December, 1935, is 22·37 for Great Britain; 4·13 for England; and 112·59 for South Wales and Monmouth. The Somerset coal-field has the highest rate, of 146·62 certificates for death per 100,000 wage-earners. No completely satisfactory explanation of this distribution has yet been reached.

Fisher [48] has shown that there is no constant relation between the amount of drilling and the incidence of silicosis. The same is true of the practice of stone-dusting. It is held by some that a risk of bronchitis is incurred,[49] by the men, overheated, travelling out of certain pits by slants through chilling currents of air which reach a velocity of 2500 ft. per minute with a temperature drop of 20° to 40° F. T. David Jones [50] found that four times as many cases of silicosis were certified from collieries with slants as from those without slants. W. R. Jones [51] stated that the mineral sericite is present in the sandstones of the anthracite coal-field, while in those of well-known Scottish collieries, sericite is either absent or rare. According to Cooke,[52] sericite is constantly present in the atmosphere of the Lancashire coal-mines, but he found that silicosis was extremely rare amongst the coal-miners there. That "prolonged exposure to the working conditions incident to coal mining is frequently associated with the gradual development of alterations in the radiological appearances of the lungs" is a conclusion reached as a result of a radiological study of certain groups of industrially healthy South Wales coal-miners.[53] The distinction in the degrees of change found between workers in anthracite coal and in bituminous coal agree with those reported by Russell.[54] In the Welsh study the anthracite group showed a degree of "mottling" greater than that found in the semi-bituminous group. Harper [55] found no radiological difference between cases of silicosis in South Wales and those seen by him in the Belgium coal-fields. Lyle Cummins [56] directed attention to the variety of occupations included in the coal-mining industry.

TABLE II.—*Silicosis in Coal-mines*

Certificates issued, by the Medical Board, for Silicosis and Silicosis with Tuberculosis during the period June 1st, 1931, to Dec. 31st, 1935.

District.	Number of wage-earners on Dec. 14th, 1935.	Number of certificates issued for—		Suspension.
		Death.	Total disablement.	
ENGLAND.				
Northumberland	43,865	..	1	..
Durham	104,244	..	2	..
Cumberland and Westmorland	4,862	..	..	..
South Yorkshire	94,427	4	8	10
West Yorkshire	41,991	..	3	1
Lancashire and Cheshire	59,607	5	5	5
North Derbyshire	41,623	..	..	..
Nottingham	44,832	1	2	..
South Derbyshire	3,179	..	..	..
Leicestershire	9,055	..	..	..
Cannock Chase	21,366	1	2	..
North Staffordshire	22,543	3	16	6
South Staffordshire and Worcestershire	4,460	..	1	..
Shropshire	2,623	..	1	..
Warwickshire	16,391	..	..	..
Forest of Dean	5,075	..	2	2
Bristol	838	..	..	..
Somerset	3,410	5	17	7
Kent	7,303	3	6	4
Total	531,694	22	66	35
WALES.				
South Wales and Monmouth	130,552	147	511	198
North Wales	8,676	..	1	..
Total	139,328	147	512	198
SCOTLAND.				
Fife and Clackmannan	21,625	..	..	1
Lothians (Mid and East)	12,472	..	2	..
Lanarkshire	38,830	..	1	3
Ayrshire	11,311	..	..	..
Total	84,238	..	3	4
GREAT BRITAIN	755,160	169	581	237

A clinical and radiological examination of workers exposed to anthracite coal dust was arranged by the Industrial Pulmonary Disease Committee of the Medical Research Council in 1933.[57] The 40 selected at random from amongst the 250 workers were coal-trimmers at Swansea docks. The committee was unable to find, in this series of cases, any evidence that the inhalation of anthracite or other coal dust had caused fibrosis of the lungs.

Collis and Gilchrist [58] described results of the clinical and radiographic examination of eleven coal-trimmers. The radiographs showed mottling in eight cases, massive bilateral shadows in one, and no definite mottling in two.

In the report of a study made in the Anthracite Region of Pennsylvania by the United States Public Health Service [59] the results of examination of a total number of 2711 workers in 3 anthracite coal-mines are described. Anthraco-silicosis was found in 23 per cent., and the incidence varied with the period of employment, and the concentration and nature of the dust to which they were exposed. Böhme [60] described the results of examination of 3318 rock-drillers working in the Ruhr coal-mines for periods from 5 to 25 years. Of these, 19·44 per cent. showed changes, due to dust, of varying degrees of severity from slight, 14·7 per cent., to grave, 0·18 per cent.

From a study of systematic examinations of 9807 stone-workers in the coal-mines of the Ruhr district

Reichmann and Schürmann [61] found the prevalence and degree of severity to be 1048 (10·6 per cent.) slight, 89 (0·9 per cent.) moderate, and 14 (0·16 per cent.) severe. In 493 cases of severe silicosis the duration of employment was, on the average, 18·3 years. Death was due to simple silicosis with heart failure in 37 per cent., and to an accompanying tuberculosis in 63 per cent.

Courtois [62] made a study of pulmonary disease in 136 coal-miners at Charleroi. Radiological examinations of these workers are classified as negative 33, positive (nodular opacities 30, confluent opacities 61) 91, other conditions (bronchitis, emphysema, and deformities) 12. Post-mortem examination showed that the nodules were typical, simple, silicotic nodules. In the massive fibrotic areas the nodular arrangement had disappeared except at the periphery. Leclercq [63] has described the occurrence of silicosis amongst coal-miners in the north of France. Magnin [64] found that the disease produced amongst workers underground in the coal-mines of Central France is silicosis, which occurs in nodular and pseudo-tumoral forms. Badham and Taylor [65] found in the coal-miners of New South Wales several different types of pulmonary fibrosis. One type was thought to be either a silicatosis due to silicates, or a pure anthracosis due to coal dust.

TABLE III.—*Pottery Industry*

Mortality-rates for silicosis in certain occupations calculated on fatal cases investigated by the Factory Department in the five years, 1930 *to* 1934.

1. EXPOSURE TO FLINT DUST

Occupation: Males (M.), Females (F.).	Estimated numbers employed.		Deaths in 5 years.		Annual mortality-rates per 1000 employed.	
	M.	F.	M.	F.	M.	F.
Flint millers and mill labourers	240	..	7	..	5·84	..
China biscuit ware placers	152	..	9	..	11·84	..
China biscuit oddmen	70	..	7	..	20·00	..
Polishers and grinders	330	..	14	..	8·48	..
China biscuit warehouse	..	223	..	7	..	6·28
Totals	792	223	37	7	9·34	6·28
2. EXPOSED TO DUST OF EARTHENWARE BODY						
Sliphouse	417	..	6	..	2·88	..
Throwers	24	..	..	..	..	..
Turners	132	..	2	..	3·02	..
Handlers	113	336	4	..	7·08	..
Platemakers	364	108	16	..	8·78	..
Dishmakers	113	6	4	..	7·08	..
Handbasin makers	95	4	..	..	..	..
Saucer makers	52	156	3	1	11·54	1·28
Cup makers	46	248	1	3	4·34	2·42
Jiggerers and jolliers	261	122	21	..	16·92	..
Pressers and casters	221	592	19	1	17·18	0·36
Hollow-ware pressers	248	..	22	..	17·74	..
Throwers' assistants	..	30	..	1	..	3·33
Totals*	2086	1602	98	9*	9·49	1·12
3. EXPOSURE TO DUST OF CHINA BODY						
Sliphouse	121	..	1	..	0·16	..
Throwers	102	8	..	..	..	..
Turners	161	18	5	..	0·62	..
Handlers	33	211	..	..	..	..
Platemakers	52	..	2	..	7·69	..
Dishmakers	10	..	..	..	..	..
Handbasin makers	7	..	..	..	..	..
Saucer makers	74	6	5	..	13·51	..
Cup and bowl makers	..	14	..	..	..	..
Jiggerers and jolliers	16	6	..	..	..	..
Jiggerers, flat ware	45	2	..	..	..	..
Pressers and casters	140	152	4	..	5·71	..
Totals	761	417	17	..	4·47	..

* Include 3 deaths of towers (females) for which numbers employed are not obtainable.

Pottery industry.—This industry includes several distinct branches, subdivision being determined by the nature of the article manufactured and the materials entering into the composition of the ware. The occurrence of silicosis is especially associated with two branches—earthenware and china.

In the manufacture of earthenware, the ingredients—ball-clay, china-clay, china-stone, felspar, and flint—in a ground state, are mixed together in the form of liquid slip, from which excess water is removed by pressing. The resulting composite body is made into the consistence convenient for the manufacture of the various articles. In the plastic state it is used by throwers, pressers, jiggerers and jolliers, handlers, modellers, and others,

TABLE IV

Summary of mortality-rates for silicosis in certain occupational groups, calculated on fatal cases investigated by the Factory Department in the five years 1930 *to* 1934.

Occupations exposing workers to dust of—	Numbers employed.	Deaths in 5 years.	Annual mortality-rates per 1000 employed.
	(M. & F.)	(S,* S+T*)	
Flint	1015	44 (27, 17)	8·66
Sanitary ware (body)	736	25 (12, 13)	6·78
General earthenware (body)	3688	107 (54, 53)	4·17
China (body)	1178	17 (7, 10)	2·89
Tiles (body)	975	12 (6, 6)	2·46
Placing sand	947	11 (5, 6)	2·32
Saggers	608	1 (1)	0·33

* S = silicosis; S+T = silicosis with tuberculosis.

according to their craft, in the potters' shops. So long as it remains moist it is harmless, but in the process of manufacture fragments fall on benches and floors or adhere to the clothing of the workers, become dry, and give rise to dust. After the article has been made in the plastic form, partly dried, it is frequently subjected to the process of towing on a revolving disc, thus giving rise to dust. Processes of fettling, to remove irregularities of surface and edges, are done by hand. The articles are then placed in fireclay saggers with some sand, and fired in the oven. On removal from the oven the loosely adherent sand is brushed off. Subsequent processes of decoration and glazing are not associated with the occurrence of silicosis.

For the manufacture of china, the ingredients are calcined bone, china-clay, and china-stone, but it contains no flint. The processes of making the articles are similar, in general, to those employed for earthenware, but the articles require much more support in the process of firing than do earthenware articles, and to provide this support they are placed in finely powdered flint. The flint is prepared by calcining and subsequently crushing it in the dry state. The placing of china in saggers with flint is done by the china biscuit placers. When the saggers of china ware are taken from the oven they are emptied by the placers and "oddmen" and the flint, which was used in placing the ware, is removed by women in a series of processes known as "scouring." Finally, after the ware has been decorated and glazed, blemishes are removed by polishers who frequently use powdered flint, mixed with water, as a finishing abrasive on a rapidly revolving wheel.

Exposure to flint dust in the pottery industry occurs in the processes of the preparation of the flint by grinding; in the manufacture of general earthenware, earthenware tiles, sanitary earthenware, and electrical fittings, amongst those manipulating the earthenware body in the plastic and semi-dry states; in the manufacture of china, amongst those employed in placing the biscuit ware in powdered flint, and in removing the flint from it after firing; and in polishing finished ware with the use of flint. It is in those processes that the highest incidence of silicosis is found to occur. This was brought out as the result of a medical inquiry made by Sutherland and Bryson.[66]

The following results are selected from counts of samples of atmospheric dust taken by Watson with the thermal precipitator at various processes in the pottery industry; at breathing level, unless otherwise stated: (*a*) Flint-grinding, dry, with exhaust, 150 (4·3); flint-milling, wet (general air of the room), 109 (3); placing china biscuit ware (general air of the room), 121 (3·4). (*b*) Earthenware

plate-making, 176 (5); fettling (damp), 168 (4·8); fettling, dry, 201 (5·7). (*c*) Tiles, pressing, 213 (6); fettling, 275 (8). (*d*) Sanitary earthenware; general air of a casting shop, 164 (4·7). (*e*) China, cup-making and turning, general air of the shop, 181 (5).

In countries in which flint is not used in the manufacture of pottery respiratory diseases amongst pottery workers appear to be less prevalent than in England.

The occurrence of silicosis amongst pottery workers has been referred to by Koelsch [67] and Hartmann [68] in Germany, Langelez [69] in Belgium, Møller [70] in Denmark, Quaintance and Morris,[71] Pancoast and Pendergrass,[72 73] and others in America.

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INHIBITION AND INDUCTION OF UTERINE BLEEDING BY MEANS OF ŒSTRONE

BY S. ZUCKERMAN, D.Sc.Lond., M.R.C.S.Eng.

BRIT MEMORIAL RESEARCH FELLOW

(*From the Department of Human Anatomy, Oxford*)

THE increasing clinical importance of œstrone* in the treatment of conditions such as amenorrhœa or sterility resulting from uterine hypoplasia makes it necessary that the available data about the physiological action of the hormone on the primate uterus should be properly understood. At the present time two apparently conflicting statements are made on this subject: the first, that the continuous injection of the hormone inhibits menstrual bleeding, and the second, that uterine bleeding occurs, even in ovariectomised human beings and monkeys, during the course of prolonged œstrin treatment. The purpose of this paper is to examine the facts bearing on these two points.

Allen's [1] observation that the cessation of injections of œstrin into castrated monkeys is succeeded after a few days by a phase of uterine bleeding constituted the first definite knowledge of the endocrine control of the primate endometrium. His finding has been repeatedly confirmed, both on monkeys and on human beings, and the bleeding occasioned in this way has been justifiably related to that occurring either after removal of the ovaries or after rupture of ovarian follicles. In both cases the bleeding that normally follows these procedures can be delayed if treatment with œstrin is begun immediately after the operative interference.

The normal histological effects of œstrin on the primate endometrium have also been elucidated. The hormone, in castrated subjects, promotes considerable mitotic activity in the uterine epithelium,

* Œstrone (keto-hydroxy-œstrin) is at present one of the more readily available and most commonly used of the œstrus-producing female sex hormones to which the general term "œstrin" was applied before their chemical nature had been elucidated. Where the term "œstrin" is used in the present paper it should be taken to mean œstrus-producing female sex hormones in general.

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THE CAUSATION OF PNEUMOCONIOSIS*

PHILIP DRINKER

From the Department of Industrial Hygiene, Harvard School of Public Health, Boston, Mass.

THERE are four different types of reaction produced in man by the inhalation of dust. The first and most important are the pneumoconioses, such as silicosis and asbestosis, which cause specific lung pathology and often are followed by pulmonary tuberculosis. The second type of reaction is caused by toxic dusts like lead, cadmium, and radium. A third type of malady follows the inhalation of finely divided metallic fume particles such as zinc oxide and is known as metal fume fever. Finally, the fourth reaction, allergic in character, is caused by breathing organic dusts such as pollen and certain types of pulverized wood and flour. In all four instances dust inhalation can be the sole cause of the disability but with the toxic dusts characteristic reactions result from swallowing as well as from inhalation. The latter route, however, is much the more important.

In the present instance we are concerned only with the pneumoconioses which result solely from the action of inhaled dust upon the lung tissue. Of necessity then, the dust particles must be small enough to float about in the air and be carried by rather slight air currents; otherwise they cannot be inhaled.

The modern word, *pneumoconiosis*, is a shortening of Zenker's original *pneumonokoniosis*. Zenker (1) depicted lungs definitely damaged by dust particles, but today pneumoconiosis is generally used to describe any lung which has been dusted to more than the normal degree—there need not necessarily be demonstrable lung pathology. Silicosis, asbestosis, anthracosis, anthraco-silicosis, and similar terms indicate the different causative agents (silica, asbestos, and coal) in various kinds of pneumoconiosis.

The International Silicosis Conference in 1930 (2) defined silicosis as a "pathological condition of the lungs due to the inhalation of free silica (SiO_2)". The American Public Health Association (3) described it as a "disease due to breathing air containing silica." Sayers and Jones (4) state that "from the viewpoint of etiology, the harmfulness of a given dust containing free silica is directly influenced by the number of particles of free silica less than 10 microns in diameter that it contains". Collis has claimed for years that free silica, especially quartz, was the all-important

* Received for publication July 14, 1936.
Read before the Harvard University Tercentenary Celebration, 1636-1936, Symposium on "The Environment and its Effect upon Man", Harvard School of Public Health, Boston, August 27, 1936.

factor. In summarizing his South African experience, Watkins-Pitchford (5) wrote in 1927 that dusts other than silica could "give rise to such non-permanent and relatively harmless conditions—one can hardly call them diseases—as anthracosis, aluminosis, siderosis etc".

In figure 1 are shown data from Collis indicating the etiological importance of free silica. The U. S. Public Health Service's data (6), shown in table 1, emphasize still further the importance of free silica.

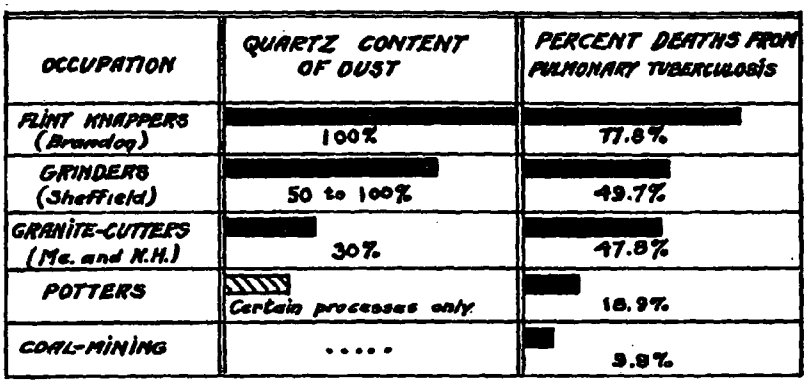

FIG. 1.—Mortality from pulmonary tuberculosis in various dusty trades. (After Collis)

Silica.—Silica occurs most commonly as the mineral, quartz, which is a natural contaminant of most ores, occurs in many rocks, and is found in a fairly pure state as beach sand, chert, flint, sandstone, gritstone, ganister, quartzite, and jasper.

Quartz is a hard crystalline mineral which is weakly birefringent, that is, its two indexes of refraction are near together. Chemically it is very inert and inactive. In fact its hardness and inertness are the two properties which make it especially useful in industry. Other varieties of pure silica such as opal, which is non-crystalline, have not as yet been appraised hygienically and there are no statistical studies to show the relative potency of the various forms of pure silica.

Silicates.—Silicatosis is a word suggested by Badham (7) to describe a lung fibrosis caused by dusts in which silicates and not free silica predominate. The distinction between free and combined silica is best shown by a simple example: A granite dust (8) might have the following mineral composition:

	Per cent
Feldspar (orthoclase)	70
Quartz	25
Mica (muscovite)	5
	100

Chemical analysis of this granite would give the following result:

	Per cent
SiO_2 (total)	72.55
Al_2O_3	14.80
K_2O	12.42
H_2O	0.23
	100.00

TABLE 1

SUMMARY OF THE SIX DUST STUDIES BY THE U. S. PUBLIC HEALTH SERVICE SHOWING THE DUST CONCENTRATION, COMPOSITION AND THE RESULTING HAZARD*

INDUSTRY	AVAERGE DUST COUNT IN MILLIONS OF PARTICLES PER CUBIC FOOT	AVERAGE PERCENTAGE OF FREE SILICA (QUARTZ)	OTHER CHARACTERISTICS OF DUST	DEGREE OF HAZARD UNDER CONDITIONS AS OBSERVED IN EACH STUDY
Granite cutting:				
Hand-pneumatic tool operators	59	35	Balance mostly combined silica	Great excess of pulmonary tuberculosis after 15 years or more exposure; silicosis in from 2 to 10 years. Silicosis after prolonged exposure; no excess of tuberculosis
Surface-machine operators, etc	36			
General air	20			
Less than general air	9			Negative except for occasional nondisabling silicosis
Anthracite coal:				
Rock drillers	82	31	Siliceous rock	Data insufficient; other studies show severe hazard
Miners and miners' helpers	232	1.5	Carbon and inorganic matter	Dyspnea and other signs of pneumoconiosis; excess sickness from respiratory conditions; excess mortality from influenza, pneumonia and possibly tuberculosis
Bituminous coal:				
Rock drillers	78	54	Sandstone	Data insufficient; other studies indicate severe hazard
Loaders and machine men	112	1.2	Carbon	Generalized fibrosis chiefly linear in character; excess mortality from influenza and pneumonia
Cement	26	6–8	Primarily lime	Some early pneumoconiosis; excess of disease of upper respiratory tract and of influenza
Cotton-cloth manufacturing	7	?	Vegetable and silica	Negative
Silverware manufacturing	5	1.7	Metal and other	Negative
Municipal	4	?	Not determined	Negative

*After Thompson, et al.

Recently Jones (9) showed that sericite was the outstanding common mineral constituent of a considerable series of lungs he analyzed. Sericite is a variety of mica with the formula $K_2O \cdot 3Al_2O_3 \cdot 6SiO_2 \cdot 2H_2O$. Its occurrence in nature is widespread but the quantities actually found are much less than those of quartz. It has not been shown that sericite, without quartz, will produce silicotic pathology; all investigations along such lines have been negative.

Asbestos is the only silicate at present recognized as causing pathology which is definitely and distinctly different from that due to silica alone. The condition known as asbestosis, like silicosis, predisposes to tuberculosis but to a much less degree. Since, however, asbestos is handled by far fewer persons than are dusts containing free silica, asbestosis is much less common than silicosis.

Asbestos is not a true mineral but is a name applied to any mineral which is easily separable into more or less flexible fibers. In this country, the commonest asbestos is the fibrous variety of serpentine in the form of the mineral chrysotile, $3MgO \cdot 2SiO_2 \cdot 2H_2O$.

Other silicates such as talc, $3MgO \cdot 4SiO_2 \cdot H_2O$, which resembles asbestos chemically, shale, kaolin, $Al_2O_3 \cdot 2SiO_2 \cdot 2H_2O$, feldspar, and pure mica have been studied in both the field and the laboratory. The pathology they produce is much less significant than that from quartz and the fibrosis rarely is disabling.

Carbon.—An extensive examination of coal miners' lungs was made by Cummins and Sladden (10) as the result of which they wrote that "coal is only retained in large amounts when there is a really high silica content" and "we believe that in the absence of the silica factor there would be, under modern mining conditions, no serious degree of anthracosis. . . ." Men who were engaged in trimming coal ships with virtually no quartz exposure but undoubtedly with very heavy dust exposure, showed some fibrosis but it was not considered disabling (11).

The study made by the U. S. Public Health Service (12) in the anthracite mining district of Pennsylvania adds much weight to statements quoted from Cummins and Sladden, namely, that the harmfulness of a coal dust varies with the silica which contaminates the coal.

Haldane considered that coal dust might even reduce the severity of a quartz dust exposure; he suggested actually blowing coal dust into a mine with high quartz content as an antidote for the quartz dust (13) but no serious attempt apparently was ever made to test the validity of Haldane's claims. The recent statistical analysis of lungs autopsied in the Pittsburgh district (14) shows beyond doubt that city air, contaminated by an unusual amount of coal dust, does not produce a disabling fibrosis. Many of the lungs were markedly pigmented but the pathology found was not, in general, significant.

Calcium and magnesium carbonates.—These substances occur in nature as the minerals calcite, $CaCO_3$, magnesite, $MgCO_3$, and dolomite, $CaCO_3 \cdot MgCO_3$. Limestone and marble contain high percentages of calcite while the bulk of the rock from which cement is made consists of these carbonates. All three minerals are a great deal more soluble in water and in body fluids than

is quartz. Furthermore the solubility of all three substances increases greatly if the solvent is saturated with carbon dioxide, a condition which occurs in the lung fluid which wets inhaled dust.

The several studies which have been made on calcium carbonate dusts indicate that the dusts are not harmful probably because they are so readily dissolved (8).

Gypsum.—This mineral, $CaSO_4 \cdot 2H_2O$, is very common and is mined and milled all over the civilized world. It is an essential ingredient of ordinary plaster and is now used extensively as a wall board. When partially dehydrated it again takes up water readily, but in neither that state nor as the native substance has it been shown to be harmful (15). It is an interesting fact that pure limestone (quartz free), dolomite, and gypsum are recognized universally as the dusts safest (hygienically) to blow into soft coal mines to prevent explosions.

Iron Oxides.—The mining of iron, next to coal, is perhaps the mining industry in which the greatest number of men are employed throughout the world. The ore generally is hematite, Fe_2O_3, but other oxides, and sometimes the sulfide, pyrites, FeS_2, are handled on a considerable scale. As a result of the improvements made in machine-tool steel, it is possible today to see iron dust created by work on lathes, drills, and the like. Yet there are no data to indicate that iron, in the absence of silica, causes pathology in any way comparable to silicosis. However, lungs which have been heavily dusted with iron in any form are generally colored distinctively and were called by Zenker (1) *siderotic.*

Estimation of Dust Exposure

The effect of any inhaled dust varies more or less directly with the duration of the exposure, the dust concentration and the volume of air breathed. In the case of the gas, carbon monoxide, our knowledge of the relationship of these factors is sufficiently exact to permit the use of a simple rule (16) for predicting the effects of breathing various gas concentrations under various conditions. Unfortunately, one cannot estimate or predict the severity of dust exposures with any such nicety.

In cases where men work for a number of years at different jobs with differing degrees of dustiness Bloomfield and DallaValle (17) compute exposures by averaging dustiness in these various jobs over the total period in question. In their anthracite coal dust study they found that computations so made agreed well with the results of the physical and x-ray examinations of the men. A typical example of their method of computing dust exposure is reproduced in table 2.

The advantage of these estimates lies in their simplicity and the fact that they have proven useful in correlating dustiness with physical examinations. The error in such calculations is that the effects of dust do not vary exactly, but only very approximately, with the dust concentration and with the exposure. Thus, Mavrogordato (18) writes that "Lesions of silicosis in a mild degree can be produced in an animal by 30 hr. exposure to intense dust clouds, and one is inclined to suspect that it is intermittent exposure to relatively dense clouds that is the deciding factor in producing the disease in susceptible human subjects." These momentary excesses of dusti-

ness Mavrogordato named *dust floods*. Bloomfield and DallaValle's calculations of necessity ignore dust floods and use only figures of average dustiness.

The Value of Dust Samples

The purpose of dust sampling is to make possible the control or elimination of dustiness rather than to obtain a precise measure of dust concentration. Conditions may vary greatly almost from moment to moment and only occasionally are they reasonably constant during a working shift. In general, then, it is advisable to give the results of the final estimate with an indication of the expected variation. "Thus, as a means of classifying processes according to their respective health hazards, it appears to be unnecessary to arrange them in concentration groups closer than 100 per cent, *i.e.*, 0 to 5, 5 to 10, 10 to 20, 20 to 40, etc." (8). Such an arrangement is consistent with the few American data we now have on standards of dustiness and is in reasonable agreement with Mavrogordato's "figures of merit" as obtained by konimeter samples.

A great deal of time can be saved by ignoring samples which are obviously too dusty—it is as well to take the sample for a matter of record but it is absurd to work long over it if it is certain to be vastly in excess of the objective. Mavrogordato dismisses such samples with the laconic symbols "T.M.C." (too many to count).

TABLE 2

Example of Method Used in Determining an Employee's Total Dust Exposure*

Occupation	Years in occupation	Average dust concentration, millions per cubic foot	Millions of particle years per cubic foot
Slate picker (dry breaker)	2	380	760
Patcher (dry mine)	2	71	142
Mule driver (dry mine)	3	71	213
Miner's laborer (chamber)	3	480	1440
Miner (chamber mining)	3	480	7200
Section foreman	5	7	35
Totals	30		9790

$$\frac{\text{9790 millions of particle years per cubic foot}}{\text{30 years}} = \text{326 millions of particles per cubic foot}$$

* After Bloomfield and DallaValle.

On the other hand, it is well to have records which indicate that the environment is as clean as is desired. In our own experience the ignoring of such samples has raised difficulties in proving in court that adequate dust control had been enforced. There is no doubt whatever that, in the United States at least, dust samples now have a very definite place in depicting working conditions to compensation boards or to a court. Under the circumstances, it is very unwise in making surveys to ignore the clean places.

Methods of Sampling

Dustiness is given gravimetrically if the dust is to be determined chemically and by counts if the chemical determination is especially difficult. Thus, lead is always recorded in milligrams per cubic meter but dust which may cause silicosis is given in particles per cubic foot or per cubic centimeter.

Sometimes one may wish to convert one set of readings into the other. If the dust particles are perfectly uniform and of definite shape such as spheres or cubes and of known composition the conversions can be made with precision. In such a case, it does not matter how dustiness is recorded. But in practice the dust particles are never uniform in size and rarely in composition. Conversions are, therefore, apt to be misleading. For practical work, 1 mg. of fine quartz as collected by the impinger contains 300 million particles (by the light-field counting technic).

A great deal of energy is being wasted in deciding which dust sampling method is best. The rapidity with which new methods are appearing, each claiming unusual points of merit, indicates that standardization is unlikely. An international agreement on a reference standard would be welcomed by all working in this field but, at present, such an agreement seems to be far off.

In the United States the impinger technic is most used, largely because our only extensive field data come from the Public Health Service whose workers favor this instrument. If one wishes to compare his results with those of the Public Health Service he must copy their technic. But copying the Public Health Service's technic for the sake of the legal prestige thus gained has its drawbacks. We know of one case where a gasoline motor-driven generator and electric pump mounted on a mine car and hauled by tractor, with three men in attendance, resulted in obtaining only four samples in one day.

In South Africa where dust sampling has been done on a scale far beyond anything attempted elsewhere the konimeter and sugar tube both have been used. In England great hopes for the new thermal precipitator have been advanced but the results published so far have added nothing significant to the data already available from methods which are less accurate. In Australia several different instruments including Owens' counter have been used while the results coming from Germany were obtained by filter methods and by a modification of Owens' counter.

In our own studies we use several different methods according to the problem. There is every reason to encourage rapid methods which are particularly applicable to routine control and to discourage the widespread use of the impinger technic except for occasional check-ups. The impinger has little to recommend it in control work while a light portable instrument, such as the konimeter, has already proven its value in practice.

Composition of Air-Floated Dusts

It is rarely that one meets exposures to pure silica; generally the dust is a mixture of which the original composition either is fixed, as in granite, or variable as in most foundry and mining operations. But the composition of

the dust breathed from a mixture is seldom the same as that of the parent material.

If a dust arises from the grinding or comminution of a comparatively pure substance, such as beach sand, the resulting material must have substantially the same chemical and mineralogical analysis in all stages of particle subdivision. But if a complex material like granite is ground and the dust then scattered in the air, samples of different particle size will have very different composition.

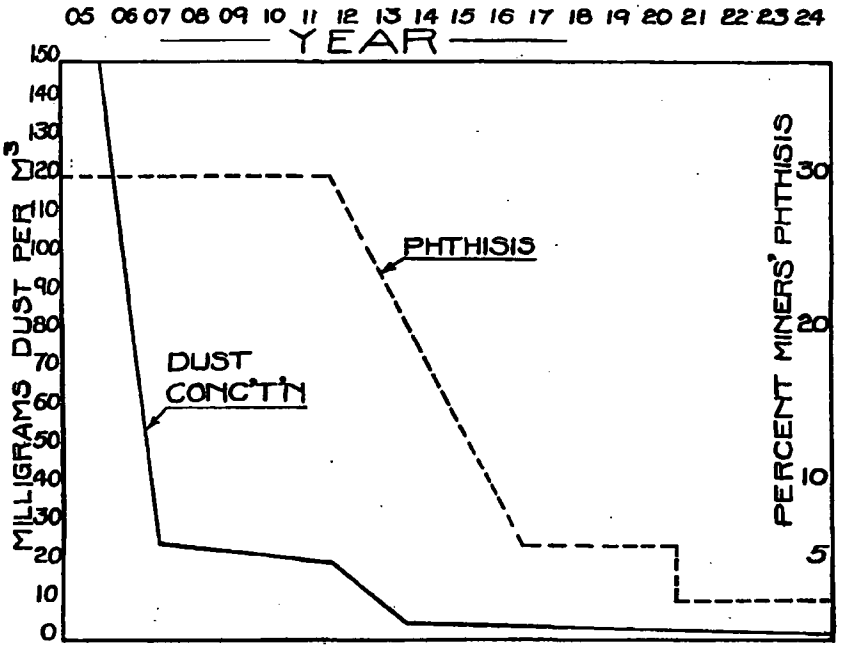

FIG. 2.—Dust control and silicosis in South African gold mines (adapted from Mavrogordato).

Many examples of the changes in composition of air-borne dust can be found in the literature to prove these statements but it is interesting that the facts generally have been ignored. Thus, in this country, investigators frequently sample and count air-borne dust by the impinger technic and then for chemical and mineralogical analyses take samples of material which has settled on rafters which will have a quite different composition. To make bad matters worse, samples for mineralogical and chemical analysis usually have not been graded into various sizes.

Jones (19) has given indisputable proof of the carelessness of which most of us have been guilty in recording the composition of air floated dusts. In support of Jones' contentions an interesting example is given by Drinker and Hatch (8) for foundry dusts. "A good molding sand is made up of about 80 per cent quartz in the shape of coarse particles; the bulk of the re-

maining material is clay in the form of fine particles which coat the coarse quartz grains. The chemical composition of the finer fraction (say below 10 microns) of material in foundry sand is markedly different from the composition of the coarse fraction." Thus, the original material contains about 76 per cent quartz, while particles above 10 microns have 85 per cent quartz and those below 10 microns have only 19 per cent (see table 3). Under the present methods of estimating dust composition and dust hazards a large error obviously would be made by assuming that the original material represented the fine air floated dust.

Another example is given by Jones (19) from the South African "banket" —large crystals of quartz held together by fibrous sericite. In figure 3 are illustrated the changes in dust composition which he noted as the dust was allowed to settle, as it would in practice. Obviously, the quartz content decreases as the sericite increases. Jones remarks that "when a wall built of quartz boulders is pulled down, the bulk of the dust comes not from the quartz boulders but from the mortar."

It would be interesting to know what changes in the composition of Barre granite, of 35 per cent original quartz composition, and of various coal-silica mixtures take place on settlement in air. Obviously, changes will occur (probably great changes) but they have not been measured as yet although we have all welcomed the figures of reasonable dustiness suggested by the U. S. Public Health Service for the two industries in question, granite cutting and coal mining. Their analyses were all made from samples which had settled out on rafters, and no samples were separated into various particle size fractions.

TABLE 3

COMPOSITION OF COARSE AND FINE FRACTIONS OF UNUSED FOUNDRY SAND (CONTAINING NATURAL BOND)

CONSTITUENT	PERCENTAGE BY WEIGHT		
	Total sample	>10μ	<10μ
Combustible	2.3	0.7	12.7
HCl soluble	5.5	1.6	30.5
H_2SiF_6 soluble (clay)	16.0	12.7	37.5
Residue (quartz)	76.3	85.0	19.2
Total	100.0	100.0	100.0

Particle Size

It is well known that in silicotic lungs dust particles under 3 microns vastly outnumber those which are larger. It has been alleged that the respiratory mechanism, the lungs, and the phagocytes are mostly responsible for this size grading. However, it is easy to show that the size grading is done in the air before the dust is breathed and not later in the human body. That is, we find an excess of small particles in the lungs simply because that is the way that they occur in air. It is physically impossible for any but particles below 5 microns

to remain afloat in air long enough to be carried about by gentle air currents and to be inhaled. It is perfectly true that the alveoli are large enough to admit particles 100 or even 200 microns in length and that such particles are occasionally found in lungs (20). But the reason that they are found in lungs so infrequently is that they rarely remain in air more than momentarily. Bloomfield (21) has recorded hundreds of measurements of air floated dusts. His average sizes are all of the order of those found in lungs and in phagocytes.

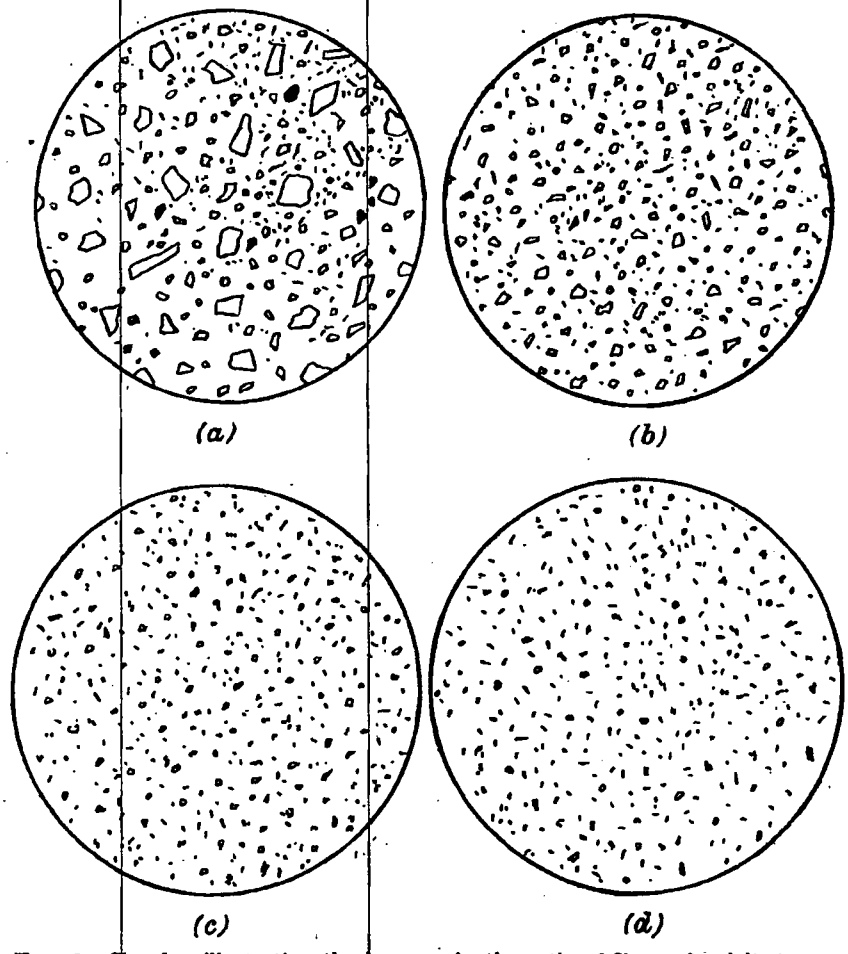

FIG. 3.—Sketches illustrating the increase in the ratio of fibers of sericite to quartz particles at different periods after blasting in a Witwatersrand gold mine. (*a*) about 15 minutes after blasting; (*b*) about 1 hour after blasting; (*c*) about 2½ hours after blasting; and (*d*) about 3 hours after blasting. (After Jones, courtesy Inst. Mining and Metallurgy.)

Gye and Kettle (22) showed that colloidal silica was extremely toxic and that it could initiate fibrosis. It

is claimed occasionally that the damage done by quartz particles is due entirely to those which approach the colloidal in size. Usually particles possessing colloidal as distinguished from crystalloidal properties are considered to be less than 0.1 micron in size. All colloidal particles are so small that they can be measured only ultra-microscopically.

There is no evidence indicating that appreciable or significant quantities of ultra-microscopic quartz can be made by any known process of grinding or comminution, including blasting. In fact, the difficulty of preparing even small specimens of quartz below 0.5 microns is considerable. Furthermore, there are no published data indicating the relative potency of quartz particles in various sizes below 3 microns. Under the circumstances, then, there is no good reason to blame colloidal particles as the initiators of the silicotic nodule when it has been demonstrated again and again that such nodules can be produced by particles approximating the common bacteria in size.

Standards of Dustiness

Under one name or another the plant or mine manager always wants an objective for his dust control program. It serves no useful purpose to evade the issue on the ground that precise figures are not available. Probably they never will be. The practical man argues, very properly, that if dust is the cause of silicosis there must be some degree of dustiness or of air cleanliness which is safe. Having been told that silicosis is a disease caused by breathing silica, the practical operating man naturally expects of the physician or hygienist some objective of air cleanliness, call it by whatever name one likes.

In this country the U. S. Public Health Service studies have furnished the only published data we have from which we may suggest dust standards. In the case of Barre granite it was pointed out that a dustiness of 10–20 million particles per cubic foot was reasonably certain not to cause disability. The coarse dust, in this case, contained about 35 per cent quartz. In their recent anthracite coal study the Public Health Service found that counts of 50 million per cubic foot, with 5 per cent quartz in the coarse dust, seemed safe.

In the case of pure quartz Cummings (4) suggests a figure of 5 million particles per cubic foot which is not far from the South African figure of 1 milligram per cubic meter (figure 3). We have then 50 million for dust with less than 5 per cent quartz and 5 million for pure quartz. It is very questionable if one has any right to interpolate for the quartz percentages between 50 and 5 but the figures certainly invite such interpolation.

These standards do not answer the question of the plant which handles dust of no proven pulmonary significance. What standard should the manager of such a plant take as his objective or need he take any precautions at all? We cannot give him any standards but we can only suggest that he investigate one of the many plants which has reduced dustiness without waiting for any physiological justification. Generally the manager and workmen of a clean plant will uphold eloquently the advantages of dust control.

It is only in South Africa that dustiness and silicosis have been correlated routinely over a considerable period. There they realized at the outset that dustiness would not be controlled properly unless measured and recorded routinely. Concerning the results of their procedure, Irvine (23), Chairman of the Miners' Phthisis Medical Bureau in 1934, stated that "No 'New Rand Miner' who has entered the industry since August, 1923, i.e. 10½ years ago, has as yet contracted silicosis. These facts demonstrate that the engineering and medical measures which have been directed against silicosis have achieved a very significant degree of success." It would be hard to devise a more eloquent or complete proof of the advantage of dust sampling and of dust standards.

Summary

The various pneumoconioses and their causative agents are discussed. Silicosis and asbestos are definite diseases; in anthraco-silicosis and various silicatoses one sees modifications of normal lung conditions. The effects of carbon, or carbonates, of gypsum and of iron oxides are also discussed.

Methods of estimating dust exposures of a workman includes the taking of dust samples from the work place and interpreting the results during the man's total period of exposure. Errors which have been made from estimating the composition of dust from samples which have settled upon rafters instead of using samples taken from the air, are emphasized.

Permissible dust concentrations need not exceed 50 million particles per cubic foot for dust with very low quartz content or 5 million for pure quartz. In defense of the use of standards of dustiness the experience in South Africa where such standards have been applied successfully for many years is cited.

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CLINICAL ASPECTS, DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS*

W. IRVING CLARK

From the Norton Company, Worcester, and Harvard School of Public Health, Boston, Mass.

THERE are two types of dust which may cause some degree of pneumoconiosis; these have been termed inert and active dusts. The determination as to whether or not a dust is active or inert has been reached by pathological examination of the lungs of those dying of pneumoconiosis, by clinical studies, especially x-ray, and by animal experimentation.

REVIEW OF ANIMAL EXPERIMENTS

The study by animal experimentation has been particularly valuable as it provides a method of determining the effect of a given dust upon the living tissues and a comparison with the effect of similar exposure to other dusts. The animals used have been white rats, rabbits, guinea pigs, monkeys, fowl, cats, and, for some experiments, tadpoles and fish (1, p. 44).

The methods used have been:

1. *Dusting.*—This consists in exposing the experimental animal to a "high concentration of dust (up to 10 or 12 billion particles per cu. ft. of air, dark-field) to produce maximum effects in short time." This method might be called the normal breathing method.

2. *Injection of suspensions of dusts.*—This consists in injecting suspensions of dusts into various parts of animals to detect the capacities of different dusts to provoke reaction in the tissues. Results are obtained more rapidly than by dusting but do not correctly show the natural development of disease in the lungs.

The dust suspensions may be introduced into the animal by:

1. Intratracheal or bronchoscopic injections.

2. Intraperitoneal injections (Miller-Sayers method (2)).

3. Subcutaneous injections.

4. Intravenous injections—showing the effects of the dust on the extrapulmonary viscera.

5. Intracutaneous injections—for gross reaction.

6. Intra-lymphatic injections—for reaction of lymph node cells (3).

All animals tested either by dusting or by injection showed a tissue reaction to silica. This reaction was typified by the formation of fibrotic nodules except in the cold blooded animals where there was necrosis followed by some fibrosis. Changes in susceptibility of animals to tuberculosis as a result of the effects of various dusts have also been studied, and the

* Received for publication June 24, 1936.
Read before the Harvard University Tercentenary Celebration, 1636–1936, Symposium on "The Environment and its Effect upon Man," Harvard School of Public Health, Boston, August 27, 1936.
When presented, the paper was illustrated by lantern slides showing x-rays and specimens of pathological conditions.

activating effect of silica demonstrated.

The effect of the inert dusts upon the animal tissue was found similar to that of any foreign body. There was reactionary enlargement of lymphatic nodes, slight fibrotic reaction immediately surrounding the injected dust but no continuation of this fibrosis and no formation of nodules. The exact pathology noted was scattered or clumped cells containing the inert dust lying in the alveoli, slight inflammation or no inflammation of the adjacent walls, a slow accumulation of dust-containing cells in the lymph nodes with enlargement of the nodes, and a deposition of dust about the lymphatics of the lung or pleura. When inert substances were injected intravenously, intraperitoneally or subcutaneously, there was no reaction beyond that of any foreign body (2).

As a result of such experiments which have been repeated in many parts of the world, it is believed that of all suspected dusts only silica has a specific action upon animal tissue which results in fibrotic nodulation. Asbestos fibres produce a peculiar reaction in the lung which is different from silica. Following the inhalation of asbestos dust there are formed "cuffs of more dense connective tissue about the terminal bronchioles" (1, p. 46). Contraction and collapse of the alveoli supplied by the affected bronchioles occurs. This is followed by induration and fibrosis of the collapsed alveoli, presenting the picture of diffuse fibrosis of parts of the affected lung with persistent foci of normal air spaces.

Silicosis

The Committee on Pneumoconiosis and the Committee on Standards of the American Public Health Association (4) adopted in 1932 the following definition: "Silicosis is a disease due to breathing air containing silica (SiO_2) characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis, (some or all of which symptoms may be present) and by characteristic x-ray findings."

I. Pathology of Silicosis

Dust particles after reaching the lung alveoli may remain quiescent for a considerable period as is the case with the inert dusts, or they may penetrate the lymph spaces in small numbers. The bulk, however, are phagocytosed, that is engulfed, by wandering endothelial cells which at first, lining the wall of the alveolus, become detached, and then, having taken up the dust particles, pass by ameboid movement through the walls of the alveolus into the lymph spaces. The cells now slowly migrate to the minute lymph islands which guard the entrance to the small lymphatic vessels, pass through these, thence to the lymph vessels, and finally are caught by the large group of lymphatic glands which form part of the hilus or root of the lung. These wandering cells are commonly called dust cells.

The piling up of cells and the reaction of the glands blocks the free circu-

lation of lymph so that the dust cells tend to move slowly toward the lung roots, blocking the lymph vessels and later the minute lymph masses which guard the entrance to the lymph vessels. The dust cells which are killed by the silica disintegrate and free the silica dust for further injury to the neighboring tissues. The reaction of the tissues to silica is the production of fibrous tissue. Thus fibrous tissue forms along the lymphatic vessels which accompany the blood vessels of the lung, invades the septa between the lobes, and spreads its shoots into the lung tissue itself. The minute masses of lymphatic tissue become fibrotic nodules and these, as time goes on, increase in number to such an extent that, combined with the fibrous tissue of the former lymph channels, conglomerate masses of fibrous tissue are formed, blocking off large portions of the lungs. The blocking of the lymph flow toward the hilus increases the spread of dust cells to the pleura and eventually leads to fibrosis of this structure.

The fibrotic nodule is characteristic of silicosis. It is a small, discrete hyaline mass surrounded by apparently normal lung. It does not exceed 6 mm. in diameter. "Occasionally some of these nodules may show microscopic foci of central necrosis" (1, p. 146).

This increasing amount of fibrous tissue obliterates the alveoli and provokes a compensatory enlargement of the alveoli elsewhere. This condition of enlargement is called emphysema. As the amount of air space is insufficient to permit the normal oxygen-carbon dioxide interchange of the lungs required by the body, the effort of the heart to pump blood fast enough may promote enlargement of that vital organ. This, however, if it occurs, is a terminal condition infrequently observed, as the patient usually develops pulmonary tuberculosis or dies of an intercurrent disease before this stage is reached. It must be remembered that only one-fourth of one lung is necessary for life, and that the amount of fibrosis must be enormous to restrict the lung capacity to this extent.

II. Symptoms

The effect of the underlying pathology upon the workman is at first too slight to be detected by physical examination and the worker shows no symptoms. While there is a pathological condition present, it is to all intents and purposes harmless, in that if it does not progress rapidly it may not provoke symptoms during a worker's life, or only during the last decade. Even respiratory disease of another nature, such as bronchitis or even pneumonia, may occur with recovery during this period.

As the condition progresses, however, more and more of the lung tissue is converted into fibrous tissue, and the patient begins to show certain symptoms. He complains of shortness of breath when he goes up-hill and says that he notices his heart beat on exertion. He also mentions a cough which is at first dry and infrequent, but which gradually becomes moist and frequent. With the cough he raises scanty, stringy sputum which is sometimes discolored. The sputum is never large in amount unless he catches cold and develops bronchitis. At this

stage an attack of bronchitis does not disappear but becomes chronic. During this period the silicotic may complain of pain in the chest due to pleurisy or to epigastric pain, anorexia, and morning vomiting. While he is able to work, the victim cannot carry on his usual tasks and seeks lighter work.

Slowly the shortness of breath increases, the patient has poor lung expansion, and his color becomes pale and his lips bluish.

At this stage pulmonary tuberculosis is a frequent complication. When this occurs, the cough becomes more severe and continuous, the sputum free and moist, areas of dulness to percussion are noted, râles are heard over the chest on physical examination, and cavitation may be detected. Tubercle bacilli may or may not be present in the sputum. Many of these cases are able to carry on light work for years before finally succumbing. Hemorrhage from the lungs occasionally occurs.

During the early stages when perivascular, peribronchial, lymph node reaction is present there are only indefinite physical signs. Irvine (5) describes these as follows:

> 1. A certain lack of elasticity of the chest wall during movements of respiration together with
> 2. A somewhat reduced air entry, and
> 3. 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.

There is little change in these signs as the disease progresses. "The percussion note is somewhat flattened without being definitely dull especially posteriorly. Breath sounds have more definite characteristic thinning, the expiration being longer and fainter" (6). With the advent of tuberculosis the physical signs are those of that disease.

III. X-ray

The most important evidence of silicosis is obtained by x-ray. While a flat film gives valuable information, an accurate diagnosis requires a stereoscopic set of films. In many cases a lateral film is desirable in order to determine the amount of emphysema present and the size of the heart. Fluoroscopic examination is of interest in cases where diaphragmatic adhesions are suspected or shown on the film.

The old classification into first, second and third stages has been recently changed to a more descriptive nomenclature. The conditions noted on the film are now called:

1. Stage of perivascular, peribronchial, lymph node proliferation. Irregular exaggeration of linear markings.
2. Stage of nodulation.
3. Stage of fibrosis with conglomerate masses.
4. Any of above stages complicated by shadows characteristic of tuberculosis.

The first is not considered as diagnostic of silicosis as it may occur in persons who are in good health or in a number of pathological conditions which have nothing to do with silicosis.

The stage of nodulation is pathognomonic of silicosis but may be confused with films of miliary tuberculosis.

The stage of fibrosis with conglomerate masses may be confused with fibroid phthisis.

IV. Diagnosis

Diagnosis is made upon:

1. Occupational history with estimated length of exposure, quality of dust and quantity of dust.
2. Physical examination.
3. X-ray examination.
4. Presence or absence of tuberculosis as a complication.

Of these measures the first and third are most important. Probably the most difficult thing to decide is whether or not tuberculosis is present. Absence of fever and maintenance of weight, with absence of physical signs suggesting active inflammation in the chest, is indicative of simple silicosis.

The absence of tubercle bacilli from the sputum is inconclusive, as many cases of silico-tuberculosis fail to show bacilli in the sputum.

V. Progress

Silicosis once established in the lung has a strong tendency to progress. This appears to be owing to the toxic properties of the silica particle. What causes this toxic action is still in doubt. "Experiments and human pathological material indicate that in high concentrations silica is toxic and kills tissue; prolonged action of lower concentrations cause proliferation and fibrosis" (1, p. 46).

In spite of the fact that quartz is generally recognized as relatively insoluble, there is great rapidity of development of body reactions to silica. "Possibly unexplored properties of silica, electrical charge, adsorption etc., are concerned" (1, p. 48). This progressive tendency has been noted by Irvine, Böhme, Russell and others (1, p. 28).

The question is not what is the present condition of the silicotic, but how rapidly it will advance and how far. This is a serious problem in industry, for a worker having no symptoms and an early stage pathology by x-ray, may develop a disabling silicosis or a complicating tuberculosis. It is, therefore, possible for a worker to contract his disease while working for one employer and to develop symptoms many years later while working for another. If the work he is doing for the second employer involves exposure to an inert dust the employer may have difficulty in proving that this was not the cause of disability.

One of the striking differences between the effects of the inert dusts and silica dust is the lack of progress of pathology in the former as compared with the latter. Böhme (7) found that silicosis progressed after removal from exposure in 20 per cent of the cases diagnosed as having silicosis grade 1, in 40 per cent of the cases in grade 2 and in practically all the cases in grade 3. The experience of the State of Wisconsin, however, suggests that silicosis detected at an early stage in many cases will not progress if the worker is transferred to non-dusty work (8) though the reports of Watkins-Pitchford (9) and Britton and Head (10) make this somewhat doubtful.

Infection may play a part in the development. In fact, it is the frequent and serious complication of silicosis, and of the common infections, pulmonary tuberculosis is by far the most serious.

Tuberculosis may develop early or late in the disease. In those who have an inactive focus in the lung, the silica dust may change the condition to one of activity, or in cases of well-developed silicosis, tuberculosis may be superimposed upon the silicotic fibrosis.

Thus Kettle writes, "harmful dusts if inhaled into the lung may excite to activity a latent tuberculosis infection; they may exaggerate an active tuberculosis lesion or a coincident infection; and they may render the lung less able to cope with a superimposed infection" (11).

The course of silicosis with a secondary tuberculosis is usually slow and without the usual symptoms of intoxication and may be carried for years without serious impairment of working capacity.

While tuberculosis is the most frequent complication and cause of death, other infections do occur. On the Rand pneumonia is a common cause of disability and death. Pope and Zacks found pneumonia occurring with great frequency among foundry workers in Massachusetts. Proske and Sayers have confirmed Cummings' discovery of fuso-spirochetal organisms as a cause of infection among miners in Picher, while chronic bronchitis with asthma is fairly common (12, p. 47).

Collis and Yule (13) compared the mortality experience of an occupational group exposed to silica dust with that of the general population, and with that of an occupational group exposed to dust not containing silica. As a result of the study, they concluded that silica is a body poison like lead. Even though it exerts its primary injurious effects on the respiratory tract, the silica slowly invades the body, involving the circulatory system, the nervous system, the digestive organs, the kidneys and liver, and eventually causes death through its harmful effect upon one or another of these organs.

Asbestosis

"Asbestosis is a pneumoconiosis caused by the inhalation of asbestos dust. It is distinct from silicosis both in its pathology and clinically. Asbestos is a hydrated magnesium silicate containing no free silica but about 44 per cent of combined silica, 43 per cent magnesium, nearly 13 per cent of water and traces of iron and nickel" (14).

I. Pathology of Asbestosis

Asbestos dust differs from other inhalable dusts in that it exists in thread-like fibres in which the diameter may be very small (5 microns or less) but which may be many microns in length.

These dust fibres do not appear to enter the alveoli. They are stopped at the neck of the alveolus where they are phagocytosed or penetrate the tissues. Acting specifically or merely as a foreign body they become surrounded first by mononuclear phagocytes, later by giant cells and last by fibrous tissue. There is no migration in dust cells to the lymphatics and lymph nodes as is seen in silicosis. The newly formed fibrous tissue contracts, constricting the neck of the alveolus so that no air can enter. Collapse of the alveolus follows and subsequent fibrosis.

In this way the lower parts of the lungs are filled with interstitial fibrosis and the air space markedly dimin-

ished. A compensatory emphysema may occur in the upper part of the lungs. Thickening of the pleura over the lower half of the chest is almost always present. Tuberculosis as a complicating factor is not common.

The asbestos fibre after it has become fixed at the neck of the alveolus frequently develops into a "curious body," the asbestosis body of Cooke (15). This is a long, narrow, cylindrical body, golden yellow and translucent, often with rounded ends like a dumbbell. These asbestosis bodies are found on autopsy and occasionally in the sputum. As similar bodies may occur in lungs not exposed to asbestos dust, the finding of these bodies is not diagnostic of asbestosis unless accompanied by a definite history of exposure to asbestos dust.

II. Symptoms of Asbestosis

The symptoms of asbestosis, like those of silicosis, are cough and dyspnea. With the cough there is sputum which may or may not contain asbestosis bodies. Hemoptysis is rare. There is loss of weight in the advanced cases, and marked reduction in the vital capacity and in chest expansion. The patient's face has an "earthy" look.

In advanced cases the dyspnea is out of proportion to the physical signs, being very severe, and is accompanied by blueness of the lips and occasionally by clubbing of the fingers. The fingers frequently show "asbestos corns" from the penetration and irritation of fine spicules of asbestos which are detached from the asbestos in handling.

III. Physical Examination

The patient, if an advanced case, shows loss of weight. The chest is emphysematous, and respiration is shallow, expansion being frequently less than 1 inch. The percussion note is one of dull tympany but with no definite dullness. The breath sounds are distant and expiration prolonged. The heart is normal in size, but lateral x-ray may show an anteroposterior enlargement.

IV. X-ray

This shows a fine mottling of a "ground glass" quality over the lower half of the chest. There is often an obliteration of the costophrenic angle and pleural thickening, shown particularly in the interlobular pleural on the right. The lateral view may show well-marked compensatory emphysema. A spot of tuberculosis at either apex is occasional but rare.

Still later in the course of the disease the appearance is that of a very fine stippling that obliterates most of the natural markings. The pleural shadow is definitely thickened. In some of the advanced cases the heart is enlarged and radiating from it into the lung fields is a series of heavy fibrous bands. This picture has been referred to as "porcupine heart."

Anthraco-Silicosis

This disease occurs among hard coal miners and is commonly known as miners' asthma. It is caused by the inhalation of large amounts of dust consisting of a mixture of anthracite coal and quartz, the quartz coming from the rock in which the coal is imbedded.

The pathology consists of a clogging of the lymph spaces with carbon particles which invade the upper lobes especially. Accompanying this is a linear or nodular fibrosis due to the

inhaled silica particles. When large areas of the lung are involved there is a compensatory emphysema.

I. Symptoms of Anthraco-Silicosis

The cardinal symptom of anthraco-silicosis is shortness of breath, which explains the term "miners' asthma." With this there is cough and sputum. The cough may be dry and the sputum scanty but if infection in the form of bronchitis is present, the sputum is muco-purulent and colored with coal dust. As in silicosis, tuberculosis is a frequent complication.

The physical signs are similar to those of simple silicosis or of silicosis with infection. Diagnosis is made largely by x-ray which gives a picture very similar to that of simple silicosis. The U. S. Public Health study on this problem (16) suggests that the true lung injury is due to the silica inhaled rather than to the carbon particles. Carbon has been found to be harmless when inhaled in the form of smoke and by animal experiment.

Inert Dusts

I. Pathology

The inert dusts are relatively harmless. They may increase the frequency of respiratory disease when inhaled in large amounts, but this has not as yet been proved statistically. Workers inhaling these dusts develop a mild fibrosis which follows the course of the lymphatics along the broncho-arterial tree and is accompanied by enlargement of the trachial lymph nodes. This reaction which is very slow, after a number of years may progress to a moderate amount of interstitial thickening. The pleura may be thickened and there may be diaphragmatic adhesions. These later manifestations only appear after many years of constant exposure to a high concentration of dust.

II. Symptoms Caused by Inert Dusts

The symptoms of the pneumoconiosis of inert dusts are negligible. If the exposure has been long and the dust discharge abnormally heavy, there may be some dyspnea on moderate working. This is not usually severe enough to interfere with the worker's normal activities and may be caused by degenerative changes of the heart due to advancing age, as much as to the pathology of the lung. There is no evidence that the inert dusts, unless mixed with an active dust, can produce disability.

III. X-ray Picture, Physical Examination and Diagnosis

The x-ray picture is that of perivascular, peribronchial, lymph node thickening which does not progress. Some diaphragmatic adhesions may show in cases with long exposure to heavy concentrations of dust. Serial pictures fail to show nodulation or the massive areas of fibrosis which characterize silicosis.

The physical examination of those exposed to inert dusts is usually negative. In a few cases where there has been prolonged exposure to great quantities of dust there may be some restriction of chest expansion and signs of emphysema.

The diagnosis is made by physical examination, occupational history, and x-ray.

A typical inert dust is that of the artificial abrasive, aluminum oxide. This material is widely used in grind-

ing wheels and for polishing, sand paper, etc. In the year 1929 over 71,000 tons were sold or used in plants in the United States and Canada (17).

The use of aluminum oxide creates a certain amount of dust. The effect of this dust has been studied by Clark (18) and Simmons clinically and by Gardner (19) on the experimental animal.

In a factory where artificial abrasives and grinding wheels are manufactured, Clark has studied the effects of inhalation of this substance for 25 years. He believes that in factories which provide proper dust removal, the continuous inhalation of artificial abrasive dust extending over many years of work does not produce the symptoms or present the x-ray findings of crippling fibrosis of the lung; that the number of cases of pulmonary tuberculosis does not greatly exceed the number normally present in the community; and that workers using wheels made of artificial abrasive or using this substance for polishing or other purposes run but slight risk of pneumoconiosis if excessive dust is removed by exhaust systems.

In spite of the fact that clinical and experimental evidence points to the relative harmlessness of the inert dusts, an effort should be made to keep the dust count around 20 million particles per cubic foot of air. This will make working conditions much more satisfactory to the worker and will reduce the hazard of respiratory disease.

Treatment of Pneumoconiosis

The only method of treatment of the pneumoconioses is prevention. Dust in quantity must be eliminated from industry wherever possible, and in most operations this is feasible. In a few where it is not, protective devices such as respirators or positive air pressure helmets must be used by the worker.

When a worker is found upon examination to have lung fibrosis it is wise to keep him at work in a non-dusty department. While his lung condition, if it is due to silica or to asbestos dust, will progress slowly, many years of profitable work are before him unless infection intervenes. As dyspnea increases, lighter work must be provided. If tuberculosis complicates the picture and the patient develops tubercle bacilli in the sputum, he must be isolated from other workers, but even then may be able to do light outdoor work. In more severe cases where there is temperature, all work must, of course, be stopped.

Exposure

The rapidity of development of silicosis and of asbestosis depends upon the amount of dust inhaled and the percentage of free silica (SiO_2) or of asbestos in the dust.

In men exposed to heavy dust clouds with a high concentration of silica, silicosis has developed in as little as 2 years, but under the usual exposures of mining and of industry where the free silica usually varies from 13 to 35 per cent, the development is slow and symptoms do not appear for 15 or more years.

In the case of asbestosis, the development of symptoms is more rapid, 5 to 8 years being the usual required time of exposure.

Efforts are now being made to correlate the exposure with the pathology, as shown by x-ray, and with the physi-

cal signs. In other words, an effort is being made to determine standards of permissible amounts of dust in the working air for both the inert and the harmful dusts. Such standards will be of the greatest value to industry in its effort to reduce its dust hazard to a minimum.

Epidemiology

I. Silicosis

The danger of inhaling inorganic dust has been recognized for centuries, but careful study of the effect of such inhalation was first instituted in South Africa and culminated in the International Conference on Silicosis held in Johannesberg in 1930.

In 1933 Van Siclen (20) published an estimate of the health hazard from dust in the mines and allied industries of the United States. In his study he found that of 7722 men examined in the Tri-State zinc-lead district of southwest Missouri in 1927–28, 5704 were classified as negative for lung disease, 1362 had signs of first stage silicosis, 253 had second stage, and 32 had third stage. The remainder showed signs of tuberculosis with or without silicosis.

In Butte mines (Montana), of 1018 miners 42.4 per cent showed definite signs of dust injury to the lungs.

In the Lead-Deadwood district mines in South Dakota the sickness rates per thousand for respiratory disease was two and a half times greater than in general industry, while the tuberculosis rate was almost ten times greater.

Grouping the various industries studied, Van Siclen found that in metal mining about 62,228 workers were exposed while among those engaged in non-metallic mining or quarrying, 23,665 had a respiratory hazard.

The prevalence of silicosis in the general population was studied by Lanza and Vane in 1934 (21). They cite the following occupations as constituting a definite silicosis hazard:

1. Anthracite-coal and metal mining, quarrying.
2. Certain manufacturing industries such as potteries, glass works, and plants manufacturing granite, sandblasting.
3. Construction work—rock drilling, handling sand and gravel.

Their rough estimate of the number of workers exposed to silica dust to a harmful degree in the United States is upward of 500,000.

A careful study of 2,600 granite and foundry workers has recently been made by Pope and Zacks (22) in which correlation of the duration of exposure to dust and the incidence of silicosis was determined. Their conclusions are as follows:

1. In representative groups of both granite and foundry workers in Massachusetts the frequency of silicosis and of silicosis with tuberculosis was found to be positively correlated with the duration of exposure to dusts containing free silica and to concentration of such dusts in the occupational environment.

2. Among the granite workers examined silicosis alone was found in 15.2 per cent and silicosis complicated with tuberculosis in 7.6 per cent.

3. Tuberculosis is the cause of death in over one-third of all granite workers, a proportionate mortality three times that in foundry workers and four times that in all males of 20 years and over.

In 1907 Summons of the Miners' Phthisis Committee of Australia reported that gold miners there who contracted silicosis died of tuberculosis

(23). "The initial studies of silicosis by workers in South Africa were started by a demand made upon health authorities to determine the cause of the excessive mortality from tuberculosis which was increasing at a rapid rate among the miners there."

Russell found an excessive number of deaths from tuberculosis in his Barre, Vermont study of the health of granite workers (24) and Gardner (25) believes that at least 75 per cent of those who develop silicosis contract tuberculosis.

TABLE 1

SILICOSIS AND ASBESTOSIS IN GREAT BRITAIN, THROUGH 1934*

	NUMBER OF DEATHS	AVERAGE AGE AT DEATH	DURATION OF EMPLOYMENT IN YEARS		
			Maximum	Minimum	Average
Silicosis	261	55.4	60	2.3	34.8
Silicosis with tuberculosis	315	52.5	67	2.0	32.0
Asbestosis	41	41.0	27	1.5	12.9
Asbestosis with tuberculosis	26	38.0	29	0.8	9.9

* J. C. Bridge: Report of Chief Inspector of Factories and Workshops, London, 1934, chapter 3.

II. Incidence of Asbestosis

The health hazard of asbestos dust has only been recently recognized. The Regulations for the Asbestos Industry in England have been in force for only 4 years. Bridge (26) says: "Over a number of years" (number not specified) "the deaths from asbestosis reported to the Department and compiled in 1934 numbered 41 while those from asbestos and tuberculosis numbered 26". (See Table 1.) Wood and Gloyne began their studies on pulmonary asbestosis in 1928 and published results of the study of 100 cases in 1934 (27). Most of these cases worked in the same factory. According to Wood, "pulmonary tuberculosis is so often associated with asbestosis that it seems probable that the association is more than accidental".

Gardner (1, p. 51) says that many autopsies show a combination of tuberculosis with asbestosis and other silicate dusts but "that surveys of living American workmen show no great excess of this infection", while Lanza (28) in a study of dust conditions in asbestos mines and mills in Canada and in fabricating plants along the Atlantic seaboard found in his study of 126 workers by x-ray that 67 were diagnosed as having asbestosis in some form, but that no predisposition to tuberculosis due to asbestos dust was indicated. Clark and Drinker (29) in summing up present beliefs say that while a secondary tubercular infection is not uncommon in asbestosis, it is far less frequent as a complication than in silicosis.

SUMMARY

1. Pneumoconiosis is a disease resulting from the inhalation of inorganic dust.

2. The two pneumoconioses which produce disability are silicosis and asbestosis.

3. The pathology of silicosis is characterized by the presence of fibrotic nodules scattered through both lungs, and that of asbestosis is characterized by an interstitial fibrosis involving the lower half of both lungs.

4. The symptoms of silicosis and asbestosis are similar, the most important being dyspnea.

5. The most common complication is pulmonary tuberculosis which in silicosis is a frequent cause of death.

6. The diagnosis of both silicosis and asbestosis is made largely by a correlation of history and symptoms with the x-ray examination.

7. The treatment of pneumoconiosis is preventive and symptomatic.

8. The course of silicosis and of asbestosis is slow but eventually leads to incapacity due either to the disease itself or to a complication, frequently chronic pulmonary tuberculosis.

9. The number of workers exposed to harmful mineral dusts in mining and in industry in the United States has been estimated as 500,000.

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