The American Ceramic Society

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CERAMIC ABSTRACTS

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Volume 19, 1940

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General

Ceramic laboratories: Georgia School of Technology. ANON. *Ceram. Age*, 34 [1] 12 (1939). Massachusetts Institute of Technology. *Ibid.*, [2] 44. New York State College of Ceramics. *Ibid.*, p. 45. Rutgers University. *Ibid.*, [3] 78. University of Illinois. *Ibid.*, [1] 13.—The laboratories are briefly described and illustrated. F.G.H.

Ceramic schools. Current survey of status and progress. ANON. *Ceram. Age*, 33 [6] 171–81 (1939).—A comprehensive survey of ceramic schools throughout the country is given. The course of instruction, the teaching staff, the research staff, and the student body of each of the following schools are described: Georgia School of Technology, University of Illinois, Massachusetts Institute of Technology, Missouri School of Mines, New York State College of Ceramics, North Carolina State College, University of North Dakota, Ohio State University, Pennsylvania State College, Rutgers University, Virginia Polytechnic Institute, University of Washington, University of Oklahoma, West Virginia University, and Carnegie Institute of Technology. Illustrated. F.G.H.

Contract law for engineers. L. T. PARKER. *Combustion*, 11 [2] 33–34 (1939).—P. explains implied and expressed contracts, as interpreted by the higher courts, with particular reference to the employment of engineers and personal services rendered by them. The liability of employers in such cases is illustrated by a number of court decisions, some of which are cited. H.E.S.

Control of speed is important factor in cement manufacture. ANON. *Concrete, Cement Mill Ed.*, 47, 238–39 (1939).—Early devices were friction driven. Later, variable-speed motors were used. Now a vari-pitch speed changer is used which is operated by a small pilot motor and which is either automatically or remotely controlled. New power self-synchronizing drives may be used to keep a kiln and the kiln feeder in synchronism. W.D.F.

Development of central-heating systems. LUIGI RANZI. *Gesundh.-Ing.*, 60 [50] 783 (1937).—R. describes the development of modern central-heating systems. A new type of installation is explained, and various examples are calculated. W.D.K.

Effect of condensate extraction on efficiency of the heat cycle. W. M. MEIER. *Combustion*, 11 [2] 30–32 (1939).—M. contends that the value of high superheat as a means of improving the efficiency of steam power plants is being overestimated and recommends the use of high initial steam pressures at moderate initial superheat combined with the mechanical extraction of condensate at appropriate stages of the expansion. H.E.S.

Fatalities from silicosis and asbestosis; their incidence in industry. ANON. *Iron & Coal Trades Rev.*, 139 [3732] 336 (1939).—The annual report of the Chief Inspector of Factories shows that from 1935 to 1938 inclusive, deaths from the two lung diseases in the refractories industries and in the manufacture of pottery have been approximately constant, i.e., 68 in 1935, 56 in 1936, 83 in 1937, and 60 in 1938. Sandstone quarrying and dressing took 40 lives in 1935, 43 in 1936, 19 in 1937, and 27 in 1938. Preventive measures have aided in a generally small decrease; some of these measures are as follows: (1) use of alumina for flint in china bedding, (2) use of alumina for siliceous material in parting powders, (3) use of nonsiliceous abrasives for sand or flint in blasting, (4) use of nonsiliceous material for kieselguhr in the slow cooling of steel, and (5) use of nonsiliceous material for silica brick for lining steel and other furnaces. See "China—," *Ceram. Abs.*, 18 [2] 54 (1939). M.H.

Gas heating installation. H. DIETRICH. *Gesundh.-Ing.*, 60 [45] 677 (1937).—D. surveys the developments of gas heating after the World War and discusses the gas single-heating system, the gas central-heating system, special gas tanks, tanks with built-in burners, rentability and gas prices, and the experimental capacity of a boiler on a low pressure steam-heating system and on a warm water heating system. Results of conducted experiments are stated. W.D.K.

Handling and disposing of recovered fly ash and dust. J. T. DEUTSCH. *Combustion*, 11 [5] 22–24 (1939).—Three methods are reviewed, including the vacuum, the sump, and the introduction of the dust by means of a water-cooled ram into a slagging-bottom furnace near the level of the molten slag. The last method is discussed in detail. H.E.S.

Health damage by means of industrial dust. OTTO SCHULTZ. *Gasmaske*, 11, 57–66 (1939).—Pure coal dust is not dangerous; neither is silicon carbide nor corundum dust. Tin, copper, bronze, and aluminum dust make slight alterations in the lungs, and iron oxide makes stronger alterations, but none of these dusts affects the health. Of the dangerous dusts, free silica usually causes silicosis, which is the most widespread and dangerous disease, after only a 5- to 10-year exposure. Asbestos dust causes asbestosis by mechanical irritation. Lung cancer is caused by radioactive dust. The symptoms are like those of silicosis. Chromate dust or mist causes nasal sores and also cancer. It has a long latent period, up to 24 years. Thomas slag causes a severe lung inflammation, as it reduces the resistance of the body to bacteria. This can occur immediately after exposure and has a high fatality. Pyrolusite causes a different lung inflammation. Glass wool acts in a similar manner to asbestos. Over sensitiveness to flour, grain, hair, etc., causes asthma. Certain fungi can grow in the lungs. Methods of prevention are discussed generally. W.D.F.

Healthy air, a guide for plant superintendents and workmen. OTTO ELSNER. *Gesundh.-Ing.*, 60 [44] 676 (1937).—A review issued by the German Society for Labor Protection gives the various kinds of air contamination and testing. Natural and artificial air-conditioning methods are discussed from the viewpoint of health, and a special chapter describes the siting of workshops, offices, etc. W.D.K.

How benzol poisoning may be avoided. ANON. *Gesundh.-Ing.*, 60 [32] 795 (1937).—Forming benzol vapors must immediately be caught by suction. A good supply of fresh air is necessary during the cold season, and the air should be replenished with prewarmed air. Benzol vapors are heavier than air and, for this reason, sink to the floor. When installing a new air-ventilating system, therefore, suction openings should be located in the floor or near it. Pieces treated with a benzol-containing solution should be dried in a well ventilating drier cupboard or special drying rooms. Benzol-containing vessels must be kept covered. W.D.K.

Improved system in the application of noncondensing or extraction turbines. H. W. CROSS AND E. S. WELLS, JR. *Combustion*, 11 [1] 32–35 (1939).—Since electrical output is definitely established by the energy drop between initial and exhaust conditions and by the quantity of steam flow, increasing the total steam pressure at the throttle for a given pressure increases the available energy and consequently the electrical output. The application of a combined desuperheater and feed-water heater at the exhaust is advantageous in realizing gains from the use of higher temperature steam. Examples demonstrating the possibilities inherent in such systems are illustrated and explained. H.E.S.

Lighting for special industrial purposes. W. R. STEVENS. *Trans. Illum. Eng. Soc.* [London], 3 [1] 3–16 (1938).—S. describes installations requiring special lamp fittings for explosive and moisture-ridden atmospheres and the use of special sources of light, such as carbon dioxide discharge, daylight tubes, special daylight tungsten filaments, and high-voltage fluorescent tubes, for color matching surfaces and special inspections. H.K.R.

Living and working conditions in the tristate mining district. EVAN JUST. *Mining Congr. Jour.*, 25 [11] 44–45 (1939).—Dust in the mines is kept within safe limits by wetting down, spraying, and ventilation. Above ground, dust from "chat piles" is practically nonexistent as they are coarse material. On windy days dust is blown from a few flat accumulations of sand. Road dust, however, ac-

tests water hardness has been developed by the German Society for Chemical Apparatus. W.D.K.

Surface heat losses from outside furnace walls. J. C. COUTANT. *Heat Treating & Forging*, 25 [9] 463 (1939).—Aluminum paint reduces heat loss from furnace walls. See *Ceram. Abs.*, 19 [1] 16 (1940). S.S.

BOOKS AND SEPARATE PUBLICATIONS

A.S.T.M. Standards, 1939. AMERICAN SOCIETY FOR TESTING MATERIALS. Philadelphia, Pa. Price to nonmembers $8.00 one part, $15.00 two parts; $22.00 three parts.—*The book is in three parts: I, Metals: Ferrous and nonferrous metals* are discussed; methods of chemical analysis are not given. General testing methods are presented. II, Nonmetallic materials—constructional. This part includes cementitious materials, concrete and aggregates, masonry building units, ceramics, refractories, pyro and tile, thermal insulating materials, timber preservatives, paints, varnishes, and lacquers, road materials, waterproofing and roofing materials, and soils. General *testing methods and thermometers* are described. III, Nonmetallic materials—general. Fuels, petroleum products, electrical insulating materials, rubber textiles, soaps and detergents, paper, plastics, and water are covered. General testing methods are given. Available from Amer. Soc. Testing Materials, 260 S. Broad St., Philadelphia, Pa. F.F.

Finding List for United States Patent, Design, Trade Mark, Reissue, Label, Print, and Plant Patent Numbers. M. RANDALL AND E. B. WATSON. University of California Press, Berkeley, 1938. 31 pp. Price 35¢. Reviewed in *Mech. Eng.*, 61 [5] 402 (1939). F.G.H.

Report of the Department of Scientific and Industrial Research, 1937–1938. H. M. Stationery Office, London. Price 3s. Reviewed in *Pottery & Glass Record*, 21 [4] 89–90 (1939).—Methods have been developed for assessing weathering characteristics of clay products fired under various conditions. Work on clay roofing tile has been continued. Investigations on refractory materials include (1) the pressing process and its relation to the texture of clay, silica, and sillimanite brick; (2) the drying of clayware; and (3) the firing operation. A careful study of the smoke emission from various intermittent kilns firing blue and brindled heavy clay products has been made, and suggestions are advanced for its reduction and control. Laboratory work aiming at relating the conditions of atmosphere and temperature with the color of blue-firing clays has also been undertaken and has progressed satisfactorily.

Drying cracks are due to differences in moisture content established in different parts of the clay body, especially between the surface and the interior. Differential shrinkage thus occurs as drying proceeds, and stresses are produced. The direct method of studying the problem consists in determining the fastest rate at which particular shapes of a given clay can be dried with safety, and the variation of that rate with such factors as the temperature and the size and shape of the body. There is an optimum temperature at which drying is most readily carried out; the smooth nature of the relationships between greatest safe rate of drying and the dimensions of the test pieces suggests that an ultimate understanding of the phenomenon of cracking will be achieved.

A theory of the mechanism of the flow of water through plastic clay during drying has been developed, in which the force causing the flow is regarded as a repulsion between the clay particles which are separated in all directions by water films. A measure of this repulsion is obtained from the moisture contents assumed by the clay when various pressures are applied through porous pistons into which the water is allowed to escape. This pressure is an exponential function of the moisture content as is also the "aqueous conductivity" or the rate of flow of water through unit volume of the clay under *unit* pressure. On this basis the distribution of moisture content at any time throughout a drying shape can be theoretically calculated, the analysis being analogous to that of heat diffusion. A strict solution is not possible, but experiments on a number of clays show a means of approximation which permits the calculation of moisture-content distribution in bars drying from one end only. The experimental verification of the theoretical results confirms the hypothesis.

Many years' study of electrical insulation and the materials used therefor has greatly improved the reliability and the advantageous selection, preparation, and use of insulating materials. Better insulators have been developed, thus reducing the size and eventually the cost of electrical apparatus of all kinds without loss of quality.

The suitability of certain glasses for use in the manufacture of telephone insulators is shown. The degree of homogeneity in optical glass is almost completely determined by the temperature conditions prevailing during the annealing process. An investigation has been made of the maximum temperature gradients in an annealing oven consonant with the production of substantially homogeneous optical glass. The cooling schedules of the annealing process were experimented with *to determine* the minimum period of annealing for glass of high optical quality.

Methods of producing light etch marks on glass have been developed, and an optical system to enable the degree of polish of a glass surface to be assessed has been devised. The preparation of filling materials for etched lines, required to be unaffected under specified conditions, is described. A.B.S.

Silicosis and Asbestosis. Edited by A. J. LANZA. *Oxford University Press, New York*, 1938. 439 pp. Price $4.25. Reviewed in *Mech. Eng.*, 61 [5] 405 (1939).—The medical and public-health aspects of these industrial dust diseases are comprehensively presented in this book, to which several physicians have contributed. The history of the diseases, their symptoms and diagnosis, their pathology, and their prevalence in various occupations are discussed, as well as methods of prevention and control. Each section has a bibliography. F.G.H.

composition and properties of the solid and liquid phases, and the gas phase. P.B. & E.S.

Prevention and treatment of lead poisoning. IMRE HELLER. *Orvosi Hetilap*, 83, 639–41 (1939); *Chem. Abs.*, 33, 8329 (1939).—In Hungarian Pb works, Pb poisoning occurs in 100 to 200 of 10,000 laborers annually. Lead accumulated within the human organism can be mobilized by modifying the Ca/P balance cautiously. This was done in clinical experiments by a diet poor in Ca and P to 10 gm. Na phosphate given each second day for 3 to 4 weeks. Any secondary effects of phosphate can be balanced by the administration of thiosulfate from time to time.

Silicic acid gels: IX. CHARLES B. HURD AND HARRIS W. PATON. *Jour. Phys. Chem.*, 44, 57–62 (1940).—The effect of a change of pH on the time of set of gels produced by mixing solutions of sodium silicate and acetic acid is discussed. For Part V see *Ceram. Abs.*, 15 [3] 108 (1936). R.A.G.

Theory of light scattering. S. PARTHASARATHY. *Phil. Mag.*, 29 [193] 148–53 (1940).—P. shows that the scattering of light observed by Krishnan in binary liquid mixtures ("Molecular. . .," *Ceram. Abs.*, 17 [3] 119 (1938)) is purely molecular, while no scattering arises from clusters, if any, at the critical solution temperature. No new theories as, e.g., those of Frenkel, Gans, or Müller, appear to be necessary to explain Krishnan's results. R.H.

BOOK

Science Front, 1939. F. SHERWOOD TAYLOR. Cassell & Co., Ltd., London. Price 7s 6d. Reviewed in *Times Lit. Supp.*, 39 [1984] 79 (1940).—T. describes recent developments in science, including theoretical researches and technical inventions. Illustrated. K.W.W.B.

PATENTS

Cadmium-red manufacture. WALTER F. MEISTER (Interchemical Corp.). Can. 387,430, March 12, 1940 (June 18, 1937; in U. S. July 9, 1936). G.M.H.

Enamels and glazes and method of producing. CARL OBERLÄNDER AND F. H. ZSCHACKE (Siloto G.m.b.H.). U. S. 2,194,246, March 19, 1940 (May 20, 1936).—A preparation for improving enamels, glazes, etc., comprises a magnesium borosilicate having substantially the following composition: silicic acid 30 to 50, boric acid 10 to 30, magnesium oxide 15 to 35, and alkali compounds 10 to 20%.

Magnesium titanates and method of making. J. A. PLUNKETT AND EUGENE WAGNER (Titanium Alloy Mfg. Co.). U. S. 2,196,325, April 9, 1940 (March 5, 1938).—A calcined synthetic magnesia-titania composition containing chiefly finely divided magnesium titanate chemically combined with 3 to 12% of silica and alumina, the ratio of silica to alumina ranging from 8:1 to 12:1, of particle size substantially all within the range of 0.5 to 2 microns, and having a refractive index of about 2.15.

Production of beryllium compounds. CARLO ADAMOLI (*Perosa Corp.*). U. S. 2,196,048, April 2, 1940 (March 22, 1937).—A process for the production of beryllium oxide from minerals containing beryllium and for the cyclic recovery of the reacting agent employed consists in powdering the beryllium-bearing ore, mixing the powder with an alkali bifluoride in an amount corresponding to 2 mols. of bifluoride for every BeO molecule present in the ore, adding water to form a paste, compressing the paste to briquettes, heating the briquettes to a temperature between 550° and 800°C. to cause the fluorine to combine with the beryllium to form a double alkali fluoride, leaching the reaction product with pure water at a temperature of about 90° to 100°C., adding alkali hydroxide to the alkali beryllium solution to precipitate beryllium as beryllium hydrate and form neutral alkali fluoride, separating the neutral alkali fluoride, calcining the beryllium hydrate to beryllium oxide, and treating the neutral alkali fluoride with a strong acid other than hydrofluoric to convert the neutral alkali fluoride to the corresponding acid fluoride.

Pure titanium dioxide. W. W. PLECHNER AND A. W. HIXSON (National Lead Co.). Can. 386,985, Feb. 20, 1940 (Oct. 12, 1937). G.M.H.

Pure titanium oxide. W. W. PLECHNER AND A. W. HIXSON (National Lead Co.). Can. 386,625, Jan. 30, 1940 (March 7, 1938). G.M.H.

Titanium pigment manufacture. ROBERT W. ANCRUM AND ASSUR G. OPPEGAARD (National Lead Co.). Can. 386,623, Jan. 30, 1940 (Nov. 29, 1937). G.M.H.

Zirconium oxide and method of making. C. J. KINZIE AND D. S. HAKE (Titanium Alloy Mfg. Co.). U. S. 2,194,420, March 19, 1940 (July 23, 1937).—A crystalline zirconium oxide obtained by the calcination of a zirconium-oxygen-carbon compound without substantial grinding of the calcined product characterized as an essentially white powder and as being essentially white in color and consisting of crystal particles having their length greater than their width and their thickness less than their width, an index of refraction of about 2.3 to 2.4, and an adsorbed colorless carbon content of about a stoichiometric equivalent of 1.64% carbon dioxide.

General

Absorption of lead and arsenic through skin. ANON. *Jour. Amer. Med. Assn., Queries & Minor Notes*, 114, 604 (Feb. 17, 1940).—Lead and arsenic absorption through the skin does not occur to an appreciable extent in contact with aqueous solutions of these compounds in concentrations below 0.5 mgm. per 100 cc. of solution. It is unlikely that significant quantities could be ingested because of contaminated hands. F.S.M.

Clinical studies in asbestosis. M. J. STRONG. *Amer. Rev. Tuberc.*, 41, 12–21 (Jan., 1940).—This report is based upon examination of 180 persons formerly employed in the opening, carding, spinning, and weaving departments of an asbestos brake lining plant. F.S.M.

Compensation for industrial injuries and occupational diseases. E. R. KOONTZ. *Jour. Amer. Med. Assn.*, 114, 383–89 (Feb. 17, 1940).—K. gives an excellent discussion of a few of the many legal and medicolegal phases of compensation laws of prime importance to physicians interested in the field of industrial medicine. Tables show the compensability of occupational diseases, limitations of time and cost of medical and hospital benefits in compensable injuries and diseases, and the determination of medical matters under compensation laws; all data are given by states. F.S.M.

Consider the audience. S. MARION TUCKER. *ASTM Bull.*, No. 103, pp. 27–29 (March, 1940).—T. stresses the importance of presenting papers effectively. The causes of complaints are considered. The preparation of a technical paper is taken up with regard to the content and style. The manner of presentation is also considered. The audience must be considered first in technical paper presentation. F.F.

Dust-control systems for the metal-finishing industry. RICHARD T. PAGE. *Metal Cleaning & Finishing*, 10 [3] 531–47 (1938).—Dust is undesirable in any industry. Many of the metal-finishing processes, e.g., sandblasting, grinding, buffing, polishing, etc., are outstanding dust producers. Control procedures discussed are (1) personal respiratory protection, (2) substitution of a less harmful material causing the dust, (3) wet methods, (4) complete enclosure of the dusty process, (5) segregation of part of the equipment, (6) improved ventilation, (7) partial enclosure under negative pressure, and (8) local exhaust ventilation. A detailed study of dust-collection systems is presented. Air motion is discussed, and a table of air speeds in ducts necessary to convey various materials is given. A table shows the minimum thickness of metal for straight exhaust piping. Fans and cleaning apparatus

investigations are of considerable importance and frequently accompany the chemical work. The department is equipped for complete mineralogical examinations by microscopic and other processes used in determining rocks, minerals, ores, and concentrates of all types. It is frequently possible from the results obtained to offer opinions upon the commercial value of a mineral product or to give advice on methods of preparation for the market. Chemical and mechanical analyses can also be made of soils. K.R.

Fatal subacute occupational lead poisoning. WERNER EHRHARDT. *Arch. Gewerbepath. & Gewerbehyg.*, 9, 407–13 (1939).—Industrial lead poisoning as a rule is a chronic poisoning. Characteristic symptoms appearing suddenly, like the colic and intestinal stoppage, show that the poison has been at work in the system for a long time. Acute poisoning as an industrial disease is seldom encountered. E. reports an acute case which came to the Berlin Clinic for Industrial Diseases and discusses its manifestations. On Dec. 4, 1938, a sprayer of lead, 49 years old, was brought to the clinic in a dangerously ill condition. Very carefully and fully he was examined; the next day he died. For several weeks as an occasional worker he had been spraying cable muffles in a cable plant. The workspace was 3 x 4 x 3m.; the spraying was expected to be done under a hood. The worker used a spray pistol in which a lead wire was inserted and the propane gas flame mixed with air, which was introduced into the pistol at the same time, melted the lead wire. The molten lead was at once ejected by the pressure of the air, spraying the muffles revolving in front of it. During this process the worker was expected to wear a mask and to manipulate the pistol through slits. About the middle of November he had been seized with violent pain and vomiting; he was off from work for 8 days and resumed work again late in November. The hood apparatus was then out of repair; three other men working in his absence in the same small space, with very low suction in the ventilating apparatus, caused the returned worker to ask for the night shift where he could be alone. On November 30 he was overcome with the same violent symptoms as before. Being somewhat better by the next night, he insisted on working, only to return home in the same condition, with additional symptoms; his wife noted that his face was as black as lead. E. details the man's further breakdown and the efforts of the company physician in palliation; these failing, the victim was transferred on Dec. 4 to the Occupational Clinic. The plant conditions under which the lead spraying took place were examined and reported on by E. after the autopsy. A large hole was found in the left side of the framework supporting the hood through which the sprayed lead had been escaping into the small enclosure; lead dust lay heavy on the glass valves of appliances carrying the cables; the clothing and shoes worn by the various shifts were loaded and sodden with the scattering lead dust; the mask of the worker was in disrepair, so that he might not have worn it on his final night shifts. He must have breathed the lead dust in its most finely divided particles, i.e., in almost molecular form. Such an aerocolloid condition is taken up by the organism with special ease and constitutes the highest possible risk to the subject. The lungs in this case were permeated with it, and the blood readily carried this fatal dust to the entire system. From the evidence derived from the illness, the autopsy, and the plant, E. concludes that the poisoning could be called acute but also subacute, the worker evidently having been sensitized by his first attack and the subsequent exposures, massive in character, sustained December 1 and 2. An inspector testified that, after being placed on the night shift, this worker had done the work of three ordinary men on the spraying. Hence his constitutional resistance was utterly broken down beyond repair. The lead content of the urine was 2140 γ in 10 gm. of dry feces, 490 γ in 10 gm. of the liver, and 370 γ in 10 gm. of the kidneys. K.R.

Fuel gas generator. HARRY A. CURTIS, RALPH B. STITZER, AND WILDER J. DARBY. *Ind. Eng. Chem.*, 32 [6] 757–62 (1940).—Developments in the use of electrical energy as a source of heat for the carbonization of coal are described. Illustrated. F.G.H.

Glass industry of Ohio. ARTHUR S. WATTS. *Ohio State Univ. Eng. Expt. Sta. News*, 11 [4] 10–13 (1939).—Ohio is the third largest glass-producing state and has twenty glass plants. In 1797 there was a glass plant on the Ohio river; by 1825 they were all over the state. The production of hollow glassware has increased twenty times since glass blowing machinery has been introduced. Ohio is an important producer of flat glass. Today this is drawn from the tank as a continuous flat sheet. Other important products are electric light bulbs, fiber glass, and glass blocks for use in building construction. W.D.F.

Plant laboratory. K. SPINGLER. *Tonind.-Ztg.*, 62 [44] 492–93 (1938).—The plant laboratory has the following tasks: the testing of raw materials and half-finished and finished products, the working out of new formulas, improvement of old shapes and construction of new shapes, and local research. W.K.

Prevention of industrial dermatitis. J. V. KLAUDER, E. R. GROSS, AND H. BROWN. *Arch. Dermatol. & Syphilol.*, 41, 331 (Feb., 1940).—Much of the industrial skin disease is caused by the methods of cleaning the skin. Skin cleansing, methods and materials used, and the use of protective applications are studied. Formulas for eight protective applications are given. Substitutes for mechanic abrasive soap are listed. Various kinds of soaps and detergents, such as triethanolamine soap, naphthenic acid soap, and sulfonated oils, are discussed. The detergent properties of the vegetable meals are not sufficiently appreciated. F.S.M.

Pyrophyllite dust; its effect and control. M. F. TAYLOR. *Amer. Inst. Mining Met. Engrs. Tech. Pub.*, No. 1179; *Mining Tech.*, 4 [3] 13 pp. (1940).—North Carolina is the only commercial source of pyrophyllite at present. Pyrophyllite occurs associated with quartz, together with chloritoid and sericite. All the pyrophyllite marketed is ground; it is classified by air separation. It is mined by open-cut, glory-hole, and underground methods. At only one operation has work been carried on since 1920. Underground, the free silica content of the dust is 35%; 80% of the particles are less than 3 microns in size; dust concentration rises to 500 million particles per cu. ft. when drilling. X-ray examination was made of 101 workers; of those with over two years' exposure, 35% were affected; two died. The lesions were the same as those produced by free silica alone. The threshold dust concentration allowable underground is 10 million particles per cu. ft. Drilling is done wet; stopes are ventilated by "ventubes." Above ground, tight housing for machinery and exhaust ventilation have been installed. These procedures give the required results. W.D.F.

Recent Commission findings. ANON. *Ohio Industrial Commission Monitor*, 13, 41–42 (March, 1940).—An employee of a steel foundry worked from May, 1922, to June, 1939, in the cleaning department in the foundry, where his duties required him to remove hardened sand from castings by use of a pneumatic hammer. A dust count in the room showed an exposure well below the accepted safe standard, but the dust contained approximately 20% of free silica. The Claims Referee was of the opinion that the exposure to silica dust was well established, and the claim was approved as compensable by the Silicosis Referees. Acting under the provisions of an amendment to the Occupational Disease Act effective May 20, 1939, the Commission granted an award of $6500 to the widow and minor children, payable at the maximum rate of $18.75 per week. F.S.M.

Recent developments in relation to silicosis. LEROY U. GARDNER. *Ind. Med.*, 9, 45–51 (Feb., 1940).—Certain tentative conclusions derived from experimental work upon silicosis in progress at the Saranac Laboratory for some years are recorded. As a basic doctrine, G. considers that it is still true that only certain forms of free silica and the group known as asbestos (fibrous silicates)

are capable of producing fibrosis in a normal lung. The trend of some of the Saranac experiments, however, has altered certain concepts previously regarded among research workers as fixed. Inhaled chrysotile asbestos fibers now appear to be irritating, not because they are silicates, but because they are stiff fibers, mechanically irritating to the lungs. Grinding them down very finely so that few of the fibers were longer than 3 microns practically destroyed this irritating property. Even with an average atmospheric concentration of 125 million particles per cu. ft. of air, no fibrosis developed. If the effect of asbestos on the lung were entirely chemical, a decrease in particle size might be expected to increase tissue response. Large particles of free silica have little effect on the body, but small particles increase the reaction of body cells. The histology of early asbestosis, as detailed by G., does not suggest chemical injury. Among dozens of different silicate minerals, only the group of five known as asbestos are unique in having fibrous structure and in being commonly recognized as pulmonary irritants. The group varies in chemical composition within itself more markedly than, as a group, from other silicates. Chrysotile asbestos has the same chemical formula as a nonfibrous silicate, serpentine, which is physiologically inert. In mills that fabricate asbestos textiles there may be much fine dust, but if the dust is composed of serpentine and very short chrysotile fibers, clinical asbestosis will not develop. Free silica dusts, unlike those from asbestos, cause a chemical form of irritation in laboratory animals. G. describes the two histological stages which characterize the tissue reaction in such animals after inhaling excessive quantities of pure quartz or flint dusts in an extremely fine state. In human subjects, only cases of rapidly developing silicosis show evidence of this two-stage reaction. The solubility hypothesis, however, hitherto rather widely used in interpreting silica reactions in the lungs, is declared to rest entirely on indirect evidence. It has never been possible to prove that silica dissolved within the body, because the soluble material immediately combines with local tissue elements. Soluble silica detected in the urine and the blood is now generally accepted as more likely derived from food and drink than from inhaled dust. G. describes many of the Saranac experiments whose outcome individually and collectively is difficult to reconcile with the theory of silica solubility. No fibrosis has ever been produced by single or repeated injections of soluble silica in its various forms. Evidences of specific toxicity exerted by silica upon living tissues have been manifested through long series of experiments lasting years; many concrete examples are given. Intravenous experiments immediately fatal to guinea pigs produced no symptoms in dogs. The toxic effects of quartz have been more or less neutralized by adding small amounts of different substances, e.g., colloidal aluminum hydroxide which gives complete protection in a 0.03% dilution. Particle size is discussed as having new and unexpected relations with both highly toxic and merely slow fibrotic conditions. G. is cautious about drawing final conclusions from animal experiments alone, but he does begin to correlate his experimentally derived facts into a coherent silicosis etiology. K.R.

Relation between the thermal expansion and the properties of ceramic products. W. H. EARHART. *Ohio State Univ. Eng. Expt. Sta. News*, 12 [2] 25–28 (1940).—The composition of a body may be modified to obtain a desired thermal expansion. The thermal history also affects the thermal expansion; certain compositions fired at low temperatures have less expansion than the same body fired at higher temperatures. Thermal expansion may be measured directly on a bar or by the interferometric method. Bodies with a considerable amount of high-expansion quartz in low-expansion clay may show dunting tendencies on cooling. Beta-quartz expands on cooling and may also set up strains. The abrupt quartz inversion point at 573°C. may be destructive. Quartz and clays have fairly uniform thermal expansions, but the feldspars are variable. Hotel china, sanitary ware, and floor tile all have thermal expansion curves lying close together. The quartz present does not seem to affect them. A quartz grain shrinks more rapidly on cooling, and it is thought to pull away from the remainder, so that it does not fill its recess in the body completely on cooling.

W.D.F.

Report of Committee on Ceramic Education. N. W. TAYLOR, *Chairman*. *Bull. Amer. Ceram. Soc.*, 19 [8] 220–21 (1940).

Report of Committee on Publications. JOHN D. SULLIVAN, *Chairman*. *Bull. Amer. Ceram. Soc.*, 19 [8] 222–23 (1940).

Report of Committee on Research. ARTHUR A. WELLS, *Chairman*. *Bull. Amer. Ceram. Soc.*, 19 [8] 214–18 (1940).

Report of Committee on Sections and Divisions. M. E. HOLMES, *Chairman*. *Bull. Amer. Ceram. Soc.*, 19 [8] 221–22 (1940).

Report of Committee on Standards. JOHN W. WHITTEMORE, *Chairman*. *Bull. Amer. Ceram. Soc.*, 19 [8] 218–20 (1940).

Roentgenologic aspects of bronchomycosis. H. P. DOUB. *Radiology*, 34, 287–73 (March, 1940).—The signs and symptoms of the bronchomycoses are similar to those of tuberculosis and they are often at first thought to be tuberculous in origin. D. describes some of the more prominent Roentgen signs of the various bronchomycoses.

F.S.M.

Silicosis. E. R. HAYHURST. *Merck Rept.*, 49, 6, 8, 9, 28, 30, 32 (April, 1940).—H. gives an excellent short review of silicosis. F.S.M.

BOOK

Engineers and Engineering in the Renaissance. WILLIAM BARCLAY PARSONS. Williams & Wilkins Co., Baltimore. 661 pp. Price $8. Reviewed in *Science News Letter*, 37 [12] 192 (1940).—P. discusses the amazing engineering achievements produced during the Renaissance.

P.G.H.

PATENT

Coating and coloration of granulated materials. N. P. HASSBERGER (Bakelite Building Products Co., Inc.). U. S. 2,202,002, May 28, 1940 (June 2, 1936).—A roofing granule has on the exterior thereof a coating comprising a hardened high-alumina cement containing principally calcium aluminates or calcium alumina ferrites and substantially free of free lime in amount to produce objectionable efflorescence in exposure, the surface of the granule being also substantially resistant to chipping and wear and stable with respect to a bituminous bond.

been taken of the advanced training centers in heavily industrialized communities, showing that there is evidently small demand for this field of practice. F.S.M.

I, Lead content of human blood. II, Lead as a poison. J. N. M. CHALMERS. *Lancet*, 238, 447–50, 493–453 (March 9, 1940).—Tompsett's method of determination was used. Blood was obtained from 21 normal people and 50 hospital cases, all with no known exposure to lead, and also from 44 factory workers with a definite lead exposure while manufacturing white lead and storage batteries. The blood of the controls showed a lead content which varied from 30 to 90 μgm. per 100 cc. with a mean of 57. The lead workers, who presented no symptoms of poisoning, had blood lead values ranging from 61 to 102 gm. per 100 cc. with a mean of 104. Augmented concentrations of lead in blood indicate increased absorption of lead but do not constitute evidence of plumbism; they are a *useful estimate* of the degree of exposure. Clinical findings must still continue to govern the diagnosis of lead poisoning. There is a discussion of the problem and also of the value of milk in the diet of lead workers. With a high calcium diet, there is less lead absorbed from the gut than with a low calcium intake. Since, industrially, most lead enters the body through the lungs, the calcium probably acts by helping to fix the lead in the bones and thus prevent it from circulating in the blood. Though of great value, blood studies and similar biochemical investigations must not be given too much consideration. F.S.M.

Lungs of cement workers. S. CACCURI AND L. DI-PRISCO. *Folia Med.*, 26, 7–20 (1940).—A review is given of Italian literature on the subject which seems to give a less favorable picture of the health of cement workers than is found in other countries. Histories of seven cases are given which show silicosis in grades 1 and 2 and emphysema. Tuberculosis was not found. F.S.M.

Manufacture of fired clays (brick, tile, and pottery). A. COULSON. *Argile*, No. 197, pp. 13–21 (1939); No. 198, pp. 3–9 (1940).—C. discusses the raw products used in the manufacture of fired clay products, the formation of clays, and their characteristics and composition. M.V.C.

Mechanism of development of lead anemia: IV, Physiochemical nature of the splenic and liver substances which decrease erythrocytes. S. KIN. *Jour. Med. Coll. Keijo*, 9, 101–107 (1939).—After injection with lead acetate, the spleen and liver seem to contain some specific chemical substances which are able to decrease the number of erythrocytes. K. attempted to determine the physiochemical nature of these substances which were obtained from the splenic and hepatic veins after injection of the lead. The anemic action of the splenic substance disappeared after heating for an hour at 100°. The substance was soluble in water, but did not dissolve in ether and alcohol; it was adsorbed easily by animal charcoal or kaolin. Its action was also retained after ultraviolet radiation for 3 hr. F.S.M.

Metabolism of nucleoproteins in lead poisoning. C. MICHETTI AND F. MOLFINO. *Rass. Med. Applicata Lavoro Ind.*, 10, 562–92 (1939).—Original research on ten cases of lead poisoning shows that there is a pronounced tendency to uric acidemia; this becomes more acute as the poisoning tends towards chronicity. No relation was seen between the degree of uric acidemia and kidney function, and no such connection is likely to exist in cases of recent lead poisoning. F.S.M.

Nature of anthracotic foci. Experimental research. A. C. SAVELLINI. *Folia Med.*, 26, 21–32 (1940).—S. gives a review of the literature and of the views of some French and Italian workers who are of the opinion that the greater part of the "anthracotic" pigment in the lungs is not truly anthracotic but iron of hemolytic origin. Animal experiments by S. lead to the conclusion that carbon and iron participate in the common anthracotic focus; some contain carbon and some iron only. F.S.M.

Occupational dermatoses under the Illinois Workmen's Occupational Diseases Act. H. R. FORRSTER. *Illinois Med. Jour.*, 77, 79–85 (Jan., 1940).—After analyzing various sections of the Illinois law, F. concludes that "any skin disease may therefore legally be an occupational disease in Illinois." F.S.M.

Preventing economizer corrosion. F. J. MATTHEWS. *Brit. Chemmeker*, 48 [373] 332–34 (1940).—M. discusses the causes of corrosion and gives suggestions for the maintenance of the economizer. R.A.H.

Priceless possessions. L. GREENBURG. *Nat. Safety News*, 41, 25–27 (Jan., 1940).—There were 1843 cases of compensated eye injuries in New York State in 1938 and the cost was $1,163,000. Of these, 50% were caused by foreign matter entering the eye and could be avoided by the simple and cheap method of wearing goggles. Ventilation controls are needed for eye hazards of toxic dusts and fumes, and in the past year the Division of Industrial Hygiene has passed and approved 3000 sets of plans for such control in factories. Special goggles are needed for protection against radiant energy, and fatigue of the eyes can be lessened or eliminated by proper illumination. G. discusses these sight-saving means and methods for promoting their adoption. F.S.M.

Two fatal cases of pulmonary asbestosis. E. C. VIGLIANI. *Rass. Med. Applicata Lavoro Ind.*, 11, 28–32 (1940).—The first of these cases concerns a woman who worked for thirty years in a factory using asbestos in the manufacture of cord. The second woman worked for seven years in an asbestos factory. Her susceptibility to colds increased while she was working and she suffered all her life from bronchitis and asthma. She died at the age of *fifty*, with symptoms of a lung disease, nearly thirty years after having stopped work. The autopsy showed interstitial sclerosis of the lungs and asbestosis bodies. F.S.M.

Unemployment benefits no bar to workmen's compensation. ANON. *U. S. Dept. Labor, Monthly Labor Rev.*, 50, 670 (March, 1940).—An employee receiving unemployment benefits under the Unemployment Compensation Act is not disqualified from recovering compensation for the same period under the Workmen's Compensation Act, according to the Supreme Court of Michigan. F.S.M.

SEPARATE PUBLICATIONS

Basophilic Aggregation Test (for Lead Absorption and Lead Poisoning). ANON. Ohio Dept. Health, Adult Hyg. Div., 1940. 7 pp.—The technique and interpretation of results of the basophilic aggregation test are given in a concise form. F.S.M.

Legal Aspects of Engineering. W. C. SADLER. John Wiley & Sons, Inc., New York. 631 pp. Price $4.00.—This is a direct application of the case method to engineering problems, three hundred cases being selected after 6500 legal cases actually dealing with engineering problems were carefully analyzed. Cases dealing with compensation are of the most interest. S. gives a general discussion of the subject. Cases are given to illustrate the following points: elimination of defenses, hazardous employment, employer-employee relation, accidents arising out of and in the course of employment, proximate cause, award, dependency, and jurisdiction. F.S.M.

Manual of Industrial Health Hazards. J. B. FITZLER. Service to Industry, West Hartford, Conn. 175 pp. Price $4.00.—F. presents methods for the evaluation of industrial hygiene exposure resulting from many dusts, vapors, and gases. The first chapter gives principles which are basic procedures for making surveys and describes sampling devices. The other chapters are given over to the materials or groups of materials which may cause industrial disease. For each substance, F. gives the occurrence and uses, certain physical properties, the clinical symptomatology it produces in man, the physiological responses of man to various concentrations, the concentrations allowable in the air of the workplace, and a simple method of estimation in air. Numerous references are given. An appendix includes useful constants, formulas, and instructions for the preparation of standard solutions. F.S.M.

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Iron and Coal Trades Review, September 8, 1939

THE

IRON & COAL TRADES REVIE

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Iron-Ore Extraction in the Midlands*

OPEN CAST WORKING AND UNDERGROUND MINING

METHODS OF SURFACE WORKING

ALL ironstone in the Midlands was originally got by hand either at outcrops or not more than 15 to 20 ft. below the surface. The reinstatement of such land for agricultural use immediately after the working presented no difficulty. No great masses of subsoil had been disturbed; the topsoil was merely thrown on one side and then returned without any cost for haulage. At the turn of the century small steam excavators with a short-arm jib were introduced for the purpose of removing the overburden, but all land was still restored by hand. When a quarry was about to become too deep for restoration it was abandoned or mined. Excavators were then introduced for removing the ore itself. In the expansion of development due to the War, the American long jib and mechanical digger with a wide dumping radius was introduced. About this time some areas were first left unrestored. This was, at first not so much due to technical difficulties as to a company going out of business.

The position became serious when, in the thirties, Stewarts and Lloyds left Scunthorpe and came to Northants. Since they were latecomers they found all the areas of easily got ore controlled by other firms, and they were forced to lease areas of deep overburden. They began work on these with an electrical excavator which, with a 9 cub. yd. bucket (1 cub. yd. represents very approximately 1 ton), a dumping radius of 101 ft., and a dumping height of 35 ft., could deal with overburden up to about 60 ft. in depth.

Ironstone is won by a number of different machines whose operations have somewhat different effects. They include the dragline which works on the principle of a rake; the excavator or "digger" with a short arm (the "jib") dumping on to a conveyor or wagons; the long-jib excavator which dispenses with the latter, and the Lubecker (land) dredger which can dump sand or other very light soil evenly. The dredger creates no problem of levelling because of the number of small bucket loads dumped, but for this reason it is only efficient in sand or light soil. There are a great number of combinations of these machines, with or without hand labour, which are used in different quarries, and it would be difficult to give a complete account of them. The following are descriptions of the work in three quarries which are representative of certain typical conditions.

(1) A 1¼ cub. yd. dragline at A (*Fig.* 1) removes the 2-ft. overburden (including 9 in. of topsoil) and deposits it at C in a very shallow "hill and dale" formation which is then levelled by the ¾th cub. yd. dragline at C. The operation is then completed by a 4-cub. yd. steam navvy following up behind dragline A and loading the *ironstone* into wagons B. The face at D gradually recedes as the ironstone is removed. The working is like a moveable cliff.

(2) A short jib excavator may be used in conjunction with a conveyor as in *Fig.* 2.

The excavator A removes overburden from the face B and then empties the load into a small truck, or dumping tram, on the steel-framed conveyor at C. This runs up to the other end at D and tips over. The function of the conveyor is therefore to move the dumped overburden away as far as possible so that there is no danger of the navvy which is removing the ironstone becoming buried. The overburden is dumped by the conveyor in a ridge and furrow formation similar to that which results from the use of a long jib excavator but in a rather more very line.

The appended description of iron-ore extraction in the Midlands has been taken from the Report* of the Committee on the Restoration of Land Affected by Iron Ore Working, and deals with methods of removing the overburden, and winning in underground workings.

The size of hill and dale varies also with the depth of overburden, since the excavator used varies generally depth of the overburden to be rem can therefore talk of a "normal" and dale for a particular depth of But it is understood that only a very ge tionship is meant. The general tendency of importance in connection costs of levelling. When the cut is a final gullet is left. This may be at of 200 to 300 ft. wide and 60 ft. de may form a considerable difficulty i screen to the quarry, particularly for restoration work.

Local Objections to Large Exc

It is the operation of these machi has alarmed local opinion and which attention to the whole question of t working of ironstone in this country. restriction to the coming of the largest has been to suggest that they are not It is seen that ironstone was won be machines came and it is suggested t ought, in view of the devastating ef to be a reversion to older methods o recovery of yet newer ones. If it is poin reply that ironstone could not be got ally at 60 ft. depth by the old met sometimes argued that, if the produ

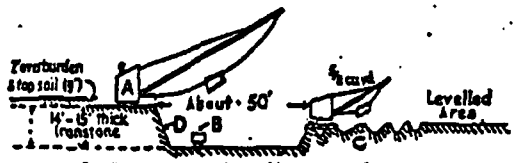

FIG. 1.—DRAG-LINE METHOD OF EXTRACTION.

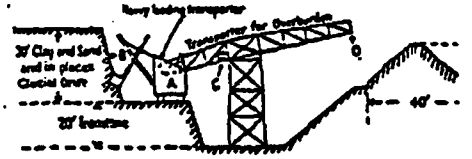

FIG. 2.—SHORT JIB EXTRACTOR AND TRANSPORTER

(3) The overburden can be removed and dumped by a long-jib excavator. The stretches (*Fig.* 3) are of 2·3-cub. yd. excavator at work. These machines are the largest in Europe, and they have an output of about 500 tons per hr. each. In fact the whole area assumes the aspect of having gigantic furrows, which are locally known as "hill and dale." The ridges consist predominantly of subsoil, and in them the topsoil is irretrievably lost. The size of these furrows depends on the size of the navvy used for the excavation. Where the largest navvies are in use, the distance from crest to crest is from 40 to 50 ft., the depth varying from 10 ft. to 16 ft., but where smaller excavators have been used in the past, the crest to crest distance is between 20 ft. and 30 ft., and the depth from 6 ft. to about 10 ft. The shape and size of the hills and dales depend not only on the type of machinery used for putting the material across, but also on the angle of repose of the dumped material. The natural angle of repose of any particular soil composition will generally only be found where it is free to trickle; e.g., on the slope of the dumped material stretching down to the quarry floor. Measurements are often made of this angle for different materials and combinations of materials, because it is this angle that determines the distance between the top of the dumped overburden and the face of the excavation. These measurements show that an average angle of repose is 1½ horizontally to 1 vertically, although it may be flatter in boulder clay where there is a good deal of slipping and steeper in limestone. The angle of actual hill and dale is nearly always flatter than the angle of repose proper because the material is partially spread as it is emptied from the excavator.

put to it, they could invent new mach would abolish or modify hill and dale. engineering developments as they app Committee from the evidence put be included:—

(1) An attempt might be made to im excavator. The present performance is function of the radius of the jib an angle of repose of the dumped soil. cause these two factors are fixed, that dale formation occurs. If the length could be varied slightly with each bu then hill and dale of the present would not be formed and a surface wo which, if very irregular, would be much level. If the length of the jib were however, so as to give a choice of which each bucket could be emptied, t of the entire machine, which is alrea 300 tons, would be increased to an imp extent. Further, if any attempt were dump material anywhere near the ang angle of repose of the tipped soil wo that the excavator itself would be imm

(2) A new conveyor might be desig position of which could be altered later load placed on it by a small excavat witness suggested this, but the Committ it an unlikely development. The pre veyors cannot be used where there is of overburden of more than 40 ft. b the strain which would be imposed arm of the conveyor when the truck was away from its point of support. It w be easy, therefore, to design a conveyo would produce better results with an ov of 60 ft. or more in depth than are the present one with an overburden of

*H.M. Stationery Office, York House, Kingsway, London, W.C.2. [Price 6s. 6d.]

Fatalities from Silicosis and Asbestosis

THEIR INCIDENCE IN INDUSTRY

The problems associated with silicosis and asbestosis amongst industrial workers are receiving close attention. The former has tended to increase, and the latter, on balance, to decrease, in recent years

A SECTION of the Annual Report* of the Chief Inspector of Factories dealing with health refers to the question of dust and its effects, including silicosis and asbestosis. It is pointed out that for some years past, particulars of the total number of deaths from these two causes, which have been investigated by the Department, have been given and brought up to date. These figures have been specially useful from the practical point of view in aiding the installation of preventive measures of the necessary standard in processes exposed to risk. Of much importance and more general interest, however, is the trend of mortality rates from these diseases with or without tuberculosis, which does, when comparison is made after a number of years, reflect the effectiveness of the preventive measures installed. For this purpose, comparison of the figures of two successive years is of no value and sometimes misleading.

Table I shows deaths attributed to silicosis (with or without tuberculosis) during the last four years, together with the industries concerned. In factory cases, the cause of death has been borne out by departmental investigations. The variety of industries will be noted, and if space permitted to give in detail the precise occupational history in each case, the very widespread nature of the risk would be still more evident. For example, refractories' industries include amongst the 52 fatalities three whose occupation was that of repairing the silica brick linings of steel furnaces or converters, one a pot maker in a glass works, one an engine driver in a silica brick works, and one a grinder of refractory bricks, the remainder being associated with processes in the getting, crushing, grinding and manufacturing of refractory materials.

The table also shows the fatalities from asbestosis and asbestosis with tuberculosis coming to the Inspector's knowledge and investigated during the years in question. The figures given under "Other Cases of Fibrosis" are the number of cases in which "Fibrosis of the Lungs" was entered on the death certificates as the primary cause of death, these figures being supplied by the courtesy of the Registrar General. It has not been possible to investigate all the deaths from fibrosis of the lung; but in the majority the occupation of the deceased appeared not to have involved risk from silica or asbestos, and it is well recognised that fibrosis may be due to causes other than the inhalation of dust. These returns are indispensable since, from time to time, they provide the starting point of further investigation which provides valuable information.

TABLE I.—*Deaths from Fibrosis of the Lung, including Silicosis and Asbestosis (England and Wales), 1935-38.*

Disease and industry.	1935.	1936.	1937.	1938.
Silicosis :—				
Refractories industries	12	9	19	12
Pottery, manufacture of	50	47	64	46
Sandstone quarrying and dressing	40	42	19	27
Sandstone masons	61	56	74	50
Metal grinding	23	24	12	23
Sand blasting	14	10	13	7
Steel dressing and cleaning of castings	1	6	3	3
Stone, pebble, flint, and sand crushing	1	1	4	6
Scouring powders, manufacture of	1	2	—	1
Enamel and glass makers	—	1	—	—
Compo mixer (Tripoli)	1	—	—	—
Abrasive-wheel manufacture	1	—	—	—
Millstone dressing	—	—	1	1
Slate dressing with sand	—	—	—	1
Electrician on steel foundry	—	—	—	1
Slate quarrying and dressing	3	6	10	7
Granite quarrying and dressing	1	—	1	1
Tunnel mining (sewage works, etc.)	2	—	2	2
Grave digging	1	—	—	—
Coal mining	111	120	127	177
Gold mining (South Africa)	10	4	16	12
Tin mining	23	14	20	20
Iron ore (fine mottle) mining	12	8	16	13
Lead mining	2	1	1	1
Copper mining	—	—	1	1
Barytes mining	2	1	1	2
Mining engineers	—	1	2	2
Total : silicosis	392	365	416	420
Asbestosis	13	11	13	9
Other cases of fibrosis	538	544	534	503
Grand total	942	920	963	942

Silicosis and Asbestosis and Cancer of the Lung

In recent years the suggestion has been made in medical publications that there is some relationship between silicosis or asbestosis and cancer of the lung. The question is a difficult one, and the figures available are not yet large enough for it to be safe to draw conclusions from them. In exploring this question the field to be covered is wider than appears at first sight. Major points which will have to be settled are: Does silica or asbestos or the fibrosis of the lung they produce tend to inhibit cancer of the lung or to produce it? If the latter, do either of these two substances act as specific carcinogenic agents like tar, or is it that the disease they produce only prepares the soil for the occurrence of cancer? The following information has been collected from the Inspector's records. No case has been included in which a post-mortem examination has not been made.

Among 943 fatal cases in which silicosis or silicosis with tuberculosis was present, cancer of the lung was associated in 23 cases (2.6 per cent.). Of these 23, in 17 it was considered that the cancer was the primary cause of death, and in 6, silicosis or silicosis with tuberculosis. Among 347 deaths which were investigated during the same period owing to the possibility of silicosis being present, post-mortem examination revealed no silicosis but cancer of the lung was found in 17 cases (4.9 per cent.).

Similarly with asbestosis, among 103 fatal cases in which asbestosis or asbestosis with tuberculosis was present, cancer of the lung was associated in 12 cases (11.6 per cent.). Of these 12, in 6 it was considered that the cancer was the primary cause of death, and in 6, asbestosis or asbestosis with tuberculosis.

It is clear from these incomplete figures that extended inquiry and collection of data and evaluation of age incidence and age distribution are necessary before any conclusions can be arrived at.

The importance of the most careful post-mortem examination is emphasised by a recent case in which naked-eye examination revealed only advanced asbestosis, but what appeared to be a small blood clot in a bronchus turned out on microscopical examination to be a carcinoma, and also another case in which silicosis, tuberculosis and primary carcinoma were all present in the lungs.

Substitution of Siliceous by Non-Siliceous Materials

In processes in which the risk of silicosis is serious and uncontrollable or only partially controllable, the prevention of silicosis lies either in substitution by harmless materials or as the last resort in prohibition of the process. have been made therefore to find and int efficient substitutes for siliceous materials tain processes. Progress has been made direction in the following processes—in w a considerable extent; in others it is cont technical experiments in the processes Alumina for flint in china bedding; (2) a for siliceous material in parting powder non-siliceous abrasives for sand or flint in ing; (4) non-siliceous material for him sand in the dry casting of steel; and (5 siliceous material for silica bricks for limin and other furnaces.

* H.M. Stationery Office, York House, Kingsway, London, W.C.2. (Price 1s. net.)

Hydraulic Stowing in Indic

(*Concluded from page 333.*)

livers sealing water to the suction gland sand pump. Sealing water may be led storage at the colliery itself when the riv flood as the river water then contains an ciable quantity of sand; but in dry periods year, when a large basin has been cre clean sealing water is obtained direct fr river. The suction pipe is submerged attached by a special flange to the pump raised or lowered by a winch mounted pontoon. By this arrangement flexible ho is not needed on the suction side, an quantity of sand to water is regulated e

From the delivery side of the pump th sand-water mixture is conveyed by means armoured flexible hose pipe to a line of steel pipes in 18-ft. to 20-ft. lengths fitte Victaulic joints; the pipe range is carr date in the stream. The pipeline delive sand-water mixture into a conical separ the rate of 8 tons per min. The same mixture is fed on to screen plates with holes; the velocity of the incoming mix is reduced that sand is deposited in the of the separator and fairly clear water through the sluices at the sides of the sep The sand contains too much water, howe be delivered direct to the belt conveyo the sand is further dewatered on a drain

Sand-Drying Device

The sand-water mixture—by weight ap mately 4 parts of sand to 1 of water—from the bottom of the separator thro control valve over a distributing plate o 4 ft. wide drying belt. The drying belt c of a series of shallow trays, 2 in. deep, on rollers; each tray contains a mixt graded slag which is kept in place by ½ in iron plates perforated with ½ in. holes. belt is driven at a speed of 20 ft. per Three rollers or squeezers are placed equidi along the upper belt surface, and a se prongs placed in series along the belt to sand over as it is carried forward. The and prongs help in the dewatering of the whilst the slag in the trays allows water but not the sand.

The sand issuing from the drying be tains by volume approximately 10 per c water. The sand is delivered from the the drying belt direct on to a 26-in. tro belt conveyor which delivers it into the bunker. The belt conveyor elevates th 43 ft. in 130 ft. run to the top of the b on account of this grade only fairly dr can be carried.

The sand is delivered by the belt conveyo a specially constructed bunker holding ap mately 1,000 tons. On the underside bunker runs the single rail around whi sand buckets of the aerial ropeway ge after being delivered from the rope. O side there are 7 shutes which deliver t the buckets; the regulator at the bottom shute controlling the sand outflow to the is of a simple type, and is kept in plac balance weight. A one-ton capacity ba filled in 3 secs. through one of the shute

Luxemburg : Iron and Steel Output 1939

Pig-iron output in Luxemburg in July 194,848 metric tons (183,563 tons in June 1 117,264 tons in July, 1938). Steel product 177,645 tons in July, against 137,161 tons i and 110,293 tons in July, 1938. A total of ovens were in blast on July 31.

This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain ... news items, reports, digests, ...or presentations, together with ...tors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine

Reg. U. S. Pat. Off.

The ... will exercise care in checking on the accuracy of data printed, but in all other respects articles and opinions of which expression is allowed are the opinions of their authors—the editors reserving in all cases the right to comment on the same, in the current or any subsequent issues, as they may be inclined.

The Journal of Occupational Diseases and Traumatic Surgery

With which are consolidated "The Industrial Doctor" and "International Journal of Medicine and Surgery."

The Science, the Law and the Economics of Industrial Health

Volume 9 FEBRUARY, 1940 Number 2

General Motors Corporation Medical Conference

—Dayton, Ohio, November 2 and 3, 1939—

THE recent MEDICAL CONFERENCE OF GENERAL MOTORS PHYSICIANS, held at the Biltmore Hotel, Dayton, Ohio, November 2 and 3, 1939, comprised five meetings and 18 presentations.

The Program was as follows:

Thursday, November 2:

MORNING SESSION: Chairman, M. M. SHAFER, M.D., Medical Director, Frigidaire Division, Dayton.

2. "Recent Developments in Relation to Silicosis," U. GARDNER, M.D., Saranac Lake, New York.

A Plan for the Control of Tuberculosis in Industry," IAN BURNELL, M.D., Medical Director, AC Spark Plug ...ivision, Flint, Michigan.

3. "X-Ray in Industry," M. WILLIAM CLIFT, M.D., ...urley Hospital, Flint, Michigan.

4. "Preliminary Report on Hygiene Studies of Welding," L. B. CASE and V. J. CASTROP, Industrial Hygiene ...aboratory, General Motors, Detroit.

AFTERNOON SESSION: Chairman, A. L. BROOKS, M.D., Medical Director Fisher Body Division, Detroit.

5. "Recent Developments in the Occupational Derma...ses," MARION B. SULZBERGER, M.D., New York City.

6. "The Course of Disabling Morbidity among Indus...ial Workers, 1921-1938," WILLIAM M. GAFAFER, D.Sc., ...S. Public Health Service, Washington, D. C.

7. "The Industrial Nurse," HAROLD M. JAMES, M.D., ...edical Director, Delco Products Division, Dayton.

8. "The Doctor's Part in the Personnel Program," ...ORGE A. COBURN, Personnel Director, Delco-Remy Divi...on, Anderson, Indiana.

EVENING SESSION: Chairman, WALTER M. SIMPSON, ... M. D., Director, Kettering Institute for Medical Re...arch, The Miami Valley Hospital, Dayton.

9. "Relationships of Industrial Medicine to Private ...actice," STANLEY J. SLEGER, M.D., Chairman Council on ...dustrial Health, American Medical Association.

10. "Unfinished Business," C. F. KETTERING, Director ...eneral Motors Research Laboratories, Detroit.

Friday, November 3:

MORNING SESSION: Chairman, G. L. BIRD, M.D., Medi...al Director General Motors of Canada, Ltd., Osha...a, Ontario, Canada.

11. "A Practical Approach to Supervision of Mental ...alth in Industry," F. S. PARNEY, M.D., Chief, Division of ...dustrial Hygiene, Dept. of Pensions and National ...alth, Ottawa, Ontario, Canada.

12. "An Example of Industry's Participation in the ...ti-Syphilis Campaign," P. J. OCHSNER, M.D., Medical Director, Fisher Body Lansing Division, Lansing, Michigan.

13. "Study of the Factors of Employability, Especially Disabilities and Infirmities of the Elderly, and Problems Arising from Employment of the Same," GEORGE A. PAUL, M.D., Medical Director, Hyatt Bearings Division, Harrison, New Jersey.

14. "Early Diagnosis of Acute Appendicitis," C. RUSH MCADAMS, M.D., Medical Director, Fisher Body St. Louis Division, St. Louis, Missouri.

AFTERNOON SESSION: Chairman, F. B. WISHARD, M.D., Medical Director, Delco-Remy Division, Anderson, Indiana.

15. "Relationship of the Length of the Inguinal Ligament to the Occurrence of Inguinal Hernia," C. M. HILLENBRAND, M.D., Medical Director, Harrison Radiator Division, Lockport, New York.

16. "Hernia in Industry; Review of the Cases During the Past Ten Years," R. L. JOHNSTON, M.D., Medical Director, Inland Mfg. Division, Dayton.

17. "Antiseptics and Their Action," R. M. FREEMAN, M.D., Medical Director, Linden Division, Linden, New Jersey.

18. "Management of Fractures of the Spine," R. W. POSORSKY, M.D., Medical Director, Electro-Motive Division, La Grange, Illinois.

Except for DR. SULZBERGER'S address on "Recent Developments in the Occupational Dermatoses" all of the papers given at this Conference are included here in full text, in the order of their places on the Program:

Recent Developments in Relation to Silicosis

LEROY U. GARDNER, M.D.,

Director, The Saranac Laboratory for the Study of Tuberculosis,

Saranac Lake, New York

WHEN your Chairman asked me to speak of new developments in the field of silicosis I was perplexed because all but a few of the pathological, clinical and diagnostic aspects of this disease are now well established. I realized

that you had become better acquainted than I with the practical problems of diagnosis because you were dealing with them every day. After some deliberation I decided to tell you of some of the experimental work in progress at the Saranac Laboratory and to permit myself to speculate as to its significance. These experiments are incomplete and no conclusions are possible now, but perhaps they may ultimately lead to a better understanding of the action of irritant dusts upon the lungs.

As far as I have been able to discover, it is still true that only certain forms of free silica and the group of fibrous silicates known as asbestos, are capable of producing fibrosis in a normal lung. Many other silicates have been incriminated by roentgenologists, but when autopsy specimens have been examined it is discovered that there is associated chronic infection or that the inhaled dust contains significant quantities of free silica.

I shall first compare the action of chrysotile asbestos with that of quartz upon experimental animals and review considerable evidence bearing on their respective modes of action. A few of these observations now have practical significance; others may prove to be quite unrelated to the pneumoconioses found in human beings.

FOR the last year or two we have been coming to the conclusion that inhaled *asbestos* fibres are irritating not because they are *silicates* but because they are stiff fibres which mechanically irritate the lungs. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body; only those in the lungs are affected. It was inferred that these organs were affected because the movements of respiration are so much more rapid and continuous than those of other viscera. Then it was discovered that if asbestos was ground very finely so that few of the fibres were longer than 2μ in length the irritating property of the asbestos was practically destroyed. Inhalation experiments with such fine chrysotile asbestos have now been continued for three years. No fibrosis has developed in spite of the fact that an average atmospheric concentration of 125 million particles per cubic foot of air has been maintained. In contrast I would point out that in a previously reported experiment[1] one third this concentration of long fibre asbestos dust produced well marked fibrosis after about two years.

If the effect of asbestos were chemical, one would expect that a decrease in size would accelerate tissue response. With free silica large particles have little effect, but as their size decreases cellular reaction becomes more vigorous and even constitutional symptoms may ensue.[2] With fibrous asbestos the reverse is true.

The histology of early asbestosis does not suggest a chemical injury. Even under the most extreme conditions that can be created by artificial injection there is no preliminary phase of tissue necrosis with infiltration of leucocytes as occurs with high concentrations of very fine quartz. The connective tissue cells merely multiply very slowly in areas where the asbestos fibres are caught in the bronchioles. As collagen forms and contracts, the air spaces are obliterated by scar tissue. In experimental animals, at least, this change is not a progressive one after cessation of exposure to the dust as is the case in the response to quartz. Perhaps the reason is the deposition of the peculiar iron-containing coating on the surface of the inhaled fibres giving rise to the characteristic "asbestosis bodies." Dr. Timothy Leary considers[3] that the resultant smooth rounded ends of these bodies are no longer capable of scratching the delicate surfaces of the air spaces.

Finally it is most suggestive that among dozens of different silicate minerals only the five known as asbestos, which are unique because they are fibrous in structure, should be commonly recognized as pulmonary irritants. The variation in chemical composition within this group is greater than that between them and many other silicates. In fact chrysotile asbestos has the same chemical formula as a non-fibrous silicate, serpentine, which is physiologically inert. Obviously irritation would seem to be associated with the physical, rather than the chemical, composition of these minerals.

One point of practical significance may be indicated by these observations, namely, that very finely ground asbestos is not dangerous. This conclusion has support in clinical observation, for it has long been known that at the Thetford mills there was no clinical asbestosis even though in former years the atmosphere was very dusty and the dust was extremely fine. The fact that fabrication of fibres of the same mineral in American plants could produce disease was one of the puzzling features of this disease. But these experiments offer a plausible explanation. The fine dust in the mills is composed of serpentine and extremely short chrysotile fibres; that in the spinning and weaving mills contains many more long fibres.

IN THE case of *free silica* everything points to a chemical form of irritation although the solubility hypothesis as now conceived is not compatible with all the observations that have been made. In experimental animals inhaling or injected with excessive quantities of pure quartz or flint in exceedingly fine state of subdivision, it can be observed that tissue reaction takes place in two stages. First there is an acute inflammation resulting in necrosis. Following it there is healing with fibrosis in the form of a granuloma whose end product is the silicotic nodule. The collagen laid down by the connective tissue cells is modified in a peculiar way analagous to that in the tanning of leather, and this is responsible for the characteristic hyalinization of silicotic fibrosis. Incidentally, leather has been tanned with silica and sections of such leather reveal that the collagen has undergone a similar hyaline transformation. In human subjects only cases of rapidly developing silicosis show evidence of a two-stage reaction. In those with ordinary chronic disease, degenerative changes are slight and overshadowed by the formation of connective tissue.

The solubility hypothesis is based solely upon indirect evidence. It is known that colloidal silica is toxic and that quartz will dissolve to some extent in slightly alkaline fluids of the same pH as those of the body. But it has never been possible to prove that silica has dissolved within the body, because the soluble material is immediately combined with local tissue elements. It is now generally accepted that the soluble silica detected in the urine and blood probably comes from ingested food and drink rather than from inhaled dust.

WE have made a good many experiments whose results are difficult to reconcile with the solubility hypothesis. They do not necessarily invalidate it because we may not have all the facts and do not know how to interpret them. For example, we have found that cellular reactions begin within 15 to 30 minutes after injecting fine particulate silica. In the test-tube traces of dissolved silica are only discoverable by an exceedingly sensitive colorimetric reaction after 12 to 24 hours. Possibly "vital" factors may accelerate the process, but no means of evaluating them has yet been discovered. No one has ever produced fibrosis by single or repeated injections of the various forms of soluble silica. The effect is apparently not due to the quantity of silica dissolved, for many of the inert silicates in vitro are more soluble than quartz or flint. Evidences of alteration of the surface of quartz particles long in contact with living tissue have thus far escaped detection.

In years of experimenting to discover the mechanisms by which free silica exerts its peculiar effects upon living tissues we have repeatedly observed evidences of a specific toxicity. Guinea pigs, injected intraperitoneally with large doses of fine quartz particles are often obviously sick for some hours. When Mr. Cummings and Mr. Redlin[2] were seeking an avenue for the quantitative introduction of silica they tried intravenous injections into rabbits and discovered the method which we now use as a standard for comparing reactions to different dusts. But incidentally they found that these animals would die very promptly if the unit dose of quartz particles were too large. These deaths were not embolic because they did not occur with large particles but only when the quartz grains were 3μ or less in diameter, and they did not develop on injecting suspensions of other minerals ground to the same size.

Mr. Dworski and Mr. Delahant have extended these early observations in a long series of experiments that have yielded interesting though puzzling information. They have shown that fine silica injected into the left side of a guinea pig's heart acts like a strong poison.

Symptoms of shock followed by death within a few minutes to one hour occur when a 400-gram guinea pig receives an intracardiac injection of 3 or 4 cc. of a 1% suspension of 1 to 3μ quartz particles in physiological salt solution. The reaction is specific in that it is not produced by quartz particles of larger size and it has not been observed on injecting suspensions of several other minerals ground to the same particle size.

Symptoms are initiated even before completing the injection by the onset of rapid, labored respiration. The skin of the face and extremities becomes pale. The animal displays uncoordinated muscular movements and sporadic convulsions terminating in death if the dose is sufficient. When smaller doses are administered complete recovery takes place.

Autopsy of fatal cases discloses an intense engorgement of the spleen, omentum and subperitoneal tissues which are deep purple-red in color. The liver, and in fact practically all the remainder of the body, is almost bloodless. Counts of ear-vein blood show only about half the number of leucocytes present just before the injection and a corresponding reduction in red blood cells. The blood no longer clots. There is no hemolysis.

Animals that have recovered from one reaction seem to acquire a tolerance so that in some instances at least twice the former dose may be injected without symptoms. The degree of tolerance, however, varies widely in different individuals.

These symptoms are not influenced by the injection of adrenalin. They are not reproduced by transfusing the blood of an animal dying in shock into another one of the same species. They are only exhibited on injecting suspensions of quartz particles less than 3μ in diameter. They are not produced by injecting the supernatant salt solution from which the quartz particles have been removed by centrifugation. The severity of the reaction does not vary with the age of the particle suspension, although the amount of soluble silica increases on standing.

The toxic effects of quartz can be more or less effectively neutralized by adding small amounts of different substances. Colloidal aluminum hydroxide gives complete protection in a dilution of 0.03%; colloidal ferric hydroxide is much less effective. Animal charcoal cannot be injected, because it flocculates with the quartz and produces emboli. The finely ground metallic aluminum, used by Denny, Robson and Irwin[4] in the prevention of silicosis has no influence upon acute intoxication. Further experiments are in progress to verify the conclusion of these authors that aluminum coats the surface of the quartz particles with insoluble material.*

Recent observations indicate that all species of animals are not equally susceptible to intravenous injections of quartz. Dogs, which develop silicotic fibrosis very slowly and under conditions not yet defined, tolerate intravenous injections of comparatively large doses of fine quartz without symptoms of any kind. Rabbits, guinea pigs and white rats all die of shock.

Soluble colloidal silica in dispersed phase is also

* Subsequent experiments have shown that sterilizing suspensions of metallic aluminum by heat destroys their power of neutralizing the action of silica. Unheated suspensions of the metal are just as effective as the hydroxide.

toxic, and intravenous injections produce the reactions just described with extremely small doses. With larger ones death occurs promptly with symptoms referable to the respiratory tract. The lesions are then confined to over-distention of the lungs and subpleural petechial hemorrhages.

THE results produced by injecting particulate silica into the circulation all suggest a relationship to a large amount of mineral surface suddenly brought into contact with the blood or tissues. This is borne out both by the absence of reaction to large particles in excess of 3μ in diameter and possibly by the observations on neutralization. Any effect due to preformed soluble silica liberated in the suspension before injection has been eliminated, and no active soluble silica could be demonstrated in the blood of an animal dying in shock by transfusion into a second host. Everything now suggests an effect taking place very rapidly at the interface between the quartz and some element in the blood or tissues. Whether this is due to the very rapid production of colloidal silica in this location or to unique electrical properties of quartz is still a fascinating subject for speculation. In the same category is the demonstration of the tolerance conferred by previous injections.

The manifestation of acute toxic symptoms by those species of animals which develop silicotic fibrosis most readily and their absence in the dog, which appears to be much more resistant, suggests that these immediate reactions may have some bearing on the etiology of silicosis. Until more is learned about the reactions in the dog no inference is possible.

The influence of particle size is just as significant in the production of advanced silicotic fibrosis as it is in eliciting acute symptoms of intoxication. Many injection experiments have convinced us that quartz particles over 3μ in diameter are of little or no physiological significance. The fact that the lungs of persons exposed to most dusts contain few particles larger than 10μ in diameter has been responsible for the establishment of this size as the maximum important in dust counting. As a matter of fact we know that with some dusts there may be many larger ones. While it would be just as well that any large fragments should be excluded from the lungs, it is felt that only ones 3μ or less in diameter are responsible for causing silicotic fibrosis.

We have long maintained that particles of this size are irritating because they present a sufficient surface of silica to the fluids. Recently Mr. Dworski decided to test this reasoning further by injecting graded doses of different sized quartz particles which would all present the same total surface area. He found, however, that regardless of the total surface, silicotic reaction only developed when the size of the particles was 3μ or less. On examining his material it was discovered that with particles 4 to 5μ in diameter or larger there were only two to four visible in a thin section of each phagocyte. These cells appeared to be quite uninfluenced by the ingested material. In the case of particles 3μ or under the number inside each phagocyte was countless, and there was marked evidence of degeneration of the cytoplasm of these cells. In other words, the effect of surface area had to be exerted inside of the cell and not upon the body fluids as a whole. Extracellular quartz seemed to have little influence, and even intracellular particles of a large size produced no visible evidence of irritation.

THIS observation brings us back to the cause of these progressive changes. Apparently they are initiated with degeneration in the primary phagocytes, an effect which is exactly paralleled in the histogenesis of tuberculosis. Some years ago I published a paper calling attention to the similarities between silicosis and tuberculosis.[3] In both of these diseases the primary irritants, quartz grains or tubercule bacilli, as the case may be, intoxicate the phagocytes. In tuberculosis the cells thus poisoned are commonly designated as "epithelioid cells;" in silicosis the morphological effect is similar. Visible fat droplets accumulate in the cytoplasm and the elements stainable by supravital dyes undergo rearrangements in characteristic patterns. Giant cells of the Langhan's type are produced in each disease by stimulation and repeated multiplication of the nucleus. In each disease many cells die and their degenerated products are liberated in the tissue spaces. Incidentally it is the pabulum of necrotic cells in silicotic lesions on which tubercle thrive so well, an effect which is responsible for the high frequency of complicating tuberculous infection in silicotics. In the tubercle it is in the necrotic or caseous tissue that tubercle bacilli are most frequent. Perhaps they not only cause such caseation but they themselves thrive on it. Coincidentally more cells migrate to the area and proliferate to form the granulomatous nodule that is characteristic of each disease.

Finally the production of necrotic tissue, high in lipoids, by two very dissimilar primary irritants recalls the experiments of Fallon[4] and his hypothesis that it is these lipoids which are directly responsible for the formation of the granuloma. He showed that the injection of such lipoids, freed at least partially of contaminating silica particles, was capable of producing a granulomatous nodule.

Such mechanisms may be involved in the production of both silicotic nodules and tubercles. It still remains to demonstrate the nature of the primary injury by each of these irritants, and it is this problem that will probably engage our attention for some time to come.

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A Plan for the Control of Tuberculosis in Industry

Max R. Burnell, M.D.,
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AC Spark Plug Division, General Motors Corp.,
Flint, Michigan

IT IS a proud day when it can be stated that one of the safest places to be in this country is at work. Statistics from the National Safety Council seem to bear this out. The reduction in the frequency and severity accident rates in our industrial institutions has been nothing short of remarkable. Prevention has been the watchword.

With such improvements, the consideration of ıe medical departments in industry has more recently been drawn to the general health of its employees. Attention to the control of tuberculosis has been a very major issue. We believe that in no adult group throughout the country can so much be accomplished in prevention and early recognition of this disease as in our industrial institutions.

Legislation varies in the different States regarding occupational disease but the ultimate objective of this legislation is clear: The general health of the employee must increasingly become the responsibility of the employer. While the status of tuberculosis has been debated by many legislative bodies, silico-tuberculosis is definitely compensable. In the state of Illinois the new occupational disease disability compensation law went into effect on October 1, 1936. Dr. C. O. Sappington states that "during the first year of the operation of this law, 10.9% of disability claims filed were for pulmonary tuberculosis while silicosis claims were 15% of the total."[1]

The corporation that is looking toward the future with the best interests of its employees in mind certainly should be actively engaged in the problem of the control of tuberculosis. Many of our industrial institutions have already shown, by ıarked reduction of the incidence of pulmonary berculosis, just how this can be accomplished. Our own corporation has a very definite program concerning tuberculosis.

THE PROGRAM: 1. The Pre-Employment Physical Examination. Every prospective employee is given a thorough physical examination. This is done with the intent of placing that individual in industry where he or she is best physically as well as mentally fitted. This examination is not done for the purpose of either passing or rejecting that person for employment. This pre-employment examination gives the examining physician the best possible opportunity to detect pulmonary tuberculosis.

Dr. Robinson Bosworth, President of the Illinois Tuberculosis Association, states:[2] "85% of adult pulmonary tuberculosis has its beginning in the age group 18 to 28. In the vast majority of cases there are no symptoms and no physical signs."

It is exactly this age group that concerns us most in our pre-employment physical examination, for it is during this age period that most employees first enter industry. We therefore feel that such an examination is not complete without an x-ray of the chest.

We believe, with Dr. Andrew R. Riddell, of the Industrial Hygiene Department, Ontario Department of Health, that the wholesale tuberculin testing in industry has many disadvantages. He states that[3] "the individuals must present themselves at least three times—there is considerable disruption of work—even after most careful explanation as to the meaning of the test, the employees who react positively are greatly disturbed."

Applicants in whom active pulmonary tuberculosis is discovered are sent to their family physician. Our roentgenogram is at his disposal, but must be returned to our files so that we can later compare the progress of the disease and determine at what future time those persons may be pronounced employable.

Applicants with arrested or quiescent tuberculosis are placed at work after being informed of their condition and instructed to return for periodic examinations. These examinations for new employees, in this classification, are conducted every three months.

It has been increasingly evident that our responsibility does not end there. We explain to the employee that while he now has an arrested tuberculosis, it was once active. We encourage him to have the members of his family examined by the family physician. We feel that not until then has industry wholly discharged its duty to the community.

2. Periodic Examinations for Old Employees. These examinations are made at different times and for varied reasons:

(a) Upon returning to work after vacations.
(b) After absence caused by an illness.
(c) Re-employment following seasonal lay-off.
(d) Transfer from one type of work to another.
(e) Medical department requesting the examination for some definite reason.
(f) Volunteer requests by the employee for such an examination.

Little comment is necessary concerning these examinations except to state that every employee comes into our medical department for a general physical check up at least once each year.

Two types of periodic examinations deserve

special attention. First, the medical department requesting the employee to appear for some specific reason. Each employee has his own physical record card. His visits to the medical department are recorded here. If it is noticed that frequent visits are being made for colds, bronchitis, loss of weight, feelings of fatigue, etc., he is requested to appear so that we may get at the underlying cause. These examinations are always accompanied by x-rays of the chest and if the disability is from early tuberculosis, we are able to detect it.

The other type of periodic examination is most gratifying to the medical man in industry — the physical check up requested by the employee himself. This is, indeed, a measure of the confidence that has been established between the medical department and the worker.

Cooperation with the family physician is of paramount importance. When some disability is uncovered and it is discussed with the employee's own physician, that employee realizes that the corporation is not attempting to deprive him of his job but is trying to keep him at work by improving his health. Again confidence is established, and that is the best foundation for a successful program to maintain a high standard of health within your organization.

One striking example of such a relationship is of unusual interest in regard to the tuberculosis problem. After the installation of excellent x-ray equipment in one of our plant hospitals, it was suggested to the employees that they take advantage of this opportunity to have their chests x-rayed. These were all old employees, some with service records of over 20 years. The response was amazing. Over 4600 employees requested the examination during the first 10 months.

All of these employees were working every day. They considered themselves in excellent health—so did the medical department as a matter of fact, as their previous physical examinations during the year had shown no disabilities. However, 12 cases of early active tuberculosis and 42 cases of arrested or quiescent tuberculosis were brought to light. The active cases are all under treatment —nine of them have been referred to sanatoria by the family physician. Active tuberculosis has also been found by the family physician in the households of those in the arrested groups.

3. Control of Tuberculosis Within the Plant. If there is to be real control of tuberculosis in industry, the activities of the industrial medical departments must not stop with examinations within the plant hospital.

Dr. G. T. Drolet[4] has shown conclusively that where there are bona fide cases of active tuberculosis the results of treatment, irrespective of where or how, are still far from satisfactory. In other words, "the hopeful sign is steady reduction in the *number* of cases and not what happens to those who actually develop the disease. Progress has come by prevention and not through treatment."[5]

We must take a page from the program of the plant safety engineer and again adopt "prevention" as our slogan.

As Dr. C. D. Selby has stated, the industrial physician is the health officer of the plant.[6] "It is his duty to locate, identify and determine the importance of all occupational and environmental sources of disease or health impairment and to see that they are corrected."

In relationship to tuberculosis he must know if "exposures" to the inhalation of dust particles or obnoxious gases and vapors are present within the plant. Also he must pay attention to overcrowding, proper lighting, ventilation, arranging rest periods for those engaged in strenuous or necessarily rapid operations, and have knowledge of the toxicity of materials handled.

Dr. Hamilton[7] has listed definite industrial poisons which are capable of irritating severely the bronchopulmonary system and activating a pre-existing pulmonary tuberculosis — chlorine gas, ammonia, benzine, brass fumes, phosgene, nitric acid fumes, selenium and vanadium, to mention a few.

The danger of exposure to the inhalation of dust, especially silica dioxide (crystalline silica), is only too well known. It is not within the province of this paper to discuss silicosis. However, as Dr. Riddell[8] has stated, "Tuberculosis is the commonest and most important complication in deaths among silicotics. Therefore in any discussion of the control of tuberculosis in industry, the subject of silico-tuberculosis must have its place."

Dr. LeRoy Gardner[9] contends that "silicosis is the menace that it is because it specifically predisposes to tuberculosis. It is the latter which is responsible for disability, lost time, and, in many cases, death, and it is really tuberculosis against which we have to fight."

This statement has been recently challenged by Dr. George Ornstein.[10]

Whatever the conclusions may be in the future concerning the exact relationship of silicosis to tuberculosis, the industrial physician must continue his attempt to eliminate dust.

Dust counts should be made to locate the points of greatest hazard. Consultations should be held with the safety engineer as to the best type of control, whether (a) mechanical ventilation of the positive or supply type or local exhaust systems; (b) isolation or total enclosure methods, or (c) respiratory protection by respirators and positive pressure masks or helmets, are to be advised.

THE purpose of industry is manufacturing. Often the installation of adequate equipment is expensive. This adds to the cost of the manufactured article. It behooves the industrial physician to study his problem with care. Our experience has proved that when management is informed of the hazards that are present and shown the necessary steps toward their elimination whole-hearted cooperation is immediately forthcoming.

To Summarize: A plan to control tuberculosis

in industry must start with pre-employment examination. The majority of employees first enter industry during the age period when adult pulmonary tuberculosis is most prevalent. X-ray of the chest is a requirement. if early cases are to be detected. When arrested or quiescent cases are discovered, we feel that it is industry's duty to the community to cooperate with the family physician in an attempt to uncover active cases in that employee's household.

Once the employee is at work. periodic examinations are the next requirement. Such an examination should be conducted at least annually. The employee quickly learns the personal value of these physical check ups. Confidence then established between the worker and the medical department goes far toward simplifying any plan for the control of tuberculosis in industry.

Lastly, the influence of the industrial medical department must extend to every department of the operating plant. Inspections must be made regularly. The industrial physician should be familiar with every possible "exposure" to health impairment. This is the final requirement of our program.

Our goal should be to make the employee's hours at work not only the safest from accident but also the freest from exposure to disease.

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X-Ray in Industry

M. William Clift, M.D., F.A.C.P.
Hurley Hospital,
Flint, Michigan

THE new attitude that has grown up between industrial physicians and the employees of this organization, stimulated by the consultant, has made it not only an interesting privilege but also a pleasure for an associate to be in contact with what we must feel are among the best ideals of the medical profession. Therefore. it seems to me that you might be interested in my experience of the past two years in the field of x-ray in industry.

That x-ray has had its definite function you all know. from the several years that industry has recognized its use. The contact of the roentgenologist with the industrial physician has been largely in the field of forensic medicine. With the new awakening of industry to the importance of preventive medicine has come a change in the qualifications of the roentgenologist and the physician. and a necessity for greater cooperation with and by the management of the organization. This has meant a study of the hazards of industry. That necessitates not only an intimate knowledge of those hazards but of those who may encounter them. Therefore, the roentgenologist must have a close contact with the factory physician, who in turn. must have contact with the individual employee and be conversant with the nature of his work.

Just how large a part the roentgenologist can contribute to this field of medicine depends upon two factors: *first*, the physical set-up of the department and, *second*, the cooperation received from the management.

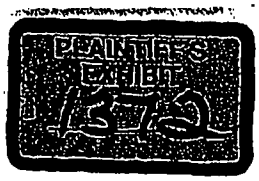
The extent of the equipment naturally depends upon the size of the plant and the burden that will be imposed upon the medical department. However, there are certain minimum requirements that can not be neglected if efficient service is to be rendered. The equipment and arrangement of the department must be such as will produce the results most economically.

In the past two years the author has had the opportunity to organize and try out, what in his own mind is a model industrial x-ray department, this because of a sympathetic management and the helpful cooperation of a medical department which was attuned to the requirements of modern industrial medicine.

It is impossible to do adequate work without efficient equipment. And it has been thoroughly demonstrated that in larger factories x-ray work can be done most economically within the plant itself. To refer cases to the private roentgenologist is both expensive and lacks the cohesive efficiency that can only be obtained within the organization.

The result of two years' experience has demonstrated that the initial cost of the x-ray equipment is an extremely small factor in the economics of a medical department. With the most modern equipment and with a well regulated plan of operation the cost per case is hardly an incident in factory medicine. My experience in the organization with which I am connected has shown that the entire capital investment for the x-ray equipment has not only been liquidated but has also shown a dividend in less than two years. However. the greatest dividend has been in the salvage of individuals working in factories and by appreciating their physical needs, placing them back in industry in positions they can adequately fulfill.

In the time that we have had the opportunity to examine the employees. we have seen upwards of 6000 cases. We have detected cases of early tuberculosis. placed them under the proper conditions for their rehabilitation. and had the pleasure



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CLINICAL STUDIES IN ASBESTOSIS[1]

MOSES J. STONE[2]

Among the newer diseases, which are a byproduct of our industrial age, asbestosis has come to occupy a fairly prominent position. The diagnosis of this form of pneumonoconiosis, its clinical course, the effect it produces on organs other than the lungs as well as its various complications and sequelae are still very much in the controversial stage. The marked development in the industrial use of asbestos has resulted in an increase in the number of people exposed to this occupational hazard. This, together with the greater interest developed by medical men and legislature boards in industrial disease, has produced a great impetus in the study of this subject. The literature on this subject is still rather meagre, and only recently has the hazardous nature of exposure to asbestos dust been recognized and studied.

Asbestos is a hydrated magnesium silicate, the composition of which varies with the sections from which it is mined. Most of the asbestos used in the United States is Canadian crysotile containing approximately 43 per cent magnesium, 13 per cent water and traces of iron and nickel. To be sure, asbestos is not a new mineral. Known to the Romans, who mined it from the Alps and from the more remote Urals, the mineral was mentioned in the writing of Herodotus and the second Pliny. Marco Polo spoke of its use by the Tartars.

Only within the last decade, has the subject of asbestosis really received the attention of the medical world. Prior to 1924, there is but one recorded case of disease due to the inhalation of asbestos dust. This was recorded in 1900 by Montague Murray in the Charing Cross Hospital Gazette. The first complete description of this disease entity appeared in 1927 when Cooke (1) and McDonald (2) reported 2 cases of asbestosis and discussed the histological changes found in the lung. Hoffman (3) was the first American to focus attention on the magnitude of the asbestosis problem in the United States. In 1930 Mills (4)

[1] Presented at a session of the Clinical Section at the 35th annual meeting of the National Tuberculosis Association, Boston, Massachusetts, June 28, 1939.

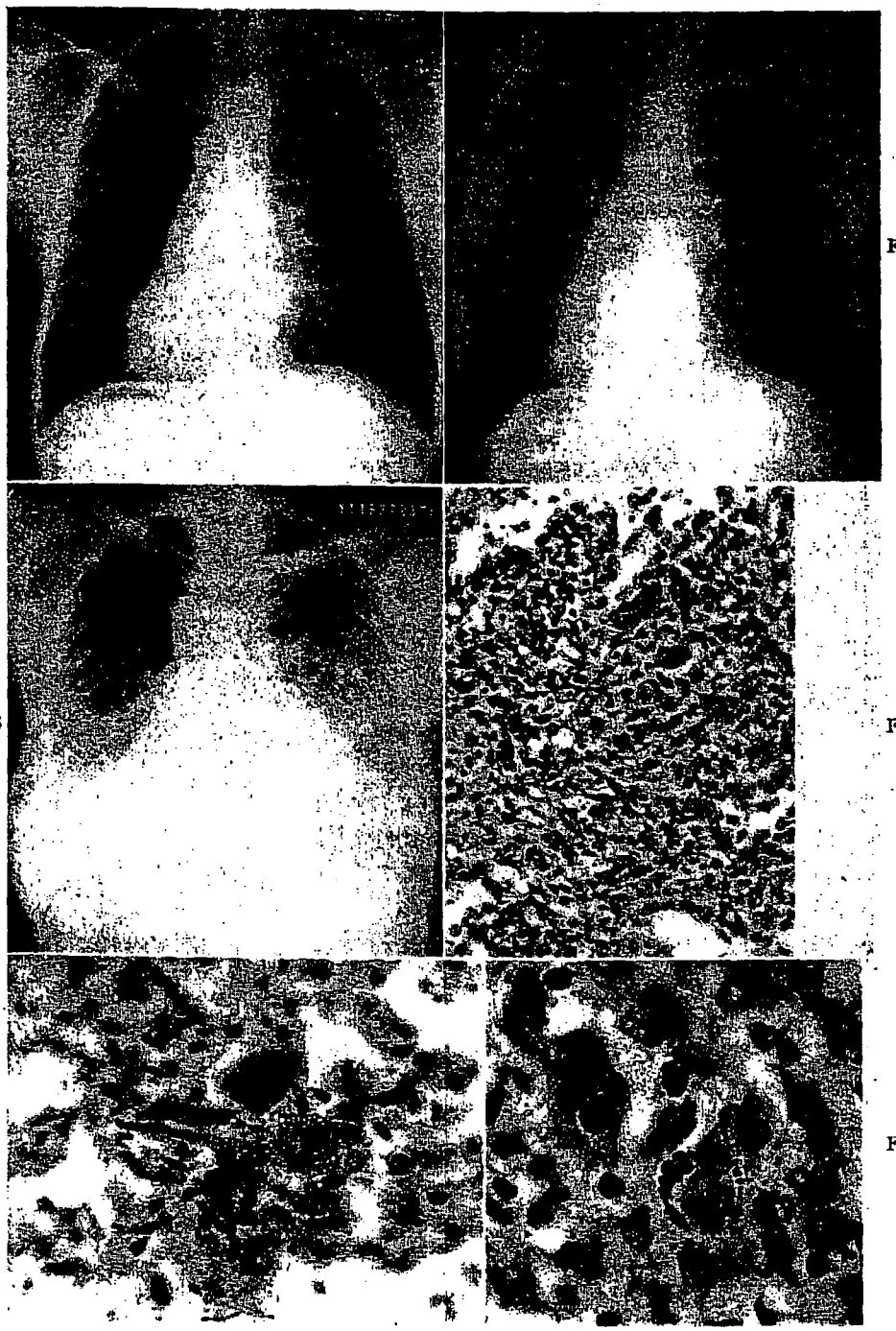
[2] 520 Beacon Street, Boston, Massachusetts.

offered the first pathological report published in the United States. In the same year, Lynch and Smith (5) reported their findings on asbestosis bodies.

An interesting feature of asbestosis is the finding of the asbestosis bodies in the lung as well as in the sputum. Gloyne and others described the asbestosis body as a core of asbestos fibre surrounded by iron containing deposits. It is golden yellow in color and does not stain with ordinary histological stains but becomes a brilliant blue when treated with potassium ferrocyanide. The bodies are slender, elongated, segmented structures with bulbous ends. The substance of the body appears homogeneous except for the centre linear thickening which is the asbestos fibre. The essential features of the pathological changes appear to be pleurisy, marked diffuse fibrosis with contraction of the lungs, and the presence of the asbestos fibres and the asbestosis bodies. Uniform thickening of the visceral pleura is found with varying degrees of pleural and pericardial thickening. The fibrosis extends into the apices with obliterating layers of fibrous tissue surrounding the bronchioles. The pulmonary endarteries are surrounded by fibrous tissue and may show thrombosis. The interlobar and intrapleural connective tissue is thickened. Numerous alveoli may be obliterated by fibrous tissue, others may be emphysematous, while still others are filled with leucocytes and alveolar cells. Occasional giant cells, not to be mistaken for tuberculous giant cells, are found. There are asbestosis bodies in the lumina of the bronchioles, in the alveoli and in the fibrous tissue. An amorphous brown pigment, often phagocytized by the alveolar cells, is scattered throughout the lung. On section the trabeculae stand out in the fibrous network and may present evidence of septic bronchitis, bronchiectasis, bronchopneumonia or lobar pneumonia as the terminal events. Lynch (6) and Gloyne (7, 8) note that the presence of asbestos fibres in the mouth and nose are indicative of exposure but not necessarily of disease. Essentially the presence of asbestosis bodies indicates tissue response to dust.

Since 1930 much has been written on this subject both from the clinical and public health points of view. Many controversial points, such as the action of asbestos dust on lung tissue, the relationship of asbestosis to infections, especially tuberculosis, and the effect it has on the circulatory system, etc., still remain that demand the attention of investigators.

This report is based on a study of 180 patients who were examined by me in collaboration with the late Dr. John B. Hawes, 2nd. The patients



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had been employed for three years or more in a factory manufacturing asbestos brake lining for automobiles and worked in the carding, spinning or weaving rooms. Many of them were engaged in the more dangerous work of crushing the crude asbestos. The great majority of them had been employed from five to fifteen years, and many had worked for more than twenty-five years. The cases came to our attention through disability claims and were given complete physical examinations during the years of 1936 and 1937. None were employed in asbestos factories at the time or since their medical examinations.

FIG. 1

CLASSIFICATION

Of the 180 patients, 32 were diagnosed as negative, although they claimed disability. X-ray film as well as physical signs failed to show any abnormalities that could be ascribed to asbestosis. The remaining 148 patients were classified as follows:

Stage I—78 patients
Stage II—54 patients
Stage III—16 patients

FIG. 2

A patient was classified as stage I when there was definite limitation of chest expansion (less than 2 inches) in addition to roentgenological evidence of increased lung markings. It must be admitted that frequently this interpretation must be somewhat arbitrary, as it is often quite difficult to draw the line between normal and exaggerated lung markings. Unlike silicosis the early fibrotic changes are very indefinite and only after considerable experience, careful standardized X-ray technique and proper history, can a diagnosis of early asbestosis be made, and even then not with any degree of certainty. In stage II were included those

FIG. 3

FIG. 1. Stage I. Patient worked for four years as an inspector in a plant manufacturing asbestos brake-lining. Two years prior to the examination he noticed lack of energy and strength, also some cough and expectoration. There was only slight dyspnoea on exertion.

FIG. 2. Stage II. Patient worked for thirteen years as an inspector in a plant manufacturing asbestos brake-lining. He has had dry cough for seven years which has continued on about the same; also dyspnoea on the slightest exertion.

FIG. 3. Stage III. Patient has been exposed to asbestos dust for twenty-five years. He has marked dyspnoea and has to sit up on a stool at side of bed and sleeps in that position. Unable to lie down. Chest expansion, one inch.

FIG. 4. Asbestosis bodies found in the lungs of patient who died of pulmonary tuberculosis. Note the various sizes and shapes of these bodies, as seen with the low power lens.

FIG. 5. Asbestosis body. High power magnification

FIG. 6. Another view of the asbestosis bodies (high power)

patients who had definite symptoms and whose X-ray films revealed definite evidence of pulmonary fibrosis. In stage III were included those patients who had both definite symptoms as well as roentgenological evidence of marked pulmonary involvement.

LENGTH OF EXPOSURE

Number of Cases	*Years of Exposure*
47	4 or less
56	4–9
40	9–14
25	14 years and over

RELATIONSHIP OF LENGTH OF EXPOSURE TO PATHOLOGICAL CHANGES IN THE LUNGS

Of the 47 patients who were exposed four years or less, 25 showed no definite pathological changes; 14 were classified as stage I and 8 as stage II. Fifty-six patients worked four to nine years. Of these, 36 cases were classified as stage I, 12 were considered as stage II or fairly marked asbestosis and 8 were found to be suffering from advanced or stage III asbestosis. Of the 40 patients who were exposed ten to fifteen years, 14 were classified as stage I, 20 as stage II and 6 as stage III. Twenty-five patients were exposed for over fifteen years. Of these, 9 showed the early pulmonary changes of stage I, 14 were classified as stage II and 2 as stage III. The average length of exposure of the stage I patients was eight years; stage II, ten years; stage III, eleven years. While there is but slight evidence that the degree of fibrosis is apt to increase with the length of exposure, there are other factors, such as intercurrent infections and other constitutional factors, that play a part in the development of fibrotic changes in the lung structures.

Since all patients were seeking compensation on account of disability, many of the complaints were undoubtedly exaggerated. On careful questioning, however, one could evaluate the symptoms fairly accurately. The outstanding symptom in all the patients was dyspnoea. Many also complained of tightness in the chest. Of the 54 patients in stage II, 32 complained of dyspnoea on slight exertion, tightness in the chest, cough and general fatigue. In addition to the above symptoms, 10 patients also complained of marked loss of weight. All of the 16 patients in stage III complained of dyspnoea, inability for any sustained effort, cough, expectoration, tightness in the chest, anorexia and loss of weight.

PHYSICAL EXAMINATION

The chest findings were mainly those of basal fibrosis with accompanying emphysema at the apices. On the whole, physical findings were quite meagre, and were not in proportion to the symptoms given by patients. The outstanding physical sign was diminution of chest expansion. Many cases showed evidence of dulness at the bases with hyperresonance near the apices. In the majority of cases auscultation disclosed prolongation of the expiratory phase with some high-pitched dry crackling râles. Moist râles were found only when there was definite evidence of associated bronchitis or bronchiectasis.

X-RAY EXAMINATION

X-ray examination constitutes the most important single procedure in the diagnosis of asbestosis. Those who have examined many cases of silicosis will find the X-ray interpretation of asbestosis extremely difficult. Early X-ray diagnosis of asbestosis is still in the realm of conjecture and should be undertaken only by those who have had the opportunity to examine many such cases.

In the early stages there is only a slight relative increase in density in the lower zones producing a filmy, hazy appearance of the bases. The shadows are much finer, lighter, and have a granular appearance, rather than the nodular or patchy type of infiltration found in silicosis. The apparently small amount of lung involvement in the early stages makes the X-ray evaluation rather difficult. As the disease progresses, signs indicative of stage II are seen. There is an increase in density in the lower lung zones, the diaphragm becomes indistinct in outline and shows, on roentgenoscopic examination, limitation of motion. The costophrenic angle is obliterated by thickened pleura. A fine lace-like network of fibrosis of interstitial or perivascular form rather than a definitely parenchymatous type is seen. The characteristic chest X-ray film shows granular or "ground-glass" appearance with more or less obliteration of the usual linear pulmonic markings. This is localized in most cases in the midlung region and bases. The bronchovascular markings are increased and often pericardial and pleural thickenings are noted. Many cases in our series showed elevation of the diaphragm. In stage III, because of the frequently associated bronchitis and bronchiectasis, the parenchymatous changes become more marked.

TUBERCULOSIS IN ASBESTOS WORKERS

The still debatable question of the relationship between asbestosis and tuberculosis has been discussed by numerous authors. This study was mainly undertaken to determine the incidence of tuberculosis among asbestosis workers. Since many patients do develop pulmonary fibrosis, their resistance to pulmonary infections is greatly decreased. Most of the English investigators, among them Stewart (9), found marked increase of tuberculosis infection among asbestos workers. He states that there can be no question that pulmonary asbestosis predisposes to tuberculous infection of the lungs. His opinion is also held by Ellman (10) who found an increasing incidence of tuberculosis in persons exposed to asbestos dust. Donnelly (11) refutes these above statements and finds no definite relationship between asbestosis and tuberculosis. In fact Merewether and Price (12), finding only 3 cases of tuberculosis among 374 cases of asbestosis, indicated that a lessened susceptibility to tuberculosis existed in such cases.

Out of 180 films taken in our series of cases, 9 showed evidence of parenchymatous tuberculous infection; two of this group had active tuberculosis and 7 showed inactive or healed lesions. In our series of 16 cases of advanced asbestosis, only one had active tuberculosis. One other active case of tuberculosis had only moderate asbestosis (stage II). Of those patients who had inactive disease, 5 had moderately advanced asbestosis (stage II), and all had been exposed to asbestos dust for more than ten years. Thus we are not impressed with tuberculosis as being a serious complication of asbestosis. We find that bronchitis, bronchiectasis and bronchopneumonia are more frequently associated with asbestosis than is tuberculosis. Indeed we feel that, because of the frequency of tuberculosis in silicosis, many patients with bronchitis or low grade bronchopneumonia were erroneously diagnosed as being tuberculous. Only one person in our total series has subsequently developed active pulmonary tuberculosis.

HEART STUDIES

One hundred and fifty patients were studied to ascertain the cardiac involvement, if any, in cases of asbestosis. Heart measurements were done by Dr. George Levene of the Massachusetts Memorial Hospitals. Ninety of the 150 patients studied, or 60 per cent, showed prominent pulmonary vessels. This prominence was apparently either due to en-

gorgement or perivascular fibrosis. The transverse diameter was increased in 50 cases, or 33 per cent, after excluding cases of organic heart disease, such as hypertension, coronary sclerosis and rheumatic heart disease. Thus it was felt that asbestosis was apparently the cause of the increased transverse diameter in 33 per cent of our cases. Thirteen cases, or 8 per cent of our total series, had right-sided hypertrophy. A study was then carried out to determine the heart findings in 56 cases classified as moderately advanced (stage II) and advanced (stage III) asbestosis. Of these 56 cases, 35, or 62 per cent, showed prominence of the pulmonary vessels; 25, or 44 per cent, showed an increase of the transverse diameter (7 cases of the latter group were excluded because of associated heart disease). In the remaining 18 patients, or 32 per cent, the enlargement of the transverse diameter was associated with asbestosis.

Apparently then the heart and the pericardium are affected in asbestosis much more so than in any other type of pneumonoconiosis. The cardiac outline is obscured by the superimposed increased fibrous tissue of the lung with advancing degrees of asbestosis. The pleuropericardium, the pleura of the lower lung fields and the diaphragmatic domes become thickened and the usually sharply demarcated heart contours are blurred. The marked emphysema and pulmonary fibrosis of stages II and III may cause rotation or lateral displacement of the heart. Unless roentgenoscopic examination is carried out, the sagittal roentgenograms alone will not convey a true impression of cardiac size or shape. In such cases lateral views together with roentgenoscopic examination are recommended to clarify the diagnosis. The roentgenographic appearance of any stage of asbestosis associated with heart disease may be greatly accentuated by passive congestion. As a result of our studies, roentgenoscopic examination is recommended to determine enlargement of the heart shadow and alterations in the cardiac silhouette. This is of special importance in cases of asbestosis associated with hypertension, mitral stenosis and cor pulmonale. It is our belief, therefore, that cardiac embarrassment is of utmost importance in the causation of early symptoms. It is evident that the dyspnoea noted early in this disease is of cardiac or circulatory rather than of pulmonary origin.

FOLLOW-UP STUDIES

We had an opportunity to reëxamine 13 patients of this group from two to three years after their first examination. Two of this group were

originally classified as stage III; 8, as stage II, and 3, as stage I. These patients were carefully studied in The Clinic for Cardiac Research of the Massachusetts Memorial Hospitals by Drs. George Levene, William Duncan Reid and Maurice A. Lesser. The vital capacity was affected in all cases, being from 50 to 75 per cent below the normal, calculated on the basis of height and weight. X-ray examination revealed definite progression of fibrosis in the two cases that were originally classified as stage III. Two patients had evidence of old coronary disease. This was corroborated both by the X-ray as well as electrocardiographic examination. The electrocardiograms of the remaining patients were entirely normal. Sedimentation rates were normal with the exception of the two with coronary disease which showed an increased rate.

Our impression gained from the study of this small group is that in advanced fibrosis due to asbestosis the disease will progress even after exposure ceases. This, however, is not the case in the earlier stages. We also feel that the heart is not affected unless pulmonary fibrosis is marked. In the light of the blood studies, fibrosis is the result of irritation due to asbestos fibres, and is not the result of infection.

Since the entire group was first examined, 18 patients have died. The cause of death was given as follows:

Pulmonary tuberculosis	3 cases
Bronchopneumonia	5 cases
Lobar pneumonia	2 cases
Carcinoma	2 cases
Cardiac disease	2 cases
Cerebral shock	1 case
Accidental	2 cases
Brain tumor	1 case

Most of the patients are still able to pursue a gainful occupation although unable to perform duties that demand much physical exertion. Seventeen patients are semi-invalids, most of them complaining of cough and marked dyspnoea.

CONCLUSIONS

1. Asbestosis, like silicosis, constitutes an occupational hazard, arising from exposure to asbestos dust.

2. Pathological findings are those of pulmonary fibrosis and thickened pleura, most marked at both bases.

3. Dyspnoea is an early symptom and is the chief cause of early disability.

4. X-ray findings are not characteristic in early stages. In advanced asbestosis X-ray reveals lace-like interstitial fibrosis and thickened pleura at the bases giving the "ground-glass" appearance.

5. Bronchial and bronchopulmonary infections are common complications.

6. Tuberculosis is not a frequent concomitant of asbestosis.

7. Advanced asbestosis not infrequently leads to right ventricular hypertrophy and failure.

8. When exposure ceases, fibrosis does not progress unless the degree of involvement is already marked.

9. All health laws relating to dusty occupations should be rigidly enforced in all asbestos factories.

10. Workers developing pulmonary fibrosis in the course of exposure to asbestos dust should be entitled to compensation.

I am deeply indebted to Drs. George Levene, William D. Reid, Maurice A. Lesser of the Clinic for Cardiac Research of the Massachusetts Memorial Hospitals and to Dr. Lois C. Miller of the Department of Radiology for their coöperation and valuable assistance given me in preparation of this paper.

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This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests, and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine

Reg. U. S. Pat. Off.

The editors will exercise care in checking on the accuracy of data printed, but in all other respects articles and opinions of which expression is allowed are the opinions of their authors—the editors reserving in all cases the right to comment on the same, in the current or any subsequent issues, as they may be inclined.

The Journal of Occupational Diseases and Traumatic Surgery

With which are consolidated "The Industrial Doctor" and "International Journal of Medicine and Surgery."

The Science, the Law and the Economics of Industrial Health

Volume 9 FEBRUARY, 1940 Number 2

General Motors Corporation Medical Conference

—Dayton, Ohio, November 2 and 3, 1939—

THE recent MEDICAL CONFERENCE OF GENERAL MOTORS PHYSICIANS, held at the Biltmore Hotel, Dayton, Ohio, November 2 and 3, 1939, comprised five meetings and 18 presentations.

The Program was as follows:

Thursday, November 2:

MORNING SESSION: Chairman, M. M. SHAFER, M.D., Medical Director, Frigidaire Division, Dayton.

1. "Recent Developments in Relation to Silicosis," LEROY U. GARDNER, M.D., Saranac Lake, New York.
2. "A Plan for the Control of Tuberculosis in Industry," MAX BURNELL, M.D., Medical Director, AC Spark Plug Division, Flint, Michigan.
3. "X-Ray in Industry," M. WILLIAM CLIFT, M.D., Hurley Hospital, Flint, Michigan.
4. "Preliminary Report on Hygiene Studies of Welding," L. B. CASE and V. J. CASTROP, Industrial Hygiene Laboratory, General Motors, Detroit.

AFTERNOON SESSION: Chairman, A. L. BROOKS, M.D., Medical Director Fisher Body Division, Detroit.

5. "Recent Developments in the Occupational Dermatoses," MARION B. SULZBERGER, M.D., New York City.
6. "The Course of Disabling Morbidity among Industrial Workers, 1921-1938," WILLIAM M. GAFAFER, D.Sc., U. S. Public Health Service, Washington, D. C.
7. "The Industrial Nurse," HAROLD M. JAMES, M.D., Medical Director, Delco Products Division, Dayton.
8. "The Doctor's Part in the Personnel Program," GEORGE A. COBURN, Personnel Director, Delco-Remy Division, Anderson, Indiana.

EVENING SESSION: Chairman, WALTER M. SIMPSON, M. D., Director, Kettering Institute for Medical Research, The Miami Valley Hospital, Dayton.

9. "Relationships of Industrial Medicine to Private Practice," STANLEY J. SEEGER, M.D., Chairman Council on Industrial Health, American Medical Association.
10. "Unfinished Business," C. F. KETTERING, Director General Motors Research Laboratories, Detroit.

Friday, November 3:

MORNING SESSION: Chairman, G. L. BIRD, M.D., Medical Director General Motors of Canada, Ltd., Oshawa, Ontario, Canada.

11. "A Practical Approach to Supervision of Mental Health in Industry," F. S. PARNEY, M.D., Chief, Division of Industrial Hygiene, Dept. of Pensions and National Health, Ottawa, Ontario, Canada.
12. "An Example of Industry's Participation in the Anti-Syphilis Campaign," P. J. OCHSNER, M.D., Medical Director, Fisher Body Lansing Division, Lansing, Michigan.
13. "Study of the Factors of Employability, Especially Disabilities and Infirmities of the Elderly, and Problems Arising from Employment of the Same," GEORGE A. PAUL, M.D., Medical Director, Hyatt Bearings Division, Harrison, New Jersey.
14. "Early Diagnosis of Acute Appendicitis," C. RUSH MCADAM, M.D., Medical Director, Fisher Body St. Louis Division, St. Louis, Missouri.

AFTERNOON SESSION: Chairman, F. B. WISHARD, M.D., Medical Director, Delco-Remy Division, Anderson, Indiana.

15. "Relationship of the Length of the Inguinal Ligament to the Occurrence of Inguinal Hernia," C. M. HILLENBRAND, M.D., Medical Director, Harrison Radiator Division, Lockport, New York.
16. "Hernia in Industry; Review of the Cases During the Past Ten Years," R. L. JOHNSTON, M.D., Medical Director, Inland Mfg. Division, Dayton.
17. "Antiseptics and Their Action," R. M. FREEMAN, M.D., Medical Director, Linden Division, Linden, New Jersey.
18. "Management of Fractures of the Spine," R. W. POBORSKY, M.D., Medical Director, Electro-Motive Division, La Grange, Illinois.

Except for DR. SULZBERGER's address on "Recent Developments in the Occupational Dermatoses" all of the papers given at this Conference are included here in full text, in the order of their places on the Program:

Recent Developments in Relation to Silicosis

LEROY U. GARDNER, M.D.,
Director, The Saranac Laboratory for the Study of Tuberculosis,
Saranac Lake, New York

WHEN your Chairman asked me to speak of new developments in the field of silicosis I was perplexed because all but a few of the pathological, clinical and diagnostic aspects of this disease are now well established. I realized

that you had become better acquainted than I with the practical problems of diagnosis because you were dealing with them every day. After some deliberation I decided to tell you of some of the experimental work in progress at the Saranac Laboratory and to permit myself to speculate as to its significance. These experiments are incomplete and no conclusions are possible now, but perhaps they may ultimately lead to a better understanding of the action of irritant dusts upon the lungs.

As far as I have been able to discover, it is still true that only certain forms of free silica and the group of fibrous silicates known as asbestos, are capable of producing fibrosis in a normal lung. Many other silicates have been incriminated by roentgenologists, but when autopsy specimens have been examined it is discovered that there is associated chronic infection or that the inhaled dust contains significant quantities of free silica.

I shall first compare the action of chrysotile asbestos with that of quartz upon experimental animals and review considerable evidence bearing on their respective modes of action. A few of these observations now have practical significance; others may prove to be quite unrelated to the pneumoconioses found in human beings.

FOR the last year or two we have been coming to the conclusion that inhaled *asbestos* fibres are irritating not because they are silicates but because they are stiff fibres which mechanically irritate the lungs. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body; only those in the lungs are affected. It was inferred that these organs were affected because the movements of respiration are so much more rapid and continuous than those of other viscera. Then it was discovered that if asbestos was ground very finely so that few of the fibres were longer than 2μ in length the irritating property of the asbestos was practically destroyed. Inhalation experiments with such fine chrysotile asbestos have now been continued for three years. No fibrosis has developed in spite of the fact that an average atmospheric concentration of 125 million particles per cubic foot of air has been maintained. In contrast I would point out that in a previously reported experiment[1] one third this concentration of long fibre asbestos dust produced well marked fibrosis after about two years.

If the effect of asbestos were chemical, one would expect that a decrease in size would accelerate tissue response. With free silica large particles have little effect, but as their size decreases cellular reaction becomes more vigorous and even constitutional symptoms may ensue.[2] With fibrous asbestos the reverse is true.

The histology of early asbestosis does not suggest a chemical injury. Even under the most extreme conditions that can be created by artificial injection there is no preliminary phase of tissue necrosis with infiltration of leucocytes as occurs with high concentrations of very fine quartz. The connective tissue cells merely multiply very slowly in areas where the asbestos fibres are caught in the bronchioles. As collagen forms and contracts, the air spaces are obliterated by scar tissue. In experimental animals, at least, this change is not a progressive one after cessation of exposure to the dust as is the case in the response to quartz. Perhaps the reason is the deposition of the peculiar iron-containing coating on the surface of the inhaled fibres giving rise to the characteristic "asbestosis bodies." Dr. Timothy Leary considers[3] that the resultant smooth rounded ends of these bodies are no longer capable of scratching the delicate surfaces of the air spaces.

Finally it is most suggestive that among dozens of different silicate minerals only the five known as asbestos, which are unique because they are fibrous in structure, should be commonly recognized as pulmonary irritants. The variation in chemical composition within this group is greater than that between them and many other silicates. In fact chrysotile asbestos has the same chemical formula as a non-fibrous silicate, serpentine, which is physiologically inert. Obviously irritation would seem to be associated with the physical, rather than the chemical, composition of these minerals.

One point of practical significance may be indicated by these observations, namely, that very finely ground asbestos is not dangerous. This conclusion has support in clinical observation, for it has long been known that at the Thetford mills there was no clinical asbestosis even though in former years the atmosphere was very dusty and the dust was extremely fine. The fact that fabrication of fibres of the same mineral in American plants could produce disease was one of the puzzling features of this disease. But these experiments offer a plausible explanation. The fine dust in the mills is composed of serpentine and extremely short chrysotile fibres; that in the spinning and weaving mills contains many more long fibres.

IN THE case of *free silica* everything points to a chemical form of irritation although the solubility hypothesis as now conceived is not compatible with all the observations that have been made. In experimental animals inhaling or injected with excessive quantities of pure quartz or flint in exceedingly fine state of subdivision, it can be observed that tissue reaction takes place in two stages. First there is an acute inflammation resulting in necrosis. Following it there is healing with fibrosis in the form of a granuloma whose end product is the silicotic nodule. The collagen laid down by the connective tissue cells is modified in a peculiar way analagous to that in the tanning of leather, and this is responsible for the characteristic hyalinization of silicotic fibrosis. Incidentally, leather has been tanned with silica and sections of such leather reveal that the collagen has undergone a similar hyaline transformation. In human subjects only cases of rapidly developing silicosis show evidence of a two-stage reaction. In those with ordinary chronic disease, degenerative changes are slight and overshadowed by the formation of connective tissue.

The solubility hypothesis is based solely upon indirect evidence. It is known that colloidal silica is toxic and that quartz will dissolve to some extent in slightly alkaline fluids of the same pH as those of the body. But it has never been possible to prove that silica has dissolved within the body, because the soluble material is immediately combined with local tissue elements. It is now generally accepted that the soluble silica detected in the urine and blood probably comes from ingested food and drink rather than from inhaled dust.

WE have made a good many experiments whose results are difficult to reconcile with the solubility hypothesis. They do not necessarily invalidate it because we may not have all the facts and do not know how to interpret them. For example, we have found that cellular reactions begin within 15 to 30 minutes after injecting fine particulate silica. In the test-tube traces of dissolved silica are only discoverable by an exceedingly sensitive colorimetric reaction after 12 to 24 hours. Possibly "vital" factors may accelerate the process, but no means of evaluating them has yet been discovered. No one has ever produced fibrosis by single or repeated injections of the various forms of soluble silica. The effect is apparently not due to the quantity of silica dissolved, for many of the inert silicates in vitro are more soluble than quartz or flint. Evidences of alteration of the surface of quartz particles long in contact with living tissue have thus far escaped detection.

In years of experimenting to discover the mechanisms by which free silica exerts its peculiar effects upon living tissues we have repeatedly observed evidences of a specific toxicity. Guinea pigs, injected intraperitoneally with large doses of fine quartz particles are often obviously sick for some hours. When Mr. Cummings and Mr. Redlin[2] were seeking an avenue for the quantitative introduction of silica they tried intravenous injections into rabbits and discovered the method which we now use as a standard for comparing reactions to different dusts. But incidentally they found that these animals would die very promptly if the unit dose of quartz particles were too large. These deaths were not embolic because they did not occur with large particles but only when the quartz grains were 3μ or less in diameter, and they did not develop on injecting suspensions of other minerals ground to the same size.

Mr. Dworski and Mr. Delahant have extended these early observations in a long series of experiments that have yielded interesting though puzzling information. They have shown that fine silica injected into the left side of a guinea pig's heart acts like a strong poison.

Symptoms of shock followed by death within a few minutes to one hour occur when a 400-gram guinea pig receives an intracardiac injection of 3 or 4 cc. of a 1% suspension of 1 to 3μ quartz particles in physiological salt solution. The reaction is specific in that it is not produced by quartz particles of larger size and it has not been observed on injecting suspensions of several other minerals ground to the same particle size.

Symptoms are initiated even before completing the injection by the onset of rapid, labored respiration. The skin of the face and extremities becomes pale. The animal displays uncoordinated muscular movements and sporadic convulsions terminating in death if the dose is sufficient. When smaller doses are administered complete recovery takes place.

Autopsy of fatal cases discloses an intense engorgement of the spleen, omentum and subperitoneal tissues which are deep purple-red in color. The liver, and in fact practically all the remainder of the body, is almost bloodless. Counts of ear-vein blood show only about half the number of leucocytes present just before the injection and a corresponding reduction in red blood cells. The blood no longer clots. There is no hemolysis.

Animals that have recovered from one reaction seem to acquire a tolerance so that in some instances at least twice the former dose may be injected without symptoms. The degree of tolerance, however, varies widely in different individuals.

These symptoms are not influenced by the injection of adrenalin. They are not reproduced by transfusing the blood of an animal dying in shock into another one of the same species. They are only exhibited on injecting suspensions of quartz particles less than 3μ in diameter. They are not produced by injecting the supernatant salt solution from which the quartz particles have been removed by centrifugation. The severity of the reaction does not vary with the age of the particle suspension, although the amount of soluble silica increases on standing.

The toxic effects of quartz can be more or less effectively neutralized by adding small amounts of different substances. Colloidal aluminum hydroxide gives complete protection in a dilution of 0.03%; colloidal ferric hydroxide is much less effective. Animal charcoal cannot be injected, because it flocculates with the quartz and produces emboli. The finely ground metallic aluminum, used by Denny, Robson and Irwin[4] in the prevention of silicosis has no influence upon acute intoxication. Further experiments are in progress to verify the conclusion of these authors that aluminum coats the surface of the quartz particles with insoluble material.*

Recent observations indicate that all species of animals are not equally susceptible to intravenous injections of quartz. Dogs, which develop silicotic fibrosis very slowly and under conditions not yet defined, tolerate intravenous injections of comparatively large doses of fine quartz without symptoms of any kind. Rabbits, guinea pigs and white rats all die of shock.

Soluble colloidal silica in dispersed phase is also

* Subsequent experiments have shown that sterilizing suspensions of metallic aluminum by heat destroys their power of neutralizing the action of silica. Unheated sus-

toxic, and intravenous injections produce the reactions just described with extremely small doses. With larger ones death occurs promptly with symptoms referable to the respiratory tract. The lesions are then confined to over-distention of the lungs and subpleural petechial hemorrhages.

THE results produced by injecting particulate silica into the circulation all suggest a relationship to a large amount of mineral surface suddenly brought into contact with the blood or tissues. This is borne out both by the absence of reaction to large particles in excess of 3μ in diameter and possibly by the observations on neutralization. Any effect due to preformed soluble silica liberated in the suspension before injection has been eliminated, and no active soluble silica could be demonstrated in the blood of an animal dying in shock by transfusion into a second host. Everything now suggests an effect taking place very rapidly at the interface between the quartz and some element in the blood or tissues. Whether this is due to the very rapid production of colloidal silica in this location or to unique electrical properties of quartz is still a fascinating subject for speculation. In the same category is the demonstration of the tolerance conferred by previous injections.

The manifestation of acute toxic symptoms by those species of animals which develop silicotic fibrosis most readily and their absence in the dog, which appears to be much more resistant, suggests that these immediate reactions may have some bearing on the etiology of silicosis. Until more is learned about the reactions in the dog no inference is possible.

The influence of particle size is just as significant in the production of advanced silicotic fibrosis as it is in eliciting acute symptoms of intoxication. Many injection experiments have convinced us that quartz particles over 3μ in diameter are of little or no physiological significance. The fact that the lungs of persons exposed to most dusts contain few particles larger than 10μ in diameter has been responsible for the establishment of this size as the maximum important in dust counting. As a matter of fact we know that with some dusts there may be many larger ones. While it would be just as well that any large fragments should be excluded from the lungs, it is felt that only ones 3μ or less in diameter are responsible for causing silicotic fibrosis.

We have long maintained that particles of this size are irritating because they present a sufficient surface of silica to the fluids. Recently Mr. Dworski decided to test this reasoning further by injecting graded doses of different sized quartz particles which would all present the same total surface area. He found, however, that regardless of the total surface, silicotic reaction only developed when the size of the particles was 3μ or less. On examining his material it was discovered that with particles 4 to 5μ in diameter or larger there were only two to four visible in a thin section of each phagocyte. These cells appeared to be quite uninfluenced by the ingested material. In the case of particles 3μ or under the number inside each phagocyte was countless, and there was marked evidence of degeneration of the cytoplasm of these cells. In other words, the effect of surface area had to be exerted inside of the cell and not upon the body fluids as a whole. Extracellular quartz seemed to have little influence, and even intracellular particles of a large size produced no visible evidence of irritation.

THIS observation brings us back to the cause of these progressive changes. Apparently they are initiated with degeneration in the primary phagocytes, an effect which is exactly paralleled in the histogenesis of tuberculosis. Some years ago I published a paper calling attention to the similarities between silicosis and tuberculosis.[5] In both of these diseases the primary irritants, quartz grains or tubercule bacilli, as the case may be, intoxicate the phagocytes. In tuberculosis the cells thus poisoned are commonly designated as "epithelioid cells;" in silicosis the morphological effect is similar. Visible fat droplets accumulate in the cytoplasm and the elements stainable by supravital dyes undergo rearrangements in characteristic patterns. Giant cells of the Langhan's type are produced in each disease by stimulation and repeated multiplication of the nucleus. In each disease many cells die and their degenerated products are liberated in the tissue spaces. Incidentally it is the pabulum of necrotic cells in silicotic lesions on which tubercle thrive so well, an effect which is responsible for the high frequency of complicating tuberculous infection in silicotics. In the tubercle it is in the necrotic or caseous tissue that tubercle bacilli are most frequent. Perhaps they not only cause such caseation but they themselves thrive on it. Coincidentally more cells migrate to the area and proliferate to form the granulomatous nodule that is characteristic of each disease.

Finally the production of necrotic tissue, high in lipoids, by two very dissimilar primary irritants recalls the experiments of Fallon[6] and his hypothesis that it is these lipoids which are directly responsible for the formation of the granuloma. He showed that the injection of such lipoids, freed at least partially of contaminating silica particles, was capable of producing a granulomatous nodule.

Such mechanisms may be involved in the production of both silicotic nodules and tubercles. It still remains to demonstrate the nature of the primary injury by each of these irritants, and it is this problem that will probably engage our attention for some time to come.

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A Plan for the Control of Tuberculosis in Industry

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IT IS a proud day when it can be stated that one of the safest places to be in this country is at work. Statistics from the National Safety Council seem to bear this out. The reduction in the frequency and severity accident rates in our industrial institutions has been nothing short of remarkable. Prevention has been the watchword.

With such improvements, the consideration of the medical departments in industry has more recently been drawn to the general health of its employees. Attention to the control of tuberculosis has been a very major issue. We believe that in no adult group throughout the country can so much be accomplished in prevention and early recognition of this disease as in our industrial institutions.

Legislation varies in the different States regarding occupational disease but the ultimate objective of this legislation is clear: The general health of the employee must increasingly become the responsibility of the employer. While the status of tuberculosis has been debated by many legislative bodies, silico-tuberculosis is definitely compensable. In the state of Illinois the new occupational disease disability compensation law went into effect on October 1, 1936. Dr. C. O. Sappington states that "during the first year of the operation of this law, 10.9% of disability claims filed were for pulmonary tuberculosis while silicosis claims were 15% of the total."[1]

The corporation that is looking toward the future with the best interests of its employees in mind certainly should be actively engaged in the problem of the control of tuberculosis. Many of our industrial institutions have already shown, by marked reduction of the incidence of pulmonary tuberculosis, just how this can be accomplished. Our own corporation has a very definite program concerning tuberculosis.

THE PROGRAM: 1. THE PRE-EMPLOYMENT PHYSICAL EXAMINATION. Every prospective tion. This is done with the intent of placing that individual in industry where he or she is best physically as well as mentally fitted. This examination is not done for the purpose of either passing or rejecting that person for employment. This pre-employment examination gives the examining physician the best possible opportunity to detect pulmonary tuberculosis.

Dr. Robinson Bosworth, President of the Illinois Tuberculosis Association, states:[2] "85% of adult pulmonary tuberculosis has its beginning in the age group 18 to 28. In the vast majority of cases there are no symptoms and no physical signs."

It is exactly this age group that concerns us most in our pre-employment physical examination, for it is during this age period that most employees first enter industry. We therefore feel that such an examination is not complete without an x-ray of the chest.

We believe, with Dr. Andrew R. Riddell, of the Industrial Hygiene Department, Ontario Department of Health, that the wholesale tuberculin testing in industry has many disadvantages. He states that[3] "the individuals must present themselves at least three times—there is considerable disruption of work—even after most careful explanation as to the meaning of the test, the employees who react positively are greatly disturbed."

Applicants in whom active pulmonary tuberculosis is discovered are sent to their family physician. Our roentgenogram is at his disposal, but must be returned to our files so that we can later compare the progress of the disease and determine at what future time those persons may be pronounced employable.

Applicants with arrested or quiescent tuberculosis are placed at work after being informed of their condition and instructed to return for periodic examinations. These examinations for new employees, in this classification, are conducted every three months.

It has been increasingly evident that our responsibility does not end there. We explain to the employee that while he now has an arrested tuberculosis, it was once active. We encourage him to have the members of his family examined by the family physician. We feel that not until then has industry wholly discharged its duty to the community.

2. PERIODIC EXAMINATIONS FOR OLD EMPLOYEES. These examinations are made at different times and for varied reasons:

(a) Upon returning to work after vacations.
(b) After absence caused by an illness.
(c) Re-employment following seasonal lay-off.
(d) Transfer from one type of work to another.
(e) Medical department requesting the examination for some definite reason.
(f) Volunteer requests by the employee for such an examination.

Little comment is necessary concerning these examinations except to state that every employee comes into our medical department for a general physical check up at least once each year.

MELLON INSTITUTE OF INDUSTRIAL RESEARCH
PITTSBURGH, PA.

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Abst. 460 June '40	75 Y			752

AUTHOR: E. C. Vigliani

TITLE: Two fatal cases of pulmonary asbestosis.

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ABSTRACT: The first of these cases concerned a woman who worked for thirty years in a factory using asbestos in the manufacture of cord. The second woman worked for seven years in an asbestos factory. Her susceptibility to colds increased while she was working and she suffered all her life from bronchitis and asthma. She died at the age of fifty, with symptoms of a lung disease, nearly thirty years after having stopped work. The autopsy showed interstitial sclerosis of the lung and asbestosis bodies.

CONT'D.

DATE EXAMINED: BY: T. L. M. JTH PURPOSE OF SEARCH:

ABSTRACT BLANK FOR GENERAL USE

DUE CASI MORTALI DI ASBESTOSI POLMONARE *

Prof. ENRICO C. VIGLIANI

L'amianto è un minerale a struttura fibrosa: le sue fibre più lunghe si prestano ad essere cardate, filate, tessute; quelle più corte servono alla preparazione di filtri, freni, isolanti, cartoni, eternit, ecc. La molteplicità dell'impiego dell'amianto deriva dalla sua utilizzazione come materiale tessile e dalle sue proprietà isolanti e incombustibili; la grande richiesta di manufatti di amianto da parte dell'industria moderna ha fatto sì che la lavorazione di questo minerale si sia più che decuplicata nel volgere di una ventina di anni.

Dal punto di vista chimico l'amianto è costituito principalmente da silicati di ferro e di magnesio: a seconda che prevale l'uno o l'altro silicato l'amianto ha colore, proprietà e denominazioni diverse.

Durante le varie lavorazioni dell'amianto si sviluppa una polvere contenente minutissime fibre del minerale, polvere che viene inalata dagli operai. Per molti anni si ignorò che essa potesse essere dannosa; l'osservazione di un caso di sclerosi polmonare da amianto fatta nel 1900 e pubblicata nel 1907 dal Dr. *Montague Murray* [1] passò sotto silenzio, finchè nel 1927-1928 furono descritti alcuni casi mortali di asbestosi in Inghilterra per opera di *Cooke* [2, 3], *Mc Donald* [4], *Oliver* [5, 6], *Seiler* [7], *Steward* [8].

Gli studi clinici, radiografici e anatomopatologici relativi a questi casi destarono vivo interesse fra i medici del lavoro. Negli anni successivi furono descritti in Inghilterra (*Haddow* [9], *Wood* [10], *Wood* e *Gloyne* [11], *Seiler* e *Gilmour* [12], ecc.), nel Sud Africa (*Hall* [13], *Simson* [14]), negli Stati Uniti (*Lynch* e *Smith* [15], *Mills* [16]) e in Germania (*Beintker* [17], *Buresch* [18], *Holtzmann* [19], *Rostoski* [20], ecc.), altri casi di asbestosi; ai quali fecero seguito accurati studi anatomo-patologici e mineralogici (*Gloyne* [21], *Beger* [23, 24, 25], *Koppenhöfer* [26], *Sundius* e *Bygdén* [27], *Di Biasi* [28]). Osservazioni compiute fra gli operai di manifatture d'amianto (*Lovisetto* [29], *Mussa* [30], *Merewether* [31, 32], *Gerbis* e *Ucko* [33], *Lanza*,

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* Dalla *Clinica Medica Generale della R. Università di Torino*: Direttore Prof. CARLO GAMNA; e dall'*Istituto di Medicina Industriale di Torino dell'E.N.P.I.*: Direttore Prof. ENRICO C. VIGLIANI.

Mc Connell e *Fehnel* [34] e alcuni bellissimi lavori riassuntivi (*Sparks* [35], *Merewether* [36], *Wood e Gloyne* [37] ecc.) fecero riconoscere una notevole frequenza di casi di sclerosi polmonare riferibili alla prolungata inalazione di polvere. Per tal modo venne dimostrata la pericolosità della lavorazione dell'amianto e si costituì la conoscenza della malattia polmonare che essa può produrre, malattia che era stata chiamata *asbestosi*.

Fondamentalmente noi sappiamo oggi che l'asbestosi è una sorta di polmonite interstiziale cronica rivestente l'aspetto finale di una sclerosi diffusa, non nodulare, del polmone. La sua patogenesi appare legata alla penetrazione nei polmoni di minutissime fibre d'amianto e alla conseguente reazione del tessuto connettivo polmonare: nelle sezioni istologiche dei polmoni asbestosici si vedono, accanto alla fibrosi e ad aree di enfisema, numerosissimi aghi e fibre di amianto, alcuni dei quali appaiono nudi e altri rivestiti di un involucro di colorito giallastro, contenente molto ferro. Gli aghi d'asbesto così rivestiti hanno aspetti curiosi e caratteristici e furono detti *corpuscoli dell'asbestosi*.

Clinicamente la malattia inizia quando la diminuzione della superficie respiratoria dovuta alla fibrosi polmonare incomincia a farsi sentire; i primi segni sono la tosse e la brevità di respiro. Estendendosi la fibrosi la dispnea diviene sempre più grave e ad essa si possono accompagnare sintomi di scompenso del cuore destro. All'esame oggettivo non si notano altro che i segni della fibrosi polmonare, più evidenti alle basi; la morte avviene per insufficienza respiratoria e cardiaca o per tubercolosi o, molto più spesso, per malattie polmonari intercorrenti, specie broncopolmonite.

Le radiografie polmonari degli asbestosici lasciano vedere una diminuzione della trasparenza dei campi inferiori e medi dei polmoni dovuta a una diffusa velatura e a un vario intreccio di ombre striiformi e reticolari; in generale però esse danno l'aspetto di una gravità minore di quello che clinicamente non sia.

Data la scarsità dei segni clinico-radiologici, la mancanza di sintomi veramente caratteristici e la frequente sovrapposizione di malattie intercorrenti o di scompenso di cuore, la malattia è difficilmente riconoscibile, se i dati anamnestici non mettono bene in evidenza la prolungata inalazione di polvere di amianto e se la esistenza di una sclerosi polmonare da amianto non è conosciuta dal medico visitante.

La grande difficoltà della diagnosi clinica dell'asbestosi è riconosciuta da tutti i ricercatori e in realtà i casi clinici diagnosticati e riportati singolarmente sono piuttosto rari. (*Cooke* [2], [3] un caso (1924-1927); *Oliver* [6] due casi (1926); *Simson* [14] due casi (1928); *Seiler* [7] un caso (1928); *Wood* e *Page* [38] un caso (1929); *Haddow* [8] quattro casi (1929); *Soper* [39] un caso (1930); *Mills* [16] un caso (1930); *Lynch* e *Smith* [15] un caso (1931); *Seiler* e *Gilmour* [12] un caso (1931); *Stewart, Butcher* e *Coleman* [40] due casi (1931); *Buresch* [18] un caso (1931); *Beintker* [17] due casi (1931), *Ste-*

wart, Tattersal e *Haddow* [41] un caso (1932); *Ellman* [42] un caso (1933); *Stock* [43] un caso (1933); *Stroebe* [44] un caso (1933); *White* [45] un caso (1935); *Egbert* [46] un caso (1935); *Martz* [47] un caso (1935); *Bohne* [48] un caso (1936); *Di Biasi* [28] un caso (1937) e senza citare i casi di carcinomi polmonari in asbestosici).

La maggior parte della casistica di cui oggi disponiamo deriva da ospedali per le malattie di petto situati nelle vicinanze di manifatture di amianto (*Wood* e *Gloyne* [37], nel « City of London Hospital » videro in pochi anni più di 100 casi di asbestosi di cui 36 mortali), o da ricerche sistematiche fatte fra le maestranze di tali manifatture (*Merewether* [31], [32], *Gerbis* e *Ucko* [33], *Shull* [49], *Mc Pheeters* [50], *Alwens* [51], *Saupe* [52], [53], *Lanza* [54], *Dreessen* e collab. [55], *Wedler* [56] ecc.); se non vi sono queste favorevoli condizioni, la grande maggioranza dei casi di asbestosi viene all'exitus colla diagnosi di scompenso cardiaco, di broncopolmonite o di tubercolosi polmonare; diagnosi magari esatte in quanto all'episodio terminale, ma dalle quali non appare la malattia primitiva e fondamentale.

In Italia, per esempio, e specialmente in Piemonte, ove vi sono numerose cave e manifatture di amianto nelle quali lavorano più di 1000 operai, non è ancora mai stato descritto un caso mortale di asbestosi; i pochi autori che si sono occupati dell'argomento, *Lovisetto* [29] e *Mussa* [30], ritennero anzi la malattia non grave in sè, ma solo pericolosa per il facile sopravvenire della tubercolosi. Deve essere però, a onor del vero, ricordato che nel 1910, quando ancora la malattia era del tutto sconosciuta (salvo la pubblicazione di *M. Murray* [1] del 1907 nel Departmental Committee on Compensation for Industrial Disease) il Dottor *Castagneri*, medico condotto di Nole Canavese, descrisse come tesi di laurea un caso mortale di asbestosi polmonare complicato da tubercolosi: malauguratamente questa tesi non venne pubblicata, ma essa è visibile negli archivi dell'Università di Torino e i pezzi anatomici di quel caso sono ancora oggi conservati nel Museo dell'Istituto di Medicina Legale della stessa città.

Mi pare quindi utile di riferire due casi mortali di asbestosi osservati nella Clinica Medica di Torino. L'interesse di questi casi non deriva solo dal fatto che essi rappresentano i primi descritti in Italia e quindi possono servire ad attirare l'attenzione dei medici su questa malattia professionale, che certamente in Piemonte è più frequente di quanto non si creda, ma anche per alcune altre considerazioni. Infatti essi per le particolari circostanze del loro sviluppo e del loro decorso si prestano egregiamente a discutere alcuni punti ancora controversi dell'asbestosi e principalmente quello dell'aggravamento della malattia cessata l'esposizione alla polvere. Infine la descrizione clinica di questi casi serve di utile completamento al bellissimo lavoro anatomopatologico che su di essi hanno compiuto il Prof. *Mottura* e il Dott. *Fagiano* [57] e che verrà pure pubblicato su questo giornale.

CASO 1° — Am. Maria, a. 56, nata a Foggia.

Anamnesi: La paziente è secondogenita di 15 figli di cui 11 morirono in tenera età. Nulla da segnalare nell'infanzia e in gioventù, tranne una affezione all'occhio sinistro che guarì con esteso leucoma corneale. Sposata a 15 anni, non ebbe gravidanze. Fino all'età di 25 anni si dedicò a lavori casalinghi, poi dal 1907 fino a dicembre 1936, e cioè per 30 anni, lavorò a Torino come operaia in una fabbrica di corde. Durante questo periodo non fu mai soggetta a disturbi degni di nota, eccetto a volte un po' di tosse senza escreato; da qualche tempo però la tosse era più frequente ed era comparsa modica dispnea da sforzo.

Nel dicembre 1936 fu colpita da un episodio influenzale con tosse ostinata pressochè senza escreato, dispepsia, dispnea durante il riposo e accentuantesi per i più piccoli sforzi, talora senso di cardiopalmo. Rimase a letto per circa 15 giorni dopodichè potè alzarsi e occuparsi delle faccende di casa accusando solo più una certa astenia, affanno di respiro e senso di palpitazione cardiaca durante la marcia e fatiche anche di poca entità. Alla fine del marzo 1937 la dispnea si aggravò nuovamente tormentando l'a. anche durante il riposo in letto; l'astenia e il cardiopalmo si rifecero assai evidenti assieme a senso di peso alla regione epigastrica; comparvero edemi malleolari mentre la diuresi si fece assai scarsa.

Per questa sintomatologia l'a. entrò in clinica il 9 Aprile 1937.

Esame oggettivo: Donna di statura bassa, di costituzione robusta, in discreto stato di nutrizione. E' intensamente dispnoica e costretta a mantenere in letto la posizione seduta. Cute fortemente cianotica in specie al volto e ovunque succulenta: edemi spiccati si notano al terzo inferiore delle gambe, alle regioni lombosacrali e alle parti declivi dell'addome. Sistema linfatico superficiale indifferente. Polso frequente (102-108 battute al minuto) ritmico, piuttosto molle; respiri 44-48 al minuto, superficiali, a tipo misto. Temperatura oscillante fra i 37° e i 38°. A carico del *capo* vi è da rilevare solo un leucoma centrale dell'occhio S e un'intensa cianosi dei pomelli, delle orecchie e specialmente delle labbra. Tonsille e faringe alquanto arrossate. Al *collo* si apprezzano assai turgide e pulsanti le vene giugulari; tiroide in limiti.

Torace: Regolare di forma, simmetrico, un po' rigido, si espande poco nelle inspirazioni. *Apici*: Aree di Krönig di eguale ampiezza bilateralmente; suono plessico chiaro, f. v. t. ben trasmesso, respiro vescicolare. *Polmoni*: basi polmonari scarsamente mobili. Suono di percussione chiaro nelle regioni superiori, alquanto smorzato bilateralmente, ma più a D., verso le basi. All'ascoltazione respiro vescicolare ovunque, un po' diminuito verso le basi ove si ascoltano numerosi rantoli a piccole e medie bolle in- ed espiratori. I rantoli si apprezzano pure numerosi, nelle regioni basali, in corrispondenza delle linee ascellari; appaiono in complesso più fitti a D. che a S. *Cuore*: itto nel V spazio intercostale sull'ascellare anteriore; area di Orsi-Grocco normale; a D. l'ottusità relativa del cuore a livello della IV e V costa si estende fino a due dita trasverse all'esterno della marginosternale destra. Alla ascoltazione toni assai frequenti, soffio sistolico sull'area mesocardica.

Addome: piuttosto espanso, dolente la palpazione della regione epatica; il fegato giunge in basso a un dito al disotto della linea ombelicale trasversa; la milza non si palpa. *Arti*: nulla di notevole, eccetto la presenza di evidenti edemi ai piedi e alle regioni malleolari e pretibiali. *Sistema nervoso* indenne. *Esame delle orine*: quantità giornaliera cc. 500, Δ 1021, albumina velo, nel sedimento scarsi gl. rossi, molti gl. bianchi e cellule di sfaldamento. *Es. ematologico*: Hb 82, G. rossi 6.740.000; V. gl. 0.61. G. bianchi 7800 (Pol. N. 64, Eo. 2, Linf. 26, Mon. 8). Pressione arteriosa 130/70 (Riva-Rocci). Reaz. Wassermann, Meinicke, Kahn negative.

Esame radiografico: I campi polmonari presentano un seminio di ombre a forma di piccole chiazze e strie, più numerose in vicinanza degli ili e dei margini del cuore, ove si addensano e quasi confluiscono in opacità maggiori. Il polmone destro è più colpito del sinistro ed è pure diffusamente velato specie nella regione apicale e sottoclavicolare. A sinistra fra le chiazze e le strie si vedono piccole aree irregolari di maggiore trasparenza. Nel complesso a sinistra è visibile una certa disposizione raggiata dalle strie, che si presentano spesse anche 4-5 mm. e ad ombra tenue e sfumata. Le chiazze non hanno, se non molto raramente, un aspetto nodulare, ma si presentano a contorni frastagliati e irregolari, apparendo spesso come punti di incrocio o di sovrapposizione di tenui ombre di strie. Qualche volta piccole chiazze sono centrate da un bronco. I margini del cuore sono quasi completamente irriconoscibili; l'area cardiaca appare però alquanto ingrandita specie verso destra. Pure i margini dell'ombra diaframmatica, particolarmente a sinistra, sono sfumati. Alcune aderenze pleurodiaframmatiche all'angolo epatocardiaco e all'emidiaframma sinistro.

Durante la degenza in Clinica, nonostante l'istituzione di una energica terapia a base di salasso, diuretici mercuriali, uabaina endovena e analettici, le condizioni della paziente, dopo un transitorio miglioramento durato 2-3 giorni, peggiorarono progressivamente. La pressione arteriosa si abbassò a valori di 90/50, la dispnea, gli edemi, la cianosi e la tachicardia si fecero ancora più intensi; i rantoli alle basi polmonari più numerosi; la temperatura ebbe qualche punta serotina sui 38°,5-38°,7, finchè l'exitus sopravvenne il 25 aprile 1937, dopo 16 giorni di degenza.

La natura della malattia che aveva portato a morte la paziente appariva estremamente oscura. Fu fatta diagnosi clinica di « scompenso cardiaco secondario a sclerosi polmonare ».

L'esame necroscopico (Prof. *Vanzetti*) fornì nelle parti essenziali il reperto seguente:

Aumento dell'area cardiaca scoperta, cuore leggermente aumentato di volume. Apparati valvolari integri. Miocardio omogeneo, rosso. Ventricolo destro del cuore notevolmente dilatato, muscolatura del ventricolo destro ipertrofica. Arteria polmonare alquanto ectasica, con focolai di degenerazione grassa.

Polmoni: Aderenze totali e difficilmente vincibili a sinistra; a destra aderenze limitate alle parti superiori del polmone. Pleure viscerali assai ispessite. Entrambi i polmoni si presentano aumentati di consistenza e di peso. Alla sezione dei polmoni si percepisce meglio l'aumento della loro consistenza. La superficie di taglio è variegata e permette di osservare una intensa sclerosi diffusa sotto forme di strie e di focolai irregolari di colore grigio non sporgenti sulla superficie. La sclerosi è maggiore nei lobi medii e inferiori dei polmoni, specie a destra, ma evidente anche in quelli superiori. Qua e là si vedono piccole aree circoscritte di enfisema. Nei bronchi note di bronchite catarrale. Gangli linfatici ilari più grossi, ma non più duri che di norma: uno di essi appare calcificato. All'infuori di questa ghiandola calcificata, probabile espressione del complesso primario, nessun altro segno di infezione tubercolare pregressa o in atto.

Modico versamento ascitico. Fegato, milza, reni da stasi. Nulla di importante a carico del canale digerente e degli organi genitali. In complesso: sclerosi interstiziale del polmone, dilatazione e ipertrofia del ventricolo destro, stasi nel grande circolo.

Il risultato dell'esame necroscopico confermava che lo scompenso cardiaco era dovuto alla intensa sclerosi dei polmoni.

Rimaneva ancora da risolvere il mistero dell'eziologia della sclerosi polmonare,

Fig. 1. - Am. Maria.

Fig. 2. - N. Teresa.

mistero che neppure gli esami istologici riuscirono a tutta prima a svelare. Essi avevano messo in luce un processo di polmonite cronica interstiziale o meglio gli esiti di questo processo polmonitico in forma di una sclerosi interstiziale diffusa non nodulare. La fibrosi interessava, con intensità aumentante dall'apice alle basi, specialmente i setti interalveolari e quelli interlobulari, che apparivano molto più spessi che di norma; in grado minore gli spazi peribronchiali e perivascolari; qua e là zone di addensamento maggiori date sia da focolai di atelettasia che da processi di sclerosi più estesi, ma senza aspetto nodulare e comunque sempre senza una netta disposizione concentrica dei fasci delle fibre collagene. Numerosi focolai acinosi e lobulari di enfisema sparsi in mezzo alle zone sclerotiche.

Che cosa poteva aver provocato una simile alterazione polmonare? Non la lieve sclerosi dell'arteria polmonare; non la lue, poichè gli esami sierologici erano stati negativi; non la tubercolosi; nemmeno si trattava di una linfangite carcinomatosa. Col Prof. *Mottura* e il Dott. *Fagiano*, che si occupavano attivamente dello studio delle pneumoconiosi, osservai un giorno alcuni preparati istologici dei polmoni di questa donna. A medio e forte ingrandimento potei riconoscere in mezzo alle zone fibrotiche, nei setti interalveolari e interlobulari e anche nel lume degli alveoli, vuoti o ripieni di detriti cellulari, di cellule desquamate e di liquido coagulato, in una parola un po' dappertutto, numerosissimi corpiccioli di colorito giallo-oro carico, stranamente foggiati a forma di clava, di bastone, di manubrio da ginnastica, di frammento di collana; corpiccioli che a un esame attento lasciavano riconoscere spesso nel centro una fibra o un ago di sostanza molto rifrangente. Il mistero si chiariva; la diagnosi diventava evidente: i corpiccioli erano i così detti « corpi dell'asbestosi »; la fibrosi polmonare una pneumoconiosi da polvere di amianto. Nessuna speciale abilità in questo riconoscimento: chi ha visto anche una sola volta i corpi dell'asbestosi in preparati microscopici o anche solo in riproduzioni microfotografiche non può più dimenticarli, tanto il loro aspetto è caratteristico.

Ma come era possibile l'esistenza di una asbestosi polmonare, se il lavoro coll'asbesto non figurava nell'anamnesi della paziente? L'inchiesta condotta fra i familiari della paziente rivelò che essa, per circa 30 anni, aveva bensì lavorato a fabbricar corde, come essa aveva raccontato durante il rilievo dell'anamnesi, ma che queste corde erano di amianto e che la loro fabbricazione cagionava la diffusione nell'atmosfera di una certa quantità di polvere di questo minerale.

Caso 2°. — N. Teresa, a. 50, nata a Nole Canavese (Torino).

Anamnesi: Nulla di importante nell'anamnesi familiare. Lo sviluppo fisico e psichico della paziente furono regolari: essa però afferma di aver sofferto nella prima infanzia di manifestazioni riconducibili a una diatesi essudativa, le quali si ripresentarono durante l'età scolara, cessando poi del tutto verso i 12 anni. A 14 anni superò l'infezione tifoide. In quello stesso anno, e cioè nel 1902, entrò a lavorare in una manifattura torinese di amianto, ove rimase fino all'età di 21 anno, cioè fino al 1909, epoca in cui abbandonò il lavoro per sposare un uomo di 26 anni, sano, dal quale ebbe tre aborti, nessun figlio vivo.

La paziente riferisce che fin dalla fanciullezza andava frequentemente soggetta a raffreddori invernali. Durante il lavoro nella manifattura di amianto comparve tosse saltuaria e si accentuò la tendenza ai raffreddori: il marito della paziente afferma che da quando egli la conobbe, essa era affetta da tosse con escreato mucoso, tosse che si presentava a volte in forma di veri accessi più o meno lunghi. Nel 1912, a 25 anni, fu assunta in una conceria di pelli e venne addetta a un la-

voro piuttosto faticoso. Dopo 10 anni e cioè nel 1922 fu colpita nel mese di novembre da febbre preceduta da brivido e accompagnata da accessi di tosse stizzosa con scarsissimo escreato, insorgenti specie durante la notte e seguiti da dispnea e senso di soffocamento. Un sanitario fece diagnosi di asma bronchiale e prescrisse una terapia antiasmatica dalla quale essa ritrasse alquanto giovamento; la febbre cessò in 8-10 giorni e la tosse diminuì molto, senza tuttavia mai scomparire. Rimessasi da questo episodio morboso la paziente non si sentì più in grado di sopportare il faticoso lavoro nella conceria per il persistere della tossetta, di una facile stancabilità e di un senso di affanno per i lavori pesanti e si impiegò per due anni alla Snia Viscosa, trascorsi i quali, tendendo la sintomatologia suaccennata ad accentuarsi, fu costretta a rimanere in casa accudendo solo più al disbrigo delle faccende domestiche.

Nell'inverno 1926 cioè a 39 anni essa fu nuovamente colta da brivido, febbre e violenti accessi di tosse accompagnati da senso di soffocazione e dispnea. Fu nuovamente posta la diagnosi di asma bronchiale. La febbre si dileguò in pochi giorni, ma la tosse permase insistente fino a primavera inoltrata. Da allora la paziente riferisce che ogni anno, all'inizio dell'inverno, ritornavano gli accessi di tosse, a volte preceduti da brividi e da un po' di febbre, mentre il senso di soffocazione e di dispnea si facevano man mano più evidenti, comparendo non solo durante le fatiche o la tosse, ma anche indipendentemente da queste, in occasione di lievi lavori fisici. La tosse era più insistente al mattino e dopo i pasti; la terapia antiasmatica non apportava più alcun sollievo, la malata sentendosi anzi ogni inverno più stanca, più tossicolosa e più dispnoica dell'inverno precedente. Nel 1935, durante un accesso violento di tosse, ebbe pure escreato abbondantemente tinto di sangue; nell'inverno 1936 fu colpita improvvisamente, nel pomeriggio, da un accesso di soffocazione che durò fino al mattino seguente; dopo questo episodio si accentuò l'astenia e comparve pure marcata disappetenza. All'inizio dell'inverno 1936-1937 comparve senso di peso all'epigastrio e talora vomito, riprese la solita tosse invernale: l'astenia era grande e la dispnea interveniva per fatiche anche minime. Nel marzo 1937 incominciò a notare un colorito cianotico delle mucose e della pelle del volto, che andò man mano accentuandosi. Nonostante il ricovero per circa un mese in un ospedale, le condizioni della paziente si fecero progressivamente più precarie finchè, essendo comparsi cospicui edemi agli arti inferiori, essa si fece ricoverare, il 10 gennaio 1938, in Clinica medica.

Esame oggettivo: Statura alta, conformazione scheletrica regolare, condizioni generali discrete; peso Kg. 73.600. Colorito piuttosto pallido; cianosi intensa alle labbra, ai pomelli delle guance, alle orecchie e alle dita delle mani. Sistema linfatico superficiale indifferente. Polso di frequenza 100 al minuto, ritmico. Respiri 24. Temperatura 37°.7. Pressione arteriosa 120/80. All'ispezione del *capo* si osserva che la cute e anche i bordi della lingua, le tonsille e la faringe presentano un colorito cianotico, bluastro. Le vene del collo si disegnano assai turgide.

Torace: angolo epigastrico acuto: fosse sopra e sottoclavicolari evidenti, spazi intercostali ristretti. Le escursioni respiratorie appaiono molto ridotte.

Apparato respiratorio: apici normali per fonesi e murmure vescicolare. Il suono di percussione polmonare chiaro in alto va gradatamente smorzandosi verso le basi, le quali sono pressochè immobili e nettamente ipofonetiche, la destra per una estensione e con un'intensità maggiore della sinistra. Il f. v. t. è ben trasmesso in alto, indebolito, quasi abolito verso le basi. All'ascoltazione si notano su tutto l'ambito respiro vescicolare piuttosto aspro e numerosi rumori bronchiali sia

secchi che umidi in- ed espiratori, i quali verso le basi vanno facendosi più abbondanti: quivi e specie a destra, il respiro si ode assai diminuito.

Cuore: L'itto non si vede nè si palpa, si delimita colla percussione nel VI spazio intercostale sull'ascellare anteriore. Il margine sinistro dell'ottusità relativa del cuore è assai convesso e ingrandito; l'area cardiaca appare pure ingrandita verso destra, ove giunge a circa 2 ½ dita dalla marginosternale sulla IV costa. All'ascoltazione alla punta il primo tono appare un po' strascicato, quasi sdoppiato. Il secondo tono è nettamente rinforzato sui focolai della base, specialmente su quello polmonare.

Addome: Un po' tumido, palpabile, indolente. Segni di modico versamento libero. Il margine inferiore del fegato si palpa al livello dell'ombelicale trasversa, di consistenza aumentata. Milza in limiti. *Arti*: edemi evidenti ai piedi e alle regioni malleolari; meno marcati alle radici delle coscie e alle regioni sacrali. *Sistema nervoso* normale. *Esame delle orine*: quantità giornaliera cc. 400. Aspetto torbido, densità 1021, reazione acida, albumina presente (+): abbondante sedimento laterizio con cellule di sfadamento e leucociti. Esame morfologico del *sangue*: Hb 78, globuli rossi 4.300.000, val. gl. 0,90, globuli bianchi 6.400 (Pol. n. 72, Eo. 1. Linf. 22, Mon. 5). Reazioni di Wassermann, Meinicke, Kahn negative. Pressione venosa alquanto aumentata (30-35 cm H_2O. Claude). Urea ipobromitica gr. 0.46 per mille. Con una puntura esplorativa eseguita alla base dell'emitorace destro si estrassero circa 70 cmc. di liquido con i caratteri di un essudato e il cui sedimento *si dimostrò costituito in prevalenza da linfociti.*

Esame radiografico del torace, eseguito dopo la toracentesi: I campi polmonari appaiono velati nella loro metà inferiore e quasi completamente opacati verso le basi. A destra, verso la base, si vede un pneumotorace parziale con un livello liquido. L'opacamento dei polmoni è diffuso, come una nebbia che sale dal diaframma: solo in qualche punto, nelle zone medie dei polmoni, si intravede il disegno secondario del polmone accentuato. Non ombre nodulari nè a chiazze. Verso la regione cardiaca l'opacamento polmonare è più intenso e si confonde con l'ombra del cuore, che appare fortemente ingrandita sia verso destra che verso sinistra. Le cupole diaframmatiche non sono visibili. Il lobo superiore sinistro e la regione apicale destra hanno una trasparenza maggiore del normale.

Nei primi giorni di degenza in Clinica la cianosi, gli edemi e gli altri segni di insufficenza cardiaca furono molto spiccati. L'escreato era pure abbondante, mucopurulento: la ricerca del bacillo di Koch, praticata ripetutamente, risultò sempre negativa. Con una energica terapia a base di tachidrolo, salassi, digitosan, le condizioni migliorarono alquanto. Permaneva però sempre a carico dei polmoni il reperto di bronchite diffusa con numerosi crepiti e rantoli, l'ipofonesi alle basi e l'indebolimento del respiro specie alla base di destra.

Dopo 15 giorni di degenza in Clinica, quando le condizioni generali erano in complesso soddisfacenti, insorse febbre irregolare con massimi serali di 38°,2-39°. Una velocità di sedimentazione diede i valori seguenti: 1ª ora 35, costante di Katz 35. In corrispondenza del lobo inferiore di sinistra, sia anteriormente che posteriormente, si accentuarono l'ipofonesi e i rantoli a piccole e medie bolle. I fenomeni bronchitici, gli edemi e la cianosi aumentarono assai. Nei giorni seguenti la febbre ebbe un decorso assai irregolare, mentre le condizioni generali andarono sempre più decadendo, con aumento degli edemi, della cianosi, della frequenza del polso e del respiro e con forte diminuzione della diuresi. Infine exitus il 15 febbraio 1938, dopo 36 giorni di degenza.

Fu fatta la diagnosi clinica di scompenso cardiaco, bronchite cronica con

probabile sclerosi interstiziale del polmone, pleurite destra, broncopolmonite terminale. Dal punto di vista clinico, ma specialmente anamnestico, era chiaro che la bronchite cronica e la sclerosi polmonare rappresentavano i fatti primitivi e che lo scompenso di cuore doveva essere secondario ad essi; solo la natura della sclerosi polmonare appariva poco chiara, non essendosi in principio attribuita importanza al dato anamnestico della esposizione all'inalazione di polvere di amianto avvenuta per soli sette anni e a così grande distanza di tempo (30 anni) dall'exitus.

L'*autopsia* (settore Prof. *Mottura*) diede, in breve riassunti, i risultati seguenti: Aumento dell'area cardiaca scoperta, versamento pericardico di piccola entità. *Cuore* globoso, alquanto aumentato nella sua metà destra; punta formata in egual misura dai due ventricoli. Aorta e arterie coronarie normali; apparati valvolari integri. Le cavità del cuore appaiono fortemente dilatate; attraverso l'ostio tricuspedale passa la punta di quattro dita. Presenza di trombi nei recessi dell'auricola destra.

Polmoni: Cavità pleuriche obliterate, polmoni fortemente aderenti; la sinfisi è però vincibile. Nella parte dorsale della doccia vertebrale destra esiste una sacca pleurica ripiena di pus. Arteria polmonare dilatata; a sinistra molte sue diramazioni contengono trombi recenti. I polmoni si presentano più piccoli, meno elastici e assai più consistenti che di norma, specie nelle loro parti medie e basali ove danno, a toccarli, impressione di un corpo duro-elastico simile a gomma. Al taglio sono assai resistenti; la palpazione e l'ispezione della superficie di taglio fanno rilevare l'esistenza di una estesa e avanzata sclerosi diffusa del parenchima, più grave nelle parti medie e inferiori. Numerosissime le strie e i focolai di fibrosi interstiziale, tutti non sporgenti sulla superficie del taglio. Pareti bronchiali cianotiche e ricoperte di muco. Linfoghiandole ilari tumefatte, non indurite, fortemente antracotiche. Nessun segno di tubercolosi spenta o in atto nè nei polmoni nè nelle linfoghiandole.

Nulla di particolare a carico del canal digerente; fegato e milza da stasi; reni pallidi, torbidi.

Diagnosi anatomica: sclerosi interstiziale dei polmoni, trombosi recente di rami dell'arteria polmonare sinistra; pleurite purulenta saccata a destra; dilatazione e ipertrofia del ventricolo sinistro; visceri da stasi.

Nelle sezioni istologiche dei polmoni si vide una estesa fibrosi a tipo diffuso, con addensamenti striiformi o a focolai, prevalente nelle parti medie e inferiori, e intercalata a piccole zone di enfisema; quasi dappertutto segni di un processo infiammatorio cronico (piccoli infiltrati parvicellulari, desquamazione alveolare, zone di metaplasia dell'epitelio alveolare e bronchiale). Inoltre, reperto singolare, aree di tessuto connettivo ialino. Corpuscoli dell'asbestosi e fibre di amianto sparsi dappertutto nel parenchima polmonare e anche nel lume di alveoli effettivamente, sebbene in misura alquanto inferiore che nel caso precedentemente descritto. Il tipo della fibrosi interstiziale e ancor più la presenza dei corpuscoli e degli agbi di amianto documentavano che la sclerosi polmonare, fenomeno evidentemente primitivo e determinante di tutta la evoluzione clinica della malattia, era da ricondursi all'inalazione di polvere di amianto durata soltanto sette anni e cessata ben 30 anni prima della morte.

Cercherò ora di mettere in evidenza, in modo estremamente sintetico, i punti delle due storie cliniche che offrono un interesse maggiore per la discussione:

Nel primo caso (A. M.) una operaia lavora per 30 anni a fabbricar corde di amianto respirando un'atmosfera ricca di polvere di questo minerale. Durante questo periodo si sviluppa, con grande lentezza e senza dar segni di sè, una fibrosi polmonare. In occasione di un episodio influenzale la fibrosi si rivela apparendo, quasi a un tratto, di una notevole gravità. L'operaia, che fino allora aveva regolarmente lavorato, diviene in pochi giorni un'invalida. Brevità estrema di respiro, debolezza cardiaca e stasi nel grande circolo costituiscono i sintomi principali della malattia. Ricoverata in Clinica, gli esami clinici e radiografici rivelano una probabile fibrosi polmonare diffusa con bronchite basale e una dilatazione con insufficienza di cuore. Nonostante una energica terapia la malata si aggrava rapidamente e muore, 16 giorni dopo il suo ingresso in Clinica, 4 mesi dopo la cessazione del lavoro. L'autopsia dimostra una grave sclerosi interstiziale cronica dei polmoni di origine asbestosica, con dilatazione del cuor destro e visceri da stasi.

Nel secondo caso (N. T.) una donna lavora in gioventù per 7 anni in una manifattura di amianto ove è esposta all'inalazione di polvere del minerale. Durante questo periodo insorge una tosse secca e molesta, a volte di tipo accessuale, tosse che non abbandonerà più la paziente per tutta la sua vita. Essa lascia la fabbrica a 21 anno per sposarsi; già allora è una tossicolosa che soffre di frequenti raffreddori e bronchiti. Pur tuttavia essa può attendere a lavori faticosi, ma a 35 anni, dopo una bronchite asmatiforme più grave ed acuta delle altre, compaiono i primi segni riferibili a una fibrosi polmonare; un po' di brevità di respiro, dispnea da sforzo, facile stancabilità. Viene dichiarata un'asmatica e curata come tale. La donna cerca un lavoro più leggero e vi attende per 2 anni; ma la tosse, la dispnea e la debolezza si fanno più insistenti e la costringono ad abbandonare ogni lavoro. Da 37 a 50 anni la sintomatologia si aggrava lentamente e progressivamente e ogni inverno, colle sue bronchiti e l'esacerbazione della tosse e della dispnea, diviene più duro da sopportare. Infine compaiono segni di scompenso cardiaco. La malata si fa ricoverare in clinica: qui si rileva un grave scompenso di cuore con bronchite diffusa, un versamento pleurico a destra e si sospetta una fibrosi polmonare. Ogni terapia è vana; la malata muore 36 giorni dopo il suo ricovero in clinica, 13 anni dopo l'abbandono di ogni lavoro, 30 anni dopo la cessazione della inalazione della polvere di amianto. L'autopsia dimostra una sclerosi diffusa interstiziale dei polmoni di origine asbestosica, una pleurite saccata a D, una grave dilatazione del cuor destro con visceri da stasi.

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Nella discussione di questi due casi è conveniente sgombrare anzitutto il campo dal problema diagnostico, il quale pone principalmente due quesiti:

1°) È esatta la diagnosi di asbestosi polmonare?

2°) L'asbestosi polmonare è stata realmente la causa della morte delle due donne?

Al primo quesito la risposta è data dall'esame istologico dei polmoni. Il tipo particolare della sclerosi polmonare a forma diffusa interstiziale, la prevalenza della sclerosi nei lobi inferiori e infine la presenza di innumerevoli fibre di amianto e corpi dell'asbestosi disseminati in tutto il parenchima polmonare, documentano in modo certo l'esistenza di una grave asbestosi dei polmoni del tutto sovrapponibile a quella che è stata descritta dal punto di vista anatomopatologico da *Mc Donald* [4], *Gloyne* [21], *Simson* [14], *Di Biasi* [28], *Wedler* [56] ecc. (per i particolari anatomici e istologici di questi casi v. il lavoro di *Mottura e Fagiano* [37]).

Anche al secondo quesito si deve rispondere affermativamente. Se dal punto di vista radiologico e clinico, come vedremo, potevano esservi dei dubbi sulla estensione e sulla gravità della fibrosi polmonare, dal punto di vista anatomico e ancor più istologico tale gravità era ben manifesta. Basta osservare per un momento qualcuno dei numerosi preparati istologici allestiti da *Mottura e Fagiano*, per convincersi della estrema riduzione della superficie respiratoria utile e del grave impaccio che le aree di fibrosi e di enfisema dovevano costituire alla circolazione del sangue nei polmoni. La causa contingente della morte è stata bensì, in entrambi i casi, lo scompenso cardiaco, ma esso riconosce la sua origine principale, se non unica, nella sclerosi polmonare. Infatti il cuore di entrambe le donne non presentava segno alcuno di una pregressa infezione reumatica nè del miocardio nè dell'endocardio e neppure manifestazioni di qualsiasi altro processo morboso capace di diminuirne la funzione o la resistenza; gli apparati valvolari, l'aorta e le coronarie erano integre, e infine la dilatazione era localizzata esclusivamente alla metà destra del cuore. L'unica spiegazione possibile dello scompenso di cuore era quindi quella di un grave aumento del lavoro del ventricolo destro a causa della fibrosi polmonare, aumento che a lungo andare era risultato eccessivo per la muscolatura di questa metà del cuore la quale, come è noto, è capace di ipertrofizzarsi soltanto in misura relativa.

Chiarito il punto essenziale, cioè che la asbestosi polmonare e le sue dirette conseguenze furono la causa della morte delle due donne, vediamo quali elementi d'interesse possono essere tratti dalla storia delle malate. Per una fortunata combinazione le due storie rappresentano due modi fondamentalmente opposti di decorrere della stessa malattia. Nel primo caso decorso silenzioso e asintomatico finchè la malattia, rivelatasi in occasione di una affezione intercorrente di scarsa importanza, si è manifestata quasi all'improvviso e ha condotto in breve all'exitus. L'operaia ha potuto lavorare praticamente priva di disturbi fino a pochi mesi dalla morte. Nel secondo caso decorso cronico con tosse e bronchiti a ripetizione e in

seguito affanno di respiro, fenomeni morbosi che hanno condotto ad una invalidità dapprima parziale, poi totale, durata parecchi anni. La malata ha dovuto cessare ogni lavoro 13 anni prima della morte.

Questi due tipi corrispondono a due modi diversi di decorrere della asbestosi, la quale venne a volte dichiarata malattia subdola e latente, a volte malattia accompagnata fin dall'inizio da tosse, bronchiti e dispnea molesta, a volte affezione costituita da un primo tempo di irritazione delle vie aeree superiori provocata dalla polvere presto seguito da un periodo di assuefazione più o meno lungo il quale infine trapassa nel periodo della malattia vera con i sintomi proprii delle fibrosi polmonari (*Merewether* [51], *Saupe* [52]). La morte sopravviene, secondo la maggior parte delle descrizioni, da poche settimane a qualche anno dopo la cessazione del lavoro polveroso.

Non vi è dubbio che queste descrizioni corrispondano a reali differenze nel decorso dell'asbestosi e non solo a insufficiente raccolta dei dati anamnestici o a maggiore o minor peso dato dai pazienti a disturbi, come la tosse e la dispnea da sforzo, ai quali non viene in genere attribuito un carattere di gravità; anch'io, che in questi ultimi mesi ho avuto occasione di osservare parecchi casi di asbestosi, ho visto operai con avanzate fibrosi e pochi o punti disturbi soggettivi e operai con fibrosi relativamente modeste tormentati da tosse insistente, brevità di respiro, dolori toracici, stanchezza e altri simili inconvenienti. Fra i due casi estremi vi è poi, come è naturale, tutta una serie di casi intermedi sia come numero che come intensità che come tempo d'insorgenza dei sintomi morbosi. Il perchè la asbestosi abbia un decorso a volte asintomatico, a volte accompagnato da un più o meno vasto corteo sintomatologico, è di difficile spiegazione. È probabile che ciò dipenda in primo luogo dal differente grado di reattività delle mucose e dalla facilità più o meno grande col quale viene liberato il riflesso della tosse. Viene comunemente affermato che la polvere di amianto, formata in parte di spicole e di frammenti aghiformi di fibre, è molto irritante per le mucose. Sono state descritte con una certa frequenza faringiti, tracheiti, bronchiti e perfino congiuntiviti da polvere di amianto (*Quarelli* [58], *Wedler* [58], *Bauer* [59]). Per contro è stato affermato non essere la polvere di amianto particolarmente irritante, meno della polvere delle manifatture di cotone. Viene a questo proposito citato il caso di un operaio che non poteva sopportare senza gravi accessi di tosse la polvere di cotone, mentre tollerava senza alcun disturbo la polvere di amianto (*Merewether* [51]).

Come è noto il rivestimento dei bronchioli terminali, dei bronchioli respiratori e degli alveoli, ove iniziano e donde procedono le alterazioni fibrotiche dell'asbestosi, è privo di terminazioni sensitive, cosicchè la tosse deve essere interpretata come espressione non della fibrosi, ma di una concomitante bronchite oppure di alterazioni pleuriche. La fibrosi interstiziale

di per sè può evolvere sino a gradi avanzati senza rendersi in alcun modo avvertita. Anche la brevità di respiro e la dispnea da sforzo non sono segni che decorrano del tutto proporzionalmente al grado della fibrosi; come è noto esistono individui che sopportano senza disturbi o quasi riduzioni anche ampie della superficie respiratoria e altri colpiti da dispnea e da senso di mancanza d'aria per riduzioni anche relativamente modeste. Infine anche il vario decorrere delle pleuriti secche, frequenti nell'asbestosi polmonare, può concorrere ad arricchire la sintomatologia morbosa soggettiva.

È quindi in ultima analisi la disposizione costituzionale alle infiammazioni della mucosa delle vie respiratorie e la facilità alla tosse, unita alla resistenza individuale di fronte alla diminuzione del parenchima polmonare funzionante, che determinano l'entità e la precocità dei disturbi soggettivi dell'asbestosi e quindi il modo di decorrere della malattia.

Noterò qui, per incidenza, che la tosse nell'asbestosi si presenta spesso, come nella seconda malata, ad accessi, specialmente al mattino al momento di levarsi dal letto, accessi che possono perfino simulare quelli della pertosse tanto da venir scambiati con essi; scarsi sono per contro nella letteratura (*Oliver Th.* [60]) accenni a episodi di carattere asmatiforme come quelli ai quali ogni tanto davano luogo la bronchite e la tosse pure nel mio secondo caso.

Anche il quadro clinico-radiologico, che le due pazienti hanno presentato durante la degenza in Clinica, si presta ad alcune considerazioni. Nonostante che, preso nel suo insieme, il quadro morboso possa apparire pressochè eguale nelle due malate, tuttavia possono essere riconosciute sia clinicamente che radiograficamente delle differenze significative ai fini della nostra discussione.

Anzitutto prenderò in considerazione la bronchite. La prima malata aveva tosse insistente, ma secca o con scarsissimo espettorato: all'esame dei polmoni numerosi rantoli in prevalenza *fini*, localizzati alle due basi posteriormente e lateralmente. Nella seconda malata tosse pure insistente, ma con abbondante escreato mucopurulento, rantoli e rumori bronchiali secchi sparsi su tutto l'ambito. Ecco qui due modi di presentarsi della bronchite, di cui solo il primo è caratteristico della bronchite asbestosica. La tosse secca con i fini rantoli basali è infatti frequentissima nelle asbestosi di una certa gravità ma non complicate, ed io mi sono potuto convincere in successive osservazioni su di un ampio materiale che la mancanza di questo reperto è un fatto eccezionale. Il tipo della bronchite presentata dalla seconda malata è invece banale e per nulla caratteristico dell'asbestosi (*Wedler* [66]); la malata, oltre che una asbestosica, era pure una bronchitica cronica in cui lo scompenso grave di cuore aveva, con la stasi polmonare, accentuato i fatti bronchiali. Noterò incidentalmente come la se-

conda malata abbia pure presentato sputi striati di sangue: questo fatto non è del tutto infrequente nelle asbestosi come in quasi tutte le malattie croniche dei polmoni, l'emorragia essendo quasi sempre cagionata dai frequenti ed estenuanti accessi di tosse. Questi sputi ematici hanno probabilmente contribuito, specie in passato, a far considerare come tisici molti operai affetti da asbestosi; un tempo, quando la asbestosi era ancora imperfettamente conosciuta, si riteneva infatti che la maggior parte dei lavoratori dell'amianto morisse per tubercolosi (*Scarpa* [61], *Collis* [62], *Hoffmann* [63]).

Un altro reperto frequentissimo nell'asbestosi, oltre alla bronchite, è quello della pleurite. I fini crepiti delle pleuriti secche sono difficili da distinguersi dai rantolini e dai crepiti, pur essi assai fini, della bronchite basale, cosicchè la diagnosi clinica di pleurite in questi casi non è facile; ma è un fatto che all'autopsia di tutti gli asbestosici si trovano ispessimenti e tenaci aderenze pleuriche e che molti di essi si lagnano in vita di dolori a tipo pleuritico localizzati prevalentemente alle regioni sopradiaframmatiche. Pure le nostre malate avevano ispessimenti e tenaci aderenze pleuriche; la seconda aveva in più una pleurite essudativa saccata alla base di destra. Le pleuriti essudative non appartengono al quadro dell'asbestosi; nel nostro caso la sua genesi è da riferire a fenomeni infiammatori pleurici o pleuro-polmonari favoriti dallo scompenso di cuore, perchè è conosciuta la frequenza colla quale nei cardiaci scompensati con ipostasi e bronchiti si formino modici versamenti pleurici a carattere essudatizio. Il pneumotorace parziale, visto alla radiografia, è dovuto alla penetrazione di aria nel cavo pleurico in occasione della piccola toracentesi praticata.

Un altro sintomo sul quale conviene soffermarci un istante è la febbre. Al loro ingresso in Clinica entrambe le malate presentavano qualche movimento febbrile che però non superava di solito, alla sera, i 38°. Solo sub finem vitae si sono osservate nella seconda paziente elevazioni febbrili sui 39°-39°,5. La asbestosi decorre di solito senza febbre; questa è sempre indice di qualche complicazione. Io credo che nei miei casi la febbre possa esser messa sul conto dello scompenso cardiaco, ricordando come spesso nei cardiaci scompensati con stasi viscerali, si osservino elevazioni termiche irregolari. Nella seconda malata anche la bronchite e la pleurite essudativa concorrevano certamente ai movimenti febbrili; le puntate osservate pochi giorni prima della morte sono probabilmente l'espressione dell'infettarsi dell'essudato pleurico e dell'istituirsi di piccoli focolai broncopneumonici che furono poi anche constatati all'autopsia.

Su tutti i sintomi finora elencati dominava lo scompenso cardiaco. Le malate erano più che tutto delle cardiache e come tali avevano cercato ricovero nella Clinica. In realtà, a prima vista, tutta la sindrome morbosa e cioè la bronchite con tosse, la dispnea, la cianosi, il senso di stanchezza, gli edemi malleolari, l'ingrossamento del fegato, l'oliguria, ecc. poteva

rientrare benissimo nel quadro di uno *scompenso* cardiaco puro e semplice. Sono state solo l'attenta osservazione clinica, la valutazione di alcune sfumature come quella della cianosi insolitamente spiccata, della dispnea eccessiva, di un quadro radiologico non interpretabile soltanto come polmone da stasi o bronchite cronica, assieme alla ricerca della causa del prevalente scompenso del cuore destro, che permisero di affermare nel primo caso e di sospettare nel secondo l'esistenza di una fibrosi polmonare.

Se l'esame autopsico non avesse messo in evidenza una grave ed estesa fibrosi dei polmoni, permettendo così di riferire sicuramente la dilatazione cardiaca e lo *scompenso* ad un intoppo cronico della circolazione del sangue nel piccolo circolo le malate sarebbero state catalogate fra i cardiopatici e la vera causa della malattia e della morte sarebbe rimasta ignorata. Ed è probabile che, come vedremo meglio in seguito, così avvenga per molti casi di asbestosi.

Abbiamo già visto come, all'infuori della sclerosi polmonare, mancasse qualsiasi altra causa che avesse potuto determinare la dilatazione del cuore destro e la sua consecutiva insufficienza e come quindi lo scompenso cardiaco si dovesse ritenere come diretta conseguenza dell'asbestosi polmonare. Il fatto che i due primi casi mortali di asbestosi descritti nel nostro Paese siano morti per insufficienza di cuore lascia pensare che questa complicazione sia piuttosto frequente nella pneumoconiosi da amianto. Infatti, scorrendo i protocolli dei casi pubblicati, si vede che in molti di essi la dilatazione e lo scompenso cardiaco furono riconosciuti già in vita o alla necroscopia: il caso di *Mills* [16], dopo aver lavorato 17 anni, morì per blocco cardiaco con una estesa fibrosi polmonare; quello di *Lynch* e *Smith* [15] morì per scompenso cardiaco; in quello di *Seiler* e *Gilmour* [12] l'autopsia mostrò che la morte era dovuta solamente a scompenso cardiaco consecutivo alla stasi cagionata dalla fibrosi massiva diffusa, senza alcuna altra infezione aggiunta. Nel caso di *Stock* [43] fu trovato ipertrofia e dilatazione cardiaca; in quello di *Egbert* [45] il ventricolo destro era dilatato, il cuore pesava 375 gr.; in quello di *White* [46] alla radiografia il cuore era « sicuramente dilatato ». Nel paziente della *Buresch* [18] la morte sopravvenne in seguito a insufficienza ventricolare destra: l'autopsia rivelò fibrosi polmonare, forte dilatazione e ipertrofia del ventricolo destro, modica ipertrofia del ventricolo sinistro; in quello di *Stroebe* [44] fu trovato ipertrofia e dilatazione di entrambi i ventricoli, ma più del destro (esisteva però una lieve insufficienza mitralica ed aortica). Dei due pazienti di *Saupe* [52] che morirono in uno « il cuore è più grosso che il pugno; il ventricolo sinistro è modicamente dilatato, il destro è notevolmente ingrandito, specie nel cono della polmonare, la muscolatura fortemente ipertrofica (spessore cm. 0,7) »; nell'altro il cuore destro era ipertrofico e dilatato. Delle tre autopsie riportate da *Wedler* [50], in due fu trovato il cuore destro molto dilatato.

La frequenza della dilatazione del cuore nella asbestosi polmonare è pure notata da *Lovisetto* [20], *Mussa* [30], *Bauer* [50], (das Herz ist durch die Ueberlastung oft verbreitet und in seiner Funktion geschädigt), *Holtzmann* [10], (che la spiega col fatto che secondo lui la fibrosi attacca di preferenza le parti dei polmoni vicini al cuore), *Lanza* [34], [54], *Pendergrass* [44], (in some istances, the cardiac silhouette is quite enlarged), *Shull* [49]. Non tutti questi Aa. sono però d'accordo sulla frequenza e la gravità dello scompenso cardiaco: alcuni, come *Lovisetto* [20], *Merewether* [31], *Haddow* [9] non lo nominano neppure fra le complicazioni o le cause di morte; altri infine come *Mc. Pheeters* [40] ritiene che l'asbestosi non impacci troppo il piccolo circolo e che quindi non vi sia ragione di un sovraccarico del cuore destro.

In generale coloro che non ammettono la frequenza e la gravità dello scompenso di circolo nell'asbestosi sono quelli che hanno compiuto inchieste in manifatture di amianto e hanno visitato operai idonei al lavoro, fra i quali sono in realtà rari i casi di dilatazione di cuore provocata dalla fibrosi polmonare. L'osservazione dei miei casi e la considerazione di quelli della letteratura mi hanno convinto che l'insufficienza di cuore è una complicazione assai tardiva, direi terminale, dell'asbestosi. Se non sopravviene nessuna infezione gli asbestosici resistono finchè resiste il loro cuore; quando questo cede, la fine è segnata a breve scadenza. Io ho calcolato che in un terzo circa dei casi nei quali è stato eseguito un controllo necroscopico la morte abbia avuto come motivo principale lo scompenso di cuore. Il caratteristico delle insufficienze cardiache degli asbestosici è che esse si instaurarono tardivamente, ma una volta manifestatesi, non vengono arrestate da nessuna, per quanto energica, terapia cardiaca. Così è stato nei miei due casi e in altri (*Saupe* [52]), nei quali è ricordato l'insuccesso della terapia digitalica e strofantinica.

Veniamo ora a considerare il fenomeno primitivo e più importante: la fibrosi polmonare. Nelle mie malate i segni clinici di essa si manifestavano soggettivamente nella brevità di respiro e nella dispnea, oggettivamente nella cianosi, nella diminuzione delle escursioni respiratorie, nella quasi totale immobilità delle basi, nella ipofonesi più spiccata nelle parti inferiori dei polmoni, nell'affievolimento del murmure vescicolare. Però, come ho già detto, durante la degenza delle pazienti nella Clinica la scena è stata talmente dominata dalla insufficienza di cuore che essa colle sue conseguenze si sovrapponeva ai segni propri della fibrosi, mascherandoli più o meno completamente. Le radiografie, che ci avrebbero potuto dare utili indicazioni se noi avessimo in vita sospettato l'esistenza di una asbestosi polmonare nelle due malate, non ci furono allora di decisiva utilità. Realmente io sono persuaso che nessun radiologo per quanto esperto in questioni di pneumoconiosi avrebbe potuto in base a quelle radiografie far diagnosi di asbestosi polmonare, tanto esse sono prive di elementi carat-

teristici. Eppure ora, dopo aver studiato le radiografie dell'asbestosi riportate nella letteratura e in special modo le belle riproduzioni di *Pendergrass* [64], di *Saupe* [52], [53] e di *Wedler* [56], e dopo aver eseguito personalmente radiografie a molti asbestosici, riconosco che le radiografie delle due malate rappresentano due frequenti espressioni radiologiche dello stadio finale dell'asbestosi.

Le due radiografie sono assai differenti l'una dall'altra: basta confrontarle un momento per accorgersi di ciò: nella prima seminio di ombre di piccole e medie dimensioni su tutto il campo polmonare, con addensamento delle stesse lungo i margini cardiaci e le regioni parailari; nella seconda opacamento pressochè totale e uniforme delle due metà inferiori dei polmoni, con note di enfisema in quelle superiori. In entrambe però, dove la lettura del disegno polmonare è ancora possibile, si riscontra la medesima nota fondamentale: ombre tenui, sfumate, striiformi o meglio reticolari, intrecciantesi in vario modo; non noduli veri, netti, delimitati e a sè stanti come quelli della tubercolosi miliare o della silicosi, ma piuttosto chiazze a contorni irregolari, prolungantesi in strie e confondentesi in parte nel reticolo; pseudonoduli, se si vuole, ma non noduli veri nel senso che noi diamo in radiologia a questo termine. Molti Aa. hanno dato buone descrizioni radiografiche dell'asbestosi polmonare e tutti insistono sulla tenuità e sfumatura delle ombre e sulla mancanza di noduli di una certa dimensione; nelle loro descrizioni si possono riconoscere due modi di presentarsi dell'asbestosi grave, corrispondenti agli aspetti presentati dai miei due casi: nel primo reticolo più o meno fine o grossolano di ombre striiformi e piccole chiazze irregolari più intense nelle regioni sovradiaframmatiche e attorno al cuore; nel secondo opacità e ombre a carattere indeterminato si elevano dal diaframma come nebbie o come nubi di vapore, oscurando più o meno completamente il terzo o i due terzi inferiori dei polmoni e confondendosi con l'opacità cardiaca, mentre le zone superiori del polmone sono ipertrasparenti per enfisema.

Deve essere notato che mentre le forme ben sviluppate, ma non ancora gravissime dell'asbestosi presentano immagini radiografiche in cui la grande intensificazione e la finezza del disegno secondario del polmone assumono aspetti fino ad un certo punto caratteristici, le forme gravi complicate da scompenso di cuore perdono quasi del tutto la finezza della trama e la delicatezza del reticolo e con ciò la maggior parte delle caratteristiche radiologiche dell'asbestosi. I gravi ispessimenti pleurici, la dilatazione del cuore con consecutiva compressione dei polmoni, la stasi polmonare e la bronchite cronica sovrappongono le loro ombre a quelle della fibrosi asbestosica per dare un quadro, come nei miei casi, nel quale può essere difficile anche la semplice diagnosi di una fibrosi polmonare.

A proposito della fibrosi noterò ancora che nella prima malata, clinicamente l'ipofonesi basale e la diminuzione del respiro e radiograficamente la velatura e le ombre reticolari e pseudonodulari erano maggiori a destra che a sinistra. Al riscontro autopsico si è infatti constatato che la fibrosi era più estesa e avanzata nel polmone destro. La prevalenza della fibrosi asbestosica a destra è segnalata da parecchi ricercatori: *Oliver* [60], *Cooke* [2], *Haddow* [9], *Merewether* [31], *Bohne* [48], *Bauer* [59]; essa potrebbe derivare dal decorso più rettilineo del bronco di destra e quindi dalle migliori condizioni anatomiche per la penetrazione della polvere nei polmoni; vi è però anche chi ha trovato la parte sinistra più spesso colpita della destra (*Pancoast* e *Pendergrass* [66]). In un gran numero di casi la fibrosi è egualmente ripartita fra i due polmoni; la mia seconda malata appartiene, sia radiograficamente che anatomoistologicamente, a questo gruppo.

Infine mi par degno di accenno il fatto che, nonostante l'estesa e grave fibrosi, non si sia mai ascoltato sui polmoni delle mie due malate un respiro soffiante o bronchiale, ma bensì solo un respiro indebolito con carattere di asprezza. Ciò è facilmente comprensibile pensando alle particolarità anatomiche dell'asbestosi, per le quali quasi mai si verificano le condizioni fisiche adatte alla produzione del respiro bronchiale; e difatti mentre nella fibrosi silicotica si ode assai spesso in corrispondenza delle grosse masse tumoriformi un respiro soffiante, nella fibrosi asbestosica un tale reperto è eccezionale; ad esso accenna solo *Wedler* [56], che lo riscontrò una volta in un caso particolarmente grave.

Fra le particolarità degne di interesse che i miei casi presentano deve essere pure ricordato che in entrambi non esistevano, nè clinicamente nè anatomopatologicamente, segni di tubercolosi, all'infuori della componente linfoghiandolare di un antico complesso primario, osservabile nel primo caso. Questo fatto mi spinge ad accennare, sia pur brevemente, ai rapporti fra asbestosi e tubercolosi.

È nota la grande frequenza colla quale la silicosi si complica con la tubercolosi. Secondo studiosi stranieri degni di fede, il 60-70 % dei silicotici muore per il sopraggiungere dell'infezione tubercolare; in Italia vi è motivo di ritenere che questa percentuale sia ancora maggiore. Le osservazioni statistiche, cliniche, anatomopatologiche e sperimentali hanno dimostrato che la silicosi favorisce grandemente l'insorgenza della tubercolosi e che fra queste due malattie esiste una grande frequenza d'associazione. Per l'asbestosi, sclerosi polmonare grave quanto la silicosi, si dovrebbe teoricamente ammettere una eguale predilezione del bacillo del tubercolo. Tralasciando antiche osservazioni (*Scarpa* [61], *Collis* [62], *Lovisetto* [29], Boll. francese dell'Ispettorato del Lavoro del 1906 [66]) che parlano di vere stragi compiute dalla tubercolosi nelle manifatture di

amianto (osservazioni fatte in un'epoca in cui non esisteva igiene alcuna nelle fabbriche e in cui la confusione fra tubercolosi e asbestosi era resa inevitabile dall'ignoranza di quest'ultima forma morbosa) sta di fatto che si ammette oggi dai più, che fra asbestosi e tubercolosi esistano bensì rapporti ma che essi siano meno stretti di quelli fra silicosi e tubercolosi.

Gli autori inglesi (*Wood* e *Gloyne* [11, 37], *Merewether* [31], ecc.) i quali hanno dell'asbestosi la maggiore esperienza, affermano che essa, specialmente negli stadi avanzati, favorisce lo sviluppo dell'infezione tubercolare, pur riconoscendo che il pericolo è minore che nella silicosi. (The added risk of tuberculosis may be in asbestosis, it is less than that associated with silicosis. *Merewether* [36]). Le loro statistiche dànno una frequenza rimarchevole di asbestosi complicatesi con tubercolosi: in quella di *Wood* e *Gloyne* [37] su 100 asbestosici ricoverati in un Ospedale 21 erano affetti da tubercolosi; secondo l'Ispettorato del Lavoro inglese [67], nei casi mortali l'asbestosi è complicata con la tubercolosi nel 38 % dei casi, la silicosi nel 56 %. Per contro, secondo l'esperienza tedesca (*Bauer* [51], *Saupe* [52], *Wedler* [56]) basata per lo più su inchieste compiute fra i lavoratori delle manifatture di amianto, non esisterebbero stretti rapporti fra asbestosi e tubercolosi, benchè vi sia alcuno (*Bohne* [48]) che li affermi; gli americani, seguendo i risultati delle loro inchieste (*Lanza* [54], *Sbull* [49], *Dreessen* [55]) e l'opinione espressa da *Gardner* e *Cummins* [68] in seguito ai loro lavori sperimentali, non ritengono anche essi che la tubercolosi costituisca un grave pericolo nelle manifatture di amianto. Delle tre autopsie di asbestosici praticate finora in Italia (i miei due casi e uno del Dr. *Castagneri*, non pubblicato) in due non esisteva tubercolosi, nel terzo esisteva una polmonite caseosa. Indubbiamente il problema necessita ancora della raccolta di molto materiale per essere risolto, pur essendo possibile che da Paese a Paese varino quelle condizioni igieniche e ambientali che hanno un gran valore nella diffusione dell'infezione tubercolare.

Dopo aver commentato e discusso i punti salienti del quadro morboso presentato dalle mie due malate desidero riaffermare, come conclusione, il concetto che più volte è già affiorato nelle pagine precedenti: che cioè la diagnosi di asbestosi polmonare è una diagnosi normalmente difficile. Molti sono i fattori che concorrono a rendere difficile questa diagnosi, che teoricamente parrebbe assai agevole: i principali sono i seguenti: esiguità del numero degli operai che lavorano l'amianto in confronto al numero totale degli operai e quindi relativa rarità della malattia; appartenenza della maggior parte degli operai delle manifatture d'amianto alle industrie tessili e quindi indicazioni di anamnesi lavorativa (cardatore, tessitore, filatore) che appaiono innocue e non sollevano alcun sospetto di malattia professionale; tenuità delle alterazioni radiografiche

polmonari scopribili a volte solo con una tecnica radiografica perfetta; mascheramento della fibrosi polmonare asbestosica per opera della tubercolosi o dello scompenso di cuore; frequenza della morte degli asbestosici per malattie intercorrenti acute, in special modo polmoniti e broncopolmoniti; ignoranza fra i medici, e anche fra molti medici del lavoro, della esistenza di una grave fibrosi polmonare da asbesto; mancanza di sintomi o di segni veramente caratteristici o patognomonici di questa malattia.

Certo durante le inchieste nelle fabbriche (*Merewether* [31], *Lanza* [34], [54], *Mc. Pheeters* [40], *Shull* [40], *Dreessen* [55], *Lovisetto* [29], *Mussa* [30], *Gerbis e Ucko* [33], *Saupe* [52], *Wedler* [56]) è facile riconoscere un certo numero di asbestosici; ma altro è visitare gli operai di una manifattura d'amianto coll'intento di scoprirvi i casi di sclerosi polmonare, altro è far diagnosi di asbestosi al letto di un ammalato grave, sofferente di debolezza cardiaca o di broncopolmonite o di tubercolosi.

Alle difficoltà di una esatta diagnosi clinica hanno accennato già parecchi studiosi del problema dell'asbestosi: *Wood* e *Gloyne* [37] scrivevano nel 1934 che non vi è affatto da sorprendersi se solo dopo morte l'anatomopatologo scopre i segni di fibrosi polmonari rimasti indiagnosticati in vita. *Donnelly* [50] vide 15 casi di asbestosi i quali tutti, meno uno, erano stati diagnosticati da altri medici come tubercolosi. *Wedler* [56] ricorda che 23 operai delle manifatture d'amianto da lui studiate, affetti da asbestosi, si erano precedentemente fatti visitare più volte da altri medici per disturbi riferibili alla loro fibrosi polmonare; la diagnosi di pneumoconiosi fu fatta una sola volta! Il giudizio più frequente fu quello di pleurite, o bronchite o tubercolosi. Egli conclude che la diagnosi di asbestosi viene fatta solo di rado esatta, benchè le cose siano ora alquanto migliorate nel senso che gli operai sanno che il loro lavoro è pericoloso e ne informano il medico in occasione delle loro malattie.

Per questi motivi io ritengo che molti casi di asbestosi siano anche in Italia passati inosservati. Ciò vale da noi specialmente per la provincia di Torino, ove sono accentrate le principali cave e manifatture d'amianto e ove i casi di asbestosi, almeno a giudicare da quelli capitati quasi contemporaneamente alla mia osservazione, non devono essere del tutto infrequenti. Solo istruendo i medici sull'esistenza di questo particolare tipo di pneumoconiosi e convincendoli della necessità di raccogliere per ogni operaio malato una approfondita e minuziosa anamnesi lavorativa, noi potremo sperare che questa malattia squisitamente professionale possa essere nel futuro più frequentemente riconosciuta e diagnosticata.

Vengo ora, dopo aver discussa la parte clinica, al rilievo più importante che i miei due casi mi permettono di fare. Esso riguarda il problema prognostico dell'asbestosi e più precisamente quello della progressione della malattia cessata l'esposizione alla polvere d'amianto.

Tale problema è stato solo da poco tempo affacciato e si può dire non sia ancora stato risolto. Per la silicosi noi sappiamo ormai con sicurezza che essa, astrazion fatta dagli stadi iniziali, ha tendenza fatalmente progressiva anche dopo l'allontanamento dal lavoro polveroso; l'incertezza che invece ancora domina nel campo dell'asbestosi è dovuta alla sua scoperta relativamente recente e all'esiguità del materiale a disposizione adatto per un simile giudizio. *Lovisetto* [29] afferma recisamente che cessando l'inalazione della polvere, l'asbestosi si arresta nella sua evoluzione; *Shull* [49] ritiene che i casi lievi migliorino con l'allontanamento dal lavoro, ma che tale probabilità sia assai minore nei casi avanzati; comunque l'asbestosi non sarebbe almeno primitivamente una malattia progressiva. *Wood* e *Gloyne* [37] videro pazienti le cui condizioni rimasero stazionarie per parecchi anni dopo la cessazione del lavoro e perciò credono che anche quadri gravi di fibrosi possano essere compatibili con molti anni di lavoro leggero in ambiente sano e non polveroso.

Altri Autori sono meno ottimisti: per *Bohne* [48] l'allontanamento dal lavoro serve solo per i casi iniziali, ma non più quando vi è cianosi o dispnea; secondo *Sparks* [36] la malattia progredisce lentamente anche dopo la cessazione dell'inalazione della polvere; quando poi compaiono corpuscoli dell'asbestosi nello sputo, la malattia sarebbe decisamente progressiva. *Alwens* [51] è assai pessimista sulla prognosi dell'asbestosi; in un suo caso la malattia progredì decisamente anche dopo la cessazione del lavoro; *Pendergrass* [44] ritiene che quando l'asbestosi può essere diagnosticata radiograficamente, « the condition is likely to become progressive », e ciò a causa della fissazione delle strutture polmonari secondaria alla partecipazione della pleura al processo fibrotico. Infine alcuni, come *Lanza* [34], giudicano impossibile al momento attuale dire se l'asbestosi progredisce, come fa la silicosi, dopo la cessazione del lavoro polveroso.

Scorrendo la bibliografia, si vede come i periodi di osservazione dopo i quali i diversi ricercatori hanno pronunciato il loro parere sul problema della progressività dell'asbestosi siamo piuttosto corti: essi servono tutt'al più per un giudizio sul destino dei casi avanzati o gravi, ma non per affermare se l'asbestosi fin dal suo inizio abbia o no tendenza decisamente progressiva. Qualsiasi osservazione ben documentata e protratta per un lungo periodo di anni è a questo riguardo preziosa.

La mia seconda malata, che avendo lavorato per sette anni in gioventù con l'amianto è morta di asbestosi dopo trent'anni dalla cessazione del lavoro polveroso, rappresenta per la sproporzione fra durata dell'inalazione e intervallo fra il cessare di questa e la morte un caso del tutto eccezionale e unico nella letteratura.

La sua anamnesi ci permette di ricostruire la progressiva evoluzione della fibrosi polmonare, evoluzione che, come hanno dimostrato *Mottura* e *Fagiano* [37] collo studio istologico, era ancora in atto e vivace al mo-

mento della morte. Nei polmoni si sono trovati innumerevoli aghi di amianto, conficcati per ogni dove nel parenchima e poichè la causa della progressione della fibrosi risiede certamente nella presenza delle fibre di amianto nel tessuto polmonare, sia che si ammetta una loro azione chimica, sia che si ritenga, come oggi appare più probabile (*Mottura* e *Fagiano* [57]) la loro azione di natura essenzialmente meccanica, la evoluzione progressiva della malattia appare l'evenienza più logica e naturale. Ci si potrà al più nel caso speciale chiedere se la tosse insistente e continua e le bronchiti di cui la malata soffriva abbiamo potuto, coi bruschi e violenti movimenti che esse imprimevano alla cassa toracica e ai polmoni, aver accentuata l'azione meccanica delle fibre e così favorito un più rapido e intenso progredire della fibrosi.

Questo mio caso dimostra chiaramente che una asbestosi anche iniziale, cioè tale da permettere ancora per parecchi anni un lavoro pesante, può progredire anche al di fuori di ogni ulteriore inalazione di polvere di amianto fino a un grado tale da cagionare la morte e pertanto riveste una particolare importanza perchè, pur dovendosi tenere nel debito conto il fattore predisponente costituzionale, esso dà la prima convincente dimostrazione della natura primitivamente progressiva dell'asbestosi polmonare.

RIASSUNTO

Vengono per la prima volta in Italia descritti sotto l'aspetto clinico-radiologico due casi mortali di asbestosi polmonare.

Il primo riguarda una donna di 56 anni che inalò per 30 anni polvere di amianto. La fibrosi polmonare che ne conseguì decorse in modo asintomatico finchè, in occasione di un episodio influenzale, si rivelò improvvisamente nella sua gravità e condusse a morte dopo soli quattro mesi dalla cessazione del lavoro polveroso e dopo poco più dall'inizio della sintomatologia.

Il secondo concerne una donna di 50 anni che inalò polvere di amianto per soli sette anni, dai 14 ai 21 anni di età. La iniziale fibrosi polmonare che ne derivò fu fin dal suo insorgere accompagnata da tosse molesta e facilità alle bronchiti; tuttavia permise l'esecuzione di lavori pesanti per 15 anni, in capo ai quali risultò così progredita da costringere la malata a occupazioni leggere e infine a rimanere a casa per 12 anni, invalida e oppressa da tosse, bronchiti asmatiformi, dispnea. La morte sopravvenne trent'anni dopo la cessazione del lavoro coll'amianto e più di altrettanti dopo l'inizio della sintomatologia morbosa.

Il differente modo di decorrere della fibrosi polmonare e alcune particolarità clinico-radiologiche presentate dalle due malate hanno permesso di discutere alcuni aspetti del problema dell'asbestosi. Viene così ricordato come la fibrosi polmonare da amianto possa decorrere in una forma latente asintomatica e in una forma precocemente accompagnata da sintomi molesti e debilitanti; vengono ricordati i principali elementi diagnostici come la tosse, la bronchite, la brevità di respiro, le pleuriti secche, i segni fisici della fibrosi polmonare e viene affermata la frequenza dello scompenso cardiaco come complicazione terminale;

l'aspetto radiografico dell'asbestosi grave viene descritto nelle sue due varietà più frequenti e vengono ricordati i rapporti fra asbestosi e tubercolosi. È fatta infine presente la difficoltà della diagnosi dell'asbestosi polmonare specie negli stadi terminali, quando complicazioni di diversa natura possono mascherare sia clinicamente che radiologicamente i segni più caratteristici della fibrosi diffusa cronica interstiziale asbestosica.

L'interesse principale della pubblicazione è però conferito dal secondo caso illustrato, nel quale a un breve periodo di esposizione alla polvere di amianto seguì un periodo di trent'anni durante il quale si sviluppò una fibrosi asbestosica mortale. La lunghezza del periodo intercorso fra cessazione del lavoro con l'amianto e morte per asbestosi è del tutto eccezionale e unico nella letteratura, e costituisce la prima documentazione sicura che una fibrosi da amianto di grado lieve può progredire fino a un grado mortale al di fuori di ogni ulteriore inalazione di polvere; in altre parole che l'asbestosi è, al pari della silicosi per quanto con un meccanismo diverso, una sclerosi primitivamente maligna e progressiva dei polmoni.

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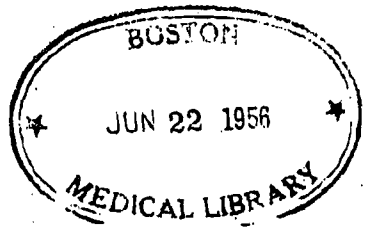

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