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This article is based upon data collected by the author from the examination of some 700 workers exposed to aluminum dust in German war industries. The examinations were primarily radiological and clinical but included also 125 vital capacity and sputum tests, and 100 blood counts. The chief complaints recorded in the personal medical histories of these workers were dry cough with stitch-like pains in breathing, dyspnoea, falling appetite and dragging gnawing pains in the abdomen. There were no pathognomonic findings on clinical examination. The blood counts exhibited a relative lymphocytosis and eosinophil cells were somewhat increased up to 5-10 per cent. Total leucocyte counts tended to be low, whilst red cell counts produced no significant changes. Blood sedimentation tests were also normal. Vital capacity tests showed a reduction in capacity. Four cases of spontaneous pneumothorax were noted, one of them fatal.

Radiologically the earliest sign consisted of focal shadows in the apical regions with, later, an increase (often one-sided) of the normal bronchial markings in the upper and middle thirds of the lung, giving a reticular appearance. There was also sometimes an increase in these markings in the lower lobes suggesting increased drainage to the hilum glands. Retraction of the diaphragm and distortion of the heart shadow were found. At a later stage the focal shadows tended to increase and become confluent (unfortunately the radiograms reproduced have been much reduced in size and do not bring out all the finer points mentioned).

One case came to autopsy. On microscopical examination the connective tissue elements of the lung showed coarse, branching hyaline collagenous fibres enclosing phagocytes containing fine and coarsely granular particles which differed from carbon particles by their jagged outlines. In parts of the lung these hyaline collagenous fibres were also found surrounding the walls of the alveoli, which contained phagocytes. These changes were most marked in the right upper lobe. (No illustrations are given.) The regional lymph glands exhibited a few of these dust cells lying chiefly in small groups. It is suggested that these histological changes result from a colloidal reaction between the aluminum dust particles and the tissue fluids.—*Bull. of Hyg.*

THE INJURIOUSNESS OF ALUMINUM DUST TO THE LUNGS.

Jöken and Eickhoff. *Arbeitsschutz*, pp. 102-109 (1942); *Chem. Zentr.*, vol. II, p. 923 (1942).

Aluminum dust presents an industrial hazard.—*Chem. Abstrs.*

EXAMINATION OF WORKERS IN AN ASBESTOS FACTORY.

A. Böhme. *Arch. f. Gewerbepath. u. Gewerbehyg.*, vol. 11, no. 4, pp. 433-452 (Nov. 10, 1942).

This is a record of clinical and radiological observations made in an asbestos factory during the years 1936-1937 and the year 1940. The factory handled

raw asbestos and manufactured a variety of asbestos products. It employed up to 500 workers. Of 132 persons examined, 29 per cent showed radiological evidence of asbestosis. Correlating these findings with exposure, it is stated that there were in workers with less than three years of exposure 5 per cent of cases, in those with 3-5 years of exposure 12 per cent, in 5-10 years 56 per cent, and in over 10 years 79 per cent. Five cases of pulmonary tuberculosis were noted. The clinical histories of these are recorded.—*Bull. of Hyg.*

FURTHER OBSERVATIONS ON OCCUPATIONAL CARCINOMA IN ASBESTOS WORKERS. A. Wels. *Arch. f. Gewerbepath. u. Gewerbehyg.*, vol. 11, no. 4, pp. 536-560 (Nov. 10, 1942).

This article is a record of two cases of asbestosis complicated by cancer of the lung. The first patient was aged 43, with an exposure of 11 years in an asbestos factory; death was due to squamous cell carcinoma. The second worker had had 30 years' experience in asbestos work and showed at autopsy an atypical epithelial cell new growth.

The author suggests there is a causal relationship between asbestosis and carcinoma.—*Bull. of Hyg.*

INVESTIGATIONS ON THE CAUSE OF INDUSTRIAL LUNG DISEASE IN THE MANSFELD COPPER MINES. J. H. Hellmers and H. Udluft. *Arch. f. Gewerbepath. u. Gewerbehyg.*, vol. 11, no. 4, pp. 480-502 (Nov. 10, 1942).

This article is a general survey of the mineralogical aspects of the industry with special emphasis on their relation to the dust hazard, but very little medical information is contained in it. The copper mines have been worked for a period of 700 years, and what is known as Mansfeld pneumoconiosis ("Mansfelder Staublungen"), has been the subject of a number of articles since 1934, some of the findings of which have been summarized (in *Bull. of Hygiene*, vol. 11, p. 282, 1936). The article consists almost entirely of very elaborate chemical and mineralogical analysis of the raw material of the mines and the dust produced, including, by way of comparison, analyses of a portion of one silicotic lung obtained from a Mansfeld miner. No details of morbid anatomy or of the histological condition of this lung, however, are given. The figures of these analyses are not suitable for abstraction. The dust encountered showed particles of numerous different substances including limestone, kaolin, silica, quartz, feldspar, mica, sericite and rutile. These varied in size from less than 2μ up to more than 50μ .—*Bull. of Hyg.*

ILLNESS CAUSED BY CADMIUM. Joseph J. Schifner and Henry Mahler. *Amer. J. Pub. Health*, vol. 33, no. 10, pp. 1224-1226 (Oct., 1943).

Two outbreaks of acute cadmium poisoning are described in which 7 persons became ill with acute gastritis

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severe cases than in the mild cases. These differences are statistically significant. The author finds that amongst the mild cases low plasma chloride concentrations were more frequent when vomiting had occurred than when there was no vomiting, and more frequent when the patients suffered from cramp than when there was no cramp. But the reviewer finds that these differences between the sub-groups of mild cases were not statistically significant.

Of the personal factors related to collapse the most important appear to be loss of acclimatization (following two to seven days' absence from work), disturbance of health, and unacclimatization. Emphasis is laid on the importance of deficient salt intake.

In the deeper levels of these mines, 7,000 to 8,000 ft., dry bulb temperatures reach 115° to 120°F., and wet bulb temperatures as high as 97°F. are reported. Collapse is liable to occur when the wet bulb temperature exceeds 90°F., and especially when it is above 93°F. The engineer should keep the wet bulb below the latter level. The benefits of air-conditioning are demonstrated by a striking fall in the incidence of collapse at Champion Reef Mine.—*T. Bedford.*

TOXIC NECROSIS OF THE LIVER FROM TRINITROTOLUENE.

Walter L. Palmer, Gerald S. McShane and William H. Lipman. J. A. M. A., vol. 123, no. 16, pp. 1025-1027 (Dec. 18, 1943).

In three cases of apparent trinitrotoluene poisoning with recovery, the evidence suggests that the toxic necrosis of the liver was severe in one, moderate in the second, and slight to moderate in the third.

A residual pigmentation and inflammation of the skin of the ankles was noted in the third patient in addition to the jaundice, anilism and gastrointestinal symptoms characteristic of trinitrotoluene intoxication.—*Authors' summary.*

CO-INCIDENCE OF PRIMARY CARCINOMA OF THE LUNGS AND PULMONARY ASBESTOSIS. *F. Homburger. Am. J. Path., vol. 19, no. 5, pp. 797-807 (Sept., 1943).*

In the medical literature there are now at least 19 known cases of asbestosis associated with primary pulmonary carcinoma. In 4137 autopsies at the pathology laboratory at Yale University School of Medicine, there were 45 cases of pulmonary carcinoma in the years from 1918 to 1938. Asbestosis was diagnosed 8 times, silicosis 17 times. Pulmonary carcinoma was found in 4 of the 8 cases of asbestosis and twice in the 17 silicotic cases.

Despite the high correlation of the foreign body asbestosis in the lungs with primary pulmonary carcinoma, this is a small series of cases and the value of these data is limited. Three cases are given in detail. The diagnosis of pulmonary asbestosis was diagnosed according to the following criteria: 1) fibrosis of the lung and 2) presence of asbestosis bodies.—*Stephen L. Mady.*

ASBESTOSIS AND PULMONARY TUBERCULOSIS. *R. Desmeules, L. Rousseau, M. Giroux and P. Richard. Laval méd., vol. 8, p. 217 (March, 1943).*

Asbestosis is an occupational disease of the lungs due to inhalation of asbestos dust. It is characterized by the transformation of normal lung tissue into fibrous tissue. Symptoms appear usually five to seven years after the beginning of the exposure. The presence of asbestos bodies in the sputum is a proof for the existence of pulmonary asbestosis. Both asbestosis and tuberculosis were seen in 8 workers exposed to asbestos dust for five to twenty years. Three of these cases are described, in whom the asbestosis was masked by tuberculosis and where the history and the presence of the asbestos bodies permitted the diagnosis.—*Am. Rev. Tuberc.*

ASBESTOSIS AND PULMONARY TUBERCULOSIS. *R. Desmeules and M. Giroux. Laval méd., vol. 8, p. 227 (March, 1943).*

The case history of a forty-four year old man is given who had worked for twenty years in an asbestos mill. After ten years he had developed productive cough which increased gradually. Only a short time prior to his admission to the hospital the patient started to run fever and had pains in his chest. The diagnosis of ulcero-fibrous tuberculosis and asbestosis was made. Tubercle bacilli and asbestos bodies were found in the sputum. The autopsy confirmed the clinical diagnosis. Besides the ulcero-fibrous tuberculosis, diffuse peribronchial and perialveolar sclerosis with numerous asbestos bodies was seen. Radiologically and pathologically it could not be decided whether the tuberculosis preceded or followed the asbestosis. However, it is believed that the first symptoms of asbestosis appeared ten years before the symptoms of tuberculosis.—*Am. Rev. Tuberc.*

PULMONARY ASBESTOSIS. *L. Rousseau. Laval méd., vol. 8, p. 233 (March, 1943).*

The radiological picture of pulmonary asbestosis is not characteristic; it is usually similar to that of tuberculous fibrosis. The diagnosis is, therefore, often made only after asbestos bodies have been found in the sputum. Asbestos fibers may be seen in the sputa of individuals who do not suffer from asbestosis; asbestos bodies are due to a pathologic tissue reaction. Microscopical examination of the lungs of patients who have died from an intercurrent disease may show asbestosis, which had not been found on X-ray examination. It cannot be decided whether asbestosis favors the development of pulmonary asbestosis. The case history of a forty-two year old man with asbestosis without tuberculosis is given. The patient had been working in the asbestos industry for eighteen years. For two years the patient had been suffering from increasing cough, expectoration and dyspnea. Roentgenological examination showed mottling of both lungs. Sputa

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and gastric examinations were negative for tubercle bacilli. Asbestos bodies were found in the sputum.—*Am. Rev. Tuberc.*

ASBESTOSIS BODIES. *M. Giroux. Laval méd., vol. 8, p. 239 (March, 1943).*

The "curious bodies" found in the sputum of patients suffering from pulmonary asbestosis usually measure 24 to 50 μ by 12 to 24 μ but can be as large as 250 μ . They consist of a central fiber of asbestos which is covered by a golden-yellow substance containing iron. Rabbits, guinea pigs and one dog were exposed to inhalation of asbestos dust. Asbestosis did not develop in the rabbits nor in the dog. The guinea pigs showed, after several weeks, macrophages with asbestos fibers in their alveoli. Erythrocytes appeared in the alveoli, and hemoglobin derivatives containing iron could be found on asbestos fibers two to three months after the beginning of the inhalation of the asbestos dust. When a suspension of asbestos in olive oil was injected subcutaneously or into the testicles of guinea pigs no asbestos bodies were formed. However, if first one cc. of blood was injected and then the asbestos dust through the same needle, asbestos bodies could be found one month later. The presence of asbestos bodies in the sputum is believed to be a definite sign of pulmonary asbestosis.—*Am. Rev. Tuberc.*

BAGASSE DISEASE OF THE LUNG. *W. A. Sodeman and R. L. Pullen. New Orleans Med. & Surg. J., vol. 95, no. 12, pp. 558-560 (June, 1943).*

A case of bagasse disease of the lung is reported together with the findings at biopsy in an involved area of the lung. The picture appears to be that of an organic pneumoconiosis. It is evident that particles of bagasse may enter the alveolar regions and produce this reaction. The mechanism of the changes, and the nature of the stimulus set up by the bagasse dust to produce the cellular reaction, remain obscure.—*Jack Bronfenbrenner.*

LIQUEFACTION NECROSIS IN BILATERAL SYMMETRICAL CONGLOMERATE LESIONS OF ANTHRACOSILICOSIS OF LUNG: REPORT OF CASE. *B. J. McCloskey. Am. J. Roentgenol., vol. 50, no. 1, pp. 42-45 (July, 1943).*

It is the intention of the author to show that the correct interpretation of the roentgen appearance of necrosis and cavitation in the conglomerate lesions of anthracosilicosis may be very difficult. A case is reported, showing these lesions, uncomplicated by tuberculosis. The only known exposure to silica was when the man worked as a pick miner in a soft coal mine for a period of 12 years and there was no exposure for a period of 44 years before his death. The roentgenologic appearance of the necrotic areas was that of consolidation or pleural thickening. At autopsy each cavity was found to contain thick, black, viscid fluid, which showed no free silica on examination. Examina-

tion of the cavity wall and surrounding lung tissue showed a content of 0.25 per cent silica.—*Jack Bronfenbrenner.*

PNEUMOCONIOSIS AND WORKING CONDITIONS IN COAL MINES. *Brit. Med. J., no. 4316, pp. 395-396 (Sept. 25, 1943).*

The Committee on Industrial Pulmonary Disease has been unable to draw final conclusions on causation or differences in incidence of pneumoconiosis in the South Wales coalfield. Anthracite dust is more dangerous than that of bituminous mines. The most important measure in prevention is the reduction of dust breathed by the miners. To this end, the committee advocates a series of procedures which are discussed in this article: (1) maintenance of adequate ventilation, (2) use of water to prevent dust dispersion, (3) allaying of dust in roadways and at loading points, (4) reduction of shot-firing to a minimum, with fewest possible men exposed, (5) regulation of ripping processes, and (6) prevention of chill during spake journeys.—*Jack Bronfenbrenner.*

RADIUM POISONING AND ITS PREVENTION. *E. Neitsel. Z. ges. Schiess- u. Sprengstoffw., vol. 37, pp. 74-75, 94-96, 114-116, 132-135, 155-156. (1942).*

Various well known examples of radium poisoning are described. On account of its relation to calcium in the periodic table radium can replace calcium wherever found in the animal organism. From its point of fixation in the body radium exercises destructive effects upon the tissues, mainly by virtue of its disintegration products. In small doses the destructive effects are frequently not noticed until after long periods of time, sometimes as long as ten years. The physiologic effects of the several disintegration products are described, as are the various means of measuring the radioactivity of samples and the several routes by which radium or its emanations may enter the human body. One of these is the breathing of radium emanations. Charcoal, silica gel and Permutit were tried, without marked success, for absorbing these emanations. A detailed method is described for examination of urine, blood, etc., for radium emanations. Various diagnostic means and a quantitative method for estimating the amount of radium in the body and in cadavers is described. Various means are given for preventing radium poisoning as prescribed by the safety precautions of the Association of Chemical Industry in Germany. A review of the literature is included.—*Chem. Abstrs.*

TREATMENT OF LEAD POISONING. CONFERENCES ON THERAPY. *N. Y. State J. Med., vol. 43, no. 21, pp. 2079-2086 (Nov. 1, 1943).*

The discussion emphasizes the facts that ingested lead, especially in small amounts, rarely causes poisoning because of poor absorption, and that the greater

EXPERIMENTAL STUDY OF STIMULANTS IN THE PROPHYLAXIS OF HEAT SICKNESS. B. Schlegel and H. Büchner. *Klin. Wochschr.*, vol. 21, pp. 533-538 (1942).

Phosphate (Recresal), cola (Kola-Dallmann) and pervitin were studied in conjunction with the effect of heat on the human organism. Cola had no effect on heat tolerance. Under the action of pervitin there was an improvement in heat tolerance measured by the duration of the experiment and the subjective findings, but the circulation was not affected. The action depended rather on a psychic change of the "dead point." Pervitin is, therefore, not a suitable prophylactic. Tests with Recresal showed that phosphate can exert a definite action in the prevention of heat injuries, especially heat exhaustion, as measured by the prolongation of the duration of the experiment, the milder rise in the pulse curve and the improvement in the subjective findings. The observed effect was too small, however, for an effective prophylaxis. It was, without doubt, inferior to that of the adrenal cortical hormone studied earlier.—*Chem. Abstrs.*

INDUSTRIAL NUTRITION PROGRAM OF W. F. A. H. F. Klander. *J. Am. Diet. Assn.*, vol. 20, no. 6, pp. 382-384 (June, 1944).

The general objective of the industrial nutrition program is that of improving the health and efficiency of industrial workers through raising their dietary standards. This objective can be furthered by serving nutritious food under proper eating conditions within the industrial plant and by obtaining the active cooperation of labor groups in improving the eating habits of workers. A corollary to this objective is that of improving the health of members of the worker's family through the carry-over value of the knowledge he has acquired through the industrial nutrition program.

An outline is given of W. F. A. activities. The following recommendations are made both to labor and management for their consideration in setting up or improving their in-plant nutrition programs: 1) That management and labor become informed on the relationship between the worker's diet and his health, working capacity, accident rate, morale and absentee record, 2) that food be made available to industrial employees through in-plant feeding, 3) that the lunch

supply at least one third the daily protective food requirements, 4) that a well balanced plate lunch served at a reasonable price, 5) that food be available to all shifts, 6) that the lunch period be at least 30 minutes in length, 7) that consideration be given to between-meal feeding, 8) that an educational program on food and nutrition be conducted, 9) that a labor-management committee on food and nutrition be formed in the plant, and 10) that management employ a dietitian.—*Stephen L. Madey.*

NUTRITION IN INDUSTRY. F. M. Gatto. *J. Am. Diet. Assn.*, vol. 20, no. 6, pp. 384-386 (June, 1944).

Incentive to better nutrition may be created by offering "Victory Lunches" in factories, and incorporating desirable food items. Booklets on adequate nutrition may add incentive also. It behooves any physician concerned with industrial health to bear in mind the necessity of encouraging the best feasible dietary conditions and the appropriate handling, transportation, preparation and serving of food.—*Stephen L. Madey.*

MANAGEMENT CONTROL IN INDUSTRIAL CAFETERIAS. Quindara O. Dodge. *J. Am. Diet. Assn.*, vol. 20, no. 6, pp. 390-394 (June, 1944).

The plant cafeteria that meets the requirements of both management and workers today needs to be scientifically organized and administered by a cafeteria director with training and experience in the applications of foods, nutrition, and institution management.

The article is noteworthy for its concise coverage of the various problems of cafeteria management, including foods, menus, labor, cleanliness, waste, etc.—*Stephen L. Madey.*

LEARNING TO BE A NUTRITIONIST IN INDUSTRY. E. P. Robins. *J. Am. Diet. Assn.*, vol. 20, no. 6, pp. 386-390 (June, 1944).

The experience of a nutritionist in the acquisition of her learning are recounted. The experiences are not remarkable and bear little relation to any clinical study. However it is a difficult topic in which to be original.—*Stephen L. Madey.*

SYSTEMIC AND INFECTIOUS OCCUPATIONAL DISEASES; AND HAZARDS

REPORT UPON THE WORK OF THE MINERS' PHTHISIS MEDICAL BUREAU FOR THE THREE YEARS ENDED 31ST JULY, 1941. J. M. Smith, Chairman. *Union of South Africa. Government Printer, Pretoria. U. G. No. 18, 1944. Price 8s 6d. pp. 46.*

There are probably no more informative papers on silicosis published than those which emanate from this bureau. Certainly no other organization has access to a fraction of the cases studied, so that the industrial world has come to look upon their publications for the epidemiological facts on silicosis.

"The Bureau now has the clinical and radiographic records of over 177,000 Europeans and 48,800 Native laborers." In their 1935-38 report they correlated their radiographic readings with the pathological conditions of 1560 deceased miners. The policy of the bureau in getting all possible autopsies has been continued and the analyses of further autopsies are contained in the present report.

In so large a group, lung cancers are bound to occur. Taking together the 1935-38 and 1939-41 reports they state that "... it appears that primary cancer of

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ing is not more common in silicotic miners than in non-silicotic miners or in a similar body of adult males of the general population."

During the last three-year period they averaged 80,000 examinations annually and about 700 deaths. It is an interesting tribute to their carefulness and skill that they found only 3 cases of silicosis at autopsy which were missed in routine X-ray, and only one case certified by X-ray to be silicotic, and which proved not to be silicotic at autopsy.

The general prevalence rate of silicosis was 23.23 per cent in 1930; it gradually dropped to 15.31 in 1937 and to 18.67 in 1940. Inasmuch as they have shown elsewhere that working conditions have been improved continuously, one naturally wonders if the prevalence rates are not unfavorably influenced by the likelihood that the examiners are getting stricter in diagnosing silicosis. In favor of this criticism is the Bureau's showing that the mean duration of employment of new cases increased from 14 years to 19 years and 6 months from 1930 to 1940.

The only discouraging note is the tabular proof that acquired silicosis now proceeds just about as it did 15 years ago.

One of the most interesting tables is that which shows the subsequent history of primary and antero-primary silicosis. The evidence is strongly in support of the fact that there is, as yet, no treatment of any demonstrable merit.

From the standpoint of the administrative problem the final sentence of the report is most significant. It seems almost certain that, if there is to be any other appreciable improvement in the prevalence of tuberculosis amongst Native laborers on the mines, the introduction of mass radiography or mass miniature radiography will have to be resorted to at the initial examination." (Routine preplacement examinations of Native workers are not done although many such examinations are performed on random samples).—*Dr. Drinker*.

ALUMINUM COMPOUNDS FOR SILICOSIS. *J.A.M.A., (Queries and Minor Notes), vol. 125, no. 14, p. 1001 (Aug. 5, 1944).*

Is there anything to the claim that aluminum dust is an antidote for silica dust and that silicosis in workers can be prevented by treating them with aluminum dust?

ANSWER: Amply confirmed experimental evidence in Canada and in this country proves that inhaled silicic aluminum and amorphous aluminum hydroxide completely neutralize the toxic action of quartz on living tissue. Given prophylactically before the quartz tissue reaction, it will limit the process to simple phagocytosis with no subsequent fibrosis; given after quartz, aluminum and its amorphous hydrate will retard the development of silicosis and cause resolution of immature nonfibrous lesions. Treatment does not dissolve silicotic nodules that have already attained maturity. Experience in North America fails to con-

firm the German reports that aluminum produces any serious effects on the lungs. A limited clinical experience indicates that some cases of silicosis are relieved of symptoms by aluminum therapy. It remains to be proved whether or not certain nonsilicious materials often associated with quartz in industrial dusts may interfere with the inhibitory action of aluminum. Theoretical and clinical evidence suggest that they may and thus may explain some of the failures that have been reported.—*J.A.M.A.*

EFFECTS OF SOUTH WALES ANTHRACITE COAL AND OF PRECIPITATED AMORPHOUS SILICA UPON THE LUNGS OF MICE: *J. Argyll Campbell. Brit. J. Exper. Path., vol. 25, no. 2, pp. 46-55 (April, 1944).*

1. Anthracite coal dust does not increase the incidence of lung tumors in mice. The statistically significant increases observed in some of the present experiments were due to other substances present—silica, aluminum and alumina. A heavy dusting of the lungs with bituminous dust produces some increase in lung tumor incidence, but only of border-line significance. A moderate amount of alcohol does not affect the incidence of lung tumors.

2. As in man, anthracite dust in itself produces an increase of the fibrous (reticular) tissue strands of the lung tissue of mice, and is more active in this respect than bituminous coal. Neither anthracite nor bituminous coal in heavy deposit increases the fibrous tissue of the mouse's tracheobronchial lymph-nodes. The nodular type of fibrosis seen often in the human lung with anthracite coal is not seen in mice. Perhaps the time—duration of mouse's life—is too short.

3. Silica does not increase the fibrous (reticular) tissue strands of the lung of mice to the same extent as anthracite coal, but it produces marked fibrotic nodules in the tracheobronchial lymph-nodes. Its effects differing from those of anthracite coal make it doubtful whether the increase of fibrous tissue in the mouse's lung produced by anthracite is due to its silica content. It might be due to silicate. Alumina and aluminum do not influence the effects of anthracite coal upon the fibrous tissue of the lung.

4. Neither alcohol in the drinking-water in moderate amount nor inhalation of cigarette smoke have any effect upon the action of anthracite coal on the fibrous tissue of the mouse's lung.

5. Metallic aluminum and alumina do not inhibit the development of fibrotic nodules in the tracheobronchial lymph-nodes of mice. The tissue of these nodes, therefore, reacts differently from the lung tissue of rabbits.—*Author's summary.*

CONTRIBUTION TO THE DIAGNOSIS OF PULMONARY SILICOSIS BY THE INVESTIGATION OF SILICA IN THE BLOOD AND SPUTUM. *H. Oyanguren and E. Bartholin. Rev. med. de Chile, vol. 71, p. 464 (May, 1943).*

The diagnosis of silicosis has been confirmed by demonstration of silica in the sputum and blood of 40

individuals. Silica which is conveyed to the lungs by inhalation is taken up in greater part by the lymphatics and the remainder is expectorated. Silica interferes with the lymphatic circulation, producing a fibrous proliferation which eventually results in the inclusion of the particles themselves. But before this occurs the silica which is inhaled passes through the blood and may be excreted in the urine. Silica may normally be found in the blood but if the quantity is less than 8 mg. per cent as determined by the colorimetric method, it is believed to be of no clinical significance. Quantities from 12 to 20 mg. per cent are expressed as 1 plus, from 20 to 50 mg. per cent 2 plus, and above 50 mg. per cent 3 plus. Positive chemical reactions for silica in the sputum may be obtained in all cases of silicosis. The positive reactions in the blood correspond to the amounts of silica contained in the sputum and positive chemical reactions are found only in those individuals who have been exposed to silica-bearing dust. The specific chemical reaction used is that described by Oberhauser and depends on the reaction obtained with nitromolybdenate of ammonia. This may be used equally on the blood and sputum and is a more certain test than the spectrographic methods.—*Am. Rev. Tuberc.*

SIGNIFICANCE OF FREE AND CRYSTALLINE SILICIC ACID IN THE OCCURRENCE OF SILICOSIS AND TUBERCULOSIS, AND THEIR PREVENTION. K. W. Jöllen. *Arbeitschutz*, vol. 3, pp. 194-202 (1941); *Chem. Zentr.*, vol. 2, p. 2108 (1941).

Experiments on rabbits showed that the silicogenic and tuberculosis-promoting effects of an industrial dust depend on the amount of SiO_2 contained in it. Addition of Al silicate (Bulus alba) acts as an antidote.—*Chem. Abstrs.*

DIRTY APRONS AND FLOOR DUST ARE SPOTTED AS SILICOSIS HAZARDS. M. Reuter. *Ceram. Ind.*, vol. 42, pp. 54, 57 (Feb., 1944).

This is a brief account of the control of silicosis hazards by Buffalo Pottery Co. At the beginning of the survey the employees and their union were assured that silicosis is no cause for discharge, and that mild cases of silicosis are not aggravated by conditions controlled within an acceptable minimum. After a thorough dust survey, steps were taken to remove the dust hazards, including: (1) furnishing clean aprons daily; (2) daily scrubbing of floors; (3) machines to eliminate the hand batting-out operation; (4) wire gratings on finishing benches; (5) grated bench tops for special batting-out operations; (6) change of speed in exhaust fans; and (7) isolation of dusty processes.—*Ind. Hyg. Found.*

MINORITY OPINION ON SILICOSIS. O. R. Troje. *J. Alabama State Med. Assn.*, vol. 13, p. 313 (April, 1944).

Simple silicosis gives no symptoms. Diagnosis depends on X-ray and occupational history. There is

lack of evidence between X-ray evidence and clinical evidence. Contrary to established belief, the changes in the lungs of silicotics are not due to fibrous connective tissue. The so-called fibrosis is really due to scattered deposits of collagen which are laid down in various patterns. The collagen deposits are a defense mechanism and the late symptoms of dyspnea are due, not to capillary occlusion and intervascular changes as a result of collagen encroachment, than to loss of alveolar air capacity.—*Stephen L. Maday.*

PNEUMOCONIOSIS OF COAL WORKERS—A METHOD OF MEASURING THE HEALTH HAZARD FROM AIR-BORNE DUST. *The Coal Dust Research Committee. Eleventh Report.* (Hann, D. A., Chairman). *Monmouthshire and South Wales Coal Owners' Association* (Nov. 18, 1943).

In studies of the pneumoconioses, concentrations of air-borne dusts are usually expressed in terms of the number of particles present in unit volume of the dust cloud. The numerical concentration measured in a given dust cloud depends on the method of sampling and the microscope technique employed: different methods may give very different results, and unless the methods of sampling and of evaluation are taken into account, particle counts can give but a rough idea of the quantity of dust present. In the Medical Research Council's Report on "Chronic Pulmonary Disease in South Wales Coalminers: II—Environmental Studies" (See Book Review, *THIS J.*, 25:453, 1943) it was suggested that standards of maximum permissible dustiness should be based on the mass concentration of dust not exceeding 5 microns in size—since particles larger than 5 microns are probably of little significance in the causation of pneumoconiosis. Concentrations in terms of the weight of dust of a restricted range of particle size cannot be determined directly by the usual gravimetric methods; and in the Medical Research Council's report it was shown that, with the thermal precipitator as the sampling instrument, a close estimate of the mass concentration of dust not exceeding 5 microns in size could be obtained by counting only the particles not less than 1 micron in size.

The present report by the Coal Dust Research Committee of the Monmouthshire and South Coal Owners' Association makes no direct reference to the Medical Research Council report, but refers instead to "the findings of Medical Authorities," which indicate that it is the particles of 5 microns and smaller size that are of pathological significance. Any method put forward for the measurement of the health hazard should, it is said, be capable of giving a measure of the amount of free and total silica, and also the amount of dust below 10 or 5 microns in size.

A method is put forward for the routine estimation of concentrations of dust not exceeding 5 microns in size. First, a gravimetric sample is obtained by the Soxhlet thimble method, in which the dust contained

measured volume of air is entrapped by a paper filter. The weight of dust so caught is ascertained and then the mass concentration of the total air-borne dust is calculated. The dust is removed from the paper filter, weighed, mixed, and a known weight of it then taken for a sedimentation test. The sedimentometer consists of a glass cell through which passes a flat horizontal beam of light which impinges on a photoelectric cell. Photometric transmission values are measured: (a) when the cell contains clean alcohol; (b) when a weighed quantity (about 20 mgm.) of the dust sample has been added to the alcohol, and the particles are still in turbulent motion after stirring; and (c) after the lapse of the calculated time which will allow all particles but those smaller than 5 microns to settle below the level at which the light beam traverses the cell.

From the photometric readings the proportion (by weight) of dust below 5 microns is estimated, allowance being made for the fineness of division of the whole dust sample (as indicated by the "specific surface" of the dust). Since the mass concentration of the whole dust sample has been ascertained by direct gravimetric methods, the mass concentration of the dust below 5 microns can be estimated.

In the report it is admitted that certain criticisms may be levelled at the procedure recommended, and that only a few samples have been so evaluated, no direct statement of the probable error of the method is given. It is said that the probable error will be under 10 per cent, an error which, it is said, is quite small in comparison with those to which other methods at present used in Great Britain are liable. The procedure gives no information about the constituents of the dust. *Bull. of Hygiene.*

ASBESTOSIS AND PULMONARY CARCINOMA. H. W. Vedder. *Deut. med. Woch.*, vol. 69, no. 31/32, pp. 575-576 (Aug. 6, 1943).

The author has collected the published autopsy records on asbestosis from various countries and finds that there have been 14 instances of malignant disease of the lungs and pleura in 92 post-mortem examinations (16 per cent). This is in excess of the proportion of lung carcinomata in autopsies generally (2-6 per cent). Carcinoma as a complication of asbestosis occurred most frequently in males between 35 and 41 years of age and was generally in the part of the lung most affected by asbestosis. Development of the lesion was slow and corresponded with the length and intensity of the exposure (12-42 years). There was often a long interval between the cessation of exposure and the development of the cancer, which, in the author's view, must now be regarded as an occupational cancer.—*Bull. of Hygiene.*

THE ASBESTOS CORN. H. S. Alden and W. M. Howell. *Arch. Dermat. & Syph.*, vol. 49, p. 312 (May, 1944). Derwitz in 1930 directed attention to warts and corns on the hands and feet of persons who worked with

asbestos and described the discovery of an "asbestos needle" in the cornified layer of tubercles removed from the hands. Alden and Howell observed small painful "warts" or "corns" in 99 of 167 workers who used amosite, a natural form of asbestos, in their work in a navy yard. The workers who have these lesions complain of an original pricking sensation and ability to feel a small splinter-like foreign body. Rough attempts to extract this splinter-like body are usually unsuccessful, but with rubbing or with neglect the sensation tends to disappear. In about ten days a small hard cornlike tumor appears, which slowly grows, often attaining the size of a small split pea. Frequently a pinpoint black center, which defies extraction, can be observed in the excrescence. At this period the small cornified tumor is tender to pressure, as though a splinter were present, and interferes with comfortable work. Cornification becomes more pronounced, a small hard corn resulting, and the area does not become normal in appearance and feeling until the hard center plug is removed by instrumentation. These corns usually appear on the tips of the fingers and knuckles, most often occurring in the thick-skinned areas of the hands. X-ray examination does not reveal abnormal shadows. Biopsy specimens from 4 patients revealed no pathologic changes except extreme thickening of the surface epithelium with hyperkeratosis. The dermis, while fibrous, was essentially normal in appearance and did not present any evidence of inflammation. Despite diligent search, foreign bodies could not be found in the sections. Observations and history of symptoms imply, however, that a foreign body is at some time present in the lesions. There is no relation to asbestosis of the lungs. No satisfactory method of prevention can be offered.—*J. A. M. A.*

EPIDEMIC KERATOCONJUNCTIVITIS: A REPORT OF A CASE WITH MARKED SYSTEMIC MANIFESTATIONS. John J. Curry and Francis C. Lowell. *New England J. Med.*, vol. 231, no. 1, pp. 11-13 (July 6, 1944).

A case is presented that showed many of the signs and symptoms of epidemic keratoconjunctivitis.

The outstanding feature was the presence of severe depression with only mild ocular disease.

Serologic studies during convalescence showed the development of antibody against the virus of epidemic keratoconjunctivitis.—*Authors' summary.*

SURVEY MADE OF HEALTH PROBLEMS IN THE FUR INDUSTRY OF NEW YORK. Harry Heimann. *N. Y. State Indust. Bull.*, vol. 23, no. 6, pp. 217-220 (June, 1944).

Based on a medical study of 694 workers in the fur industry it is concluded that:

1. Traumatic affections of the skin appear characteristically among the occupational groups of fleshers, unhairers, hand-stretchers and nailers.

2. The cases of dermatosis appear among those workers who handle the dyed furs and are not related

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