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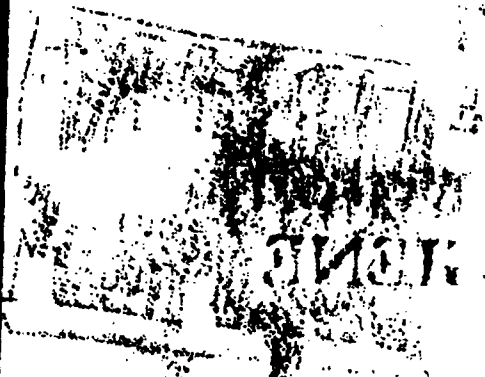
**ABSTRACT OF THE  
LITERATURE  
OF  
INDUSTRIAL HYGIENE**

**SUPPLEMENTARY TO  
THE JOURNAL OF INDUSTRIAL HYGIENE  
AND TOXICOLOGY**

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cover the wide range of practical conditions."—*Philip Drinker*.

3. THE EXAMINATION OF AIR-BORNE DUST IN COAL-MINES FOR THE EVALUATION OF THE POSSIBLE HEALTH HAZARD. *D. G. Skinner and J. Howard Griffiths*. pp. 166-175.

The authors suggest that the most important thing to know is the mass distribution in sizes below 5  $\mu$ . Assuming an adequate sample is obtained by a gravimetric procedure (see previous abst) "it appears likely that the most suitable means available for physical evaluation are the method of total turbidity measurement, to give a conventional value for specific surface, together with microscopical examination, the great merit of the latter being that in the hands of an experienced operator, semi-quantitative discrimination between coal and other particles is possible.

The pipette method, as modified by Fryer, probably deserves more attention than it has received up to the present. Whatever method is used, it is recommended that in view of the lack of medical knowledge, as much information as possible should be obtained with reference to the total mass concentration, the surface area of the total dust, and the mass and area of the particles below 5  $\mu$  in size."—*Philip Drinker*.

WEIL'S DISEASE: ANALYSIS OF 195 CASES IN ENGLAND. *J. C. Broom and J. M. Alston*. *Lancet*, vol. 2, p. 96 (July 17, 1948).

Broom and Alston reviewed 195 cases of leptospirosis which were diagnosed by serologic tests. The diagnostic criterion was the agglutination of a culture of *Leptospira icterohemorrhagiae* treated with formaldehyde solution which was observed by the microscopic technic described by Schuffner and Mochtar. Of 189 cases, 181 were in males. This probably reflects the greater risk of infection in certain predominantly male occupations and avocations and does not indicate a real difference in susceptibility. The proportion of females is much higher when chances of infection are equal for the two sexes. The prevalence of Weil's disease among coal miners, fish cleaners and workers in sewers and on the land has long been recognized; so has its occurrence after bathing in canals and stagnant rivers. The mortality rate was high in this group; of 114 patients 25 died, a rate of 22 per cent, compared to 9.4 per cent reported for Holland. The jaundice rate likewise was unusually high, the anicteric cases accounting for only about 10 per cent of the total. The authors believe that many cases of leptospirosis are missed, because they do not present the classical symptoms or the patient was not employed in one of the occupations recognized as carrying a risk of infection.—*J. A. M. A.*

CHEMISTRY OF CARCINOGENS. *William T. Saller*. *Occ. Med.*, vol. 5, pp. 441-465 (May, 1948).

Despite a wealth of data indicating carcinogenesis in man from crude mixtures, only three unadulterated agents have thus far been proved carcinogenic for man. These are (1) radiation, (2) betanaphthylamine and (3) arsenic. Chemical compounds which are outstanding carcinogens in rodents have usually proved ineffective in other species and have not been shown effective in man. Therefore, carcinogenic activity in rodents should not be used as a criterion of possible danger to man; nor should its absence in rodents be regarded as valid evidence of safety for human workers. At present, the only valid type of evidence of danger of carcinogenesis in man is carefully analyzed epidemiologic data. As a safety precaution in exposed workers, sensitive microchemical tests should be developed for each individual hazard to prevent more than minimal assimilation of possibly carcinogenic materials.—*Author's summary*.

ENVIRONMENTAL AND OCCUPATIONAL CANCER.—By *W. C. Hueper, M.D.* *Federal Security Agency Public Health Service, Supplement 209. Public Health Reports, 1948. U. S. Government Printing Office, Washington, D. C., 1949. 59 pages of text include 3 tables; 8 pages of references (For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C., Price, 20 cents).*

The text is based on Dr. Hueper's paper given at the Fourth International Cancer Research Congress in St. Louis, September, 1947, and on a series of lectures under the auspices of the Department of Occupational Medicine, Yale University Medical School, March, 1948. The author's mission was "to make available a concise and yet comprehensive presentation of current knowledge on the environmental cancer problem, for use not only by cancer specialists but also by the medical profession in general, public health officials, legislatures, social welfare workers, and members of Management, and of labor organizations."

From the standpoint of industrial hygiene, this is essentially a historical review. Dr. Hueper introduces a certain amount of dekrufism in an attempt to popularize the subject of environmental and occupational cancer; for example, in a statement such as "Carcinoma of the scalp is frequent among Mohammedans in Northern India who shave their heads with blunt razors, thereby causing frequent abrasions and infections," which may be true, there is an absence of critical supporting data. Throughout the text, the reader finds citations in a bibliographic vein, from the four hundred and eight references, without sufficiency of critical review.

Results of animal experiments with carcinogens not infrequently find little or no parallel in humans, but the general public is not sufficiently aware of this.

May 1949]

## ABSTRACTS

Thus, a serious danger lurks in the potential misinterpretations, which can arise in quotations from this Supplement by "legislatures, social welfare workers and members of management and of labor organizations," for whom the text is in part intended. Such material without critical screening, quoted from a public document will have the implied authority and reputation of the U. S. Public Health Service, to support it.—*L. S. Snegireff.*

CLINICAL ASPECTS OF OCCUPATIONAL CANCER. *W. C. Hueper. Occ. Med., vol. 5, pp. 157-165 (February, 1948).*

In viewing the development of the problem of occupational cancers, the fact stands out that there was a steady and rapid rise in the number and variety of occupational carcinogenic agents, of the operations with carcinogenic hazards and of the industrial cancers, paralleling the expansion and diversification of industrial activities during the last seventy-five years. The relative importance of industrial cancers in the general problem of cancer would be more clearly evident if all cases of occupational cancer were recorded and if physicians engaged in cancerology would take the time and effort to obtain complete occupational histories of their patients with cancer. The medical profession in general has been slow in the past in properly appreciating the causative role which prolonged unobtrusive exposure to extrinsic physical and chemical injurious agents might play in the production of many chronic and etiologically obscure diseases, such as arteriosclerosis, arthritis and cancer. Industrial physicians have a unique opportunity to make valuable contributions to the existing knowledge on the type and action mechanism of exogenous carcinogens in human beings. They are in the position to investigate the local and systemic reactions elicited by such agents, during the preparatory and manifestation periods of the cancerization process, and thereby add materially to the information so urgently needed for more effectively controlling cancer.—*Author's summary.*

COTTON-DUST DISEASE. *British Medical Journal, p. 1069 (Dec. 18, 1948).*

This article describes the symptoms and various forms of cotton-dust disease; principally mill fever, byssinosis, weaver's cough and the acute bacterial form of the illness which has been reported as due to the endotoxin of *Aerobacter cloacae*.

The article reviews the form of the disability but does not present anything new.

It is mentioned in the last section that the disease may be related to other diseases caused by contaminated organic vegetable material such as "farmer's lung" and "thresher's lung" and bagasse.—*Leslie Silverman.*

PNEUMONOCOONIOSIS FROM SYNTHETIC ABRASIVE MATERIALS. *Addaide Ross Smith and Arthur E. Perina. Occ. Med., vol. 5, pp. 396-402 (April, 1948).*

Evidence is presented that exposure to dust from synthetic abrasive materials in high concentrations may produce pneumoconiosis. Further pathologic studies of the behavior of "carborundum" dust in the lungs, including analysis of pulmonary tissue for free silica after the inhalation of "carborundum" are indicated. Possibly, the role of "carborundum" in the causation of nodular fibrosis has been incorrectly appraised.—*Authors' summary.*

MILKER'S ECZEMA. AN ANALYSIS OF FORTY-TWO CASES.

*Stephen Epstein. J. Allergy, vol. 19, no. 5, pp. 333-341 (Sept., 1948).*

1. Milker's eczema is a fairly common chronic eczema of the hands occurring in farmers or in other people who milk.

2. The recognition of milker's eczema is not difficult. The diagnosis is suggested by the clinical picture (Figs. 1 and 2), the history, and the course of the dermatitis.

3. Forty-two cases of milker's eczema are reported. Sensitivity to cattle was the most important specific cause. This sensitivity was demonstrated or suggested by positive skin tests to cattle antigens. Some patients gave positive immediate urticarial reactions; others, delayed tuberculin-type reactions; and others, positive patch test to cattle dander. Most of the cases reported are considered localized atopic dermatitis.

4. In three instances contact-type sensitivity to weeds was considered significant (Fig. 4). In general, however, milker's eczema differs from contact dermatitis from weeds by its clinical appearance and by the seasonal as well as age and sex incidence.

5. In a number of cases other factors were major or minor causes of milker's eczema.

6. Recognition of the causative relationship of this form of eczema to milking is essential for successful treatment.—*Author's summary.*

INDUSTRIAL DISEASES OF THE CHEST. *L. E. Hamlin. Indiana State Med. Assn. J., vol. 41, p. 1013 (October, 1948).*

Hamlin points out that it is most difficult to overcome the effect on a worker when once he has been told by his family physician that shadows observed in the roentgenogram of his chest are the result of silicosis, when actually there has been no significant exposure. Such interpretations frequently result in unjustified claims for compensation as well as unnecessary apprehension on the part of the employee. The author discusses the chest conditions the roentgen patterns of which may simulate silicosis. He stresses that unless there has been definite exposure to hazardous dust, as demonstrated by actual counts of respirable particles,

of free silica in the breathing zone of the worker, one should be extremely cautious about making an interpretation of silicosis even though the roentgenogram strongly suggests it.—*J. A. M. A.*

CHEMICAL CARCINOGENESIS: A REVIEW. *I. Hieger. Brit. J. Industr. Med., vol. 6, pp. 1-23 (January, 1949).*

This article is a review of a group of eighteen papers published in the British Medical Bulletin, Vol. 4, Nos. 5 and 6, 1947, covering the complicated subject

of chemistry of carcinogenic compounds. It is worth the real effort involved for the general reader to study this material in order to sense the amount of work done and the questions to be answered in the whole field of carcinogenesis. For the specialist and the experimentalist the critical judgment of the author in evaluating available studies must be vital. The chemistry of carcinogenic compounds studied is handled in considerable detail. As might be expected, no original work is presented in a review of this kind. The bibliography is full without being confusing.—*Harriet L. Hardy, M.D.*

## INDUSTRIAL TOXICOLOGY

AN EPISODE OF CARBON TETRACHLORIDE POISONING WITH RENAL COMPLICATIONS. *E. L. Morgan, M.D., J. P. Wyatt, M.D. and R. B. Sutherland, M.D. Canad. M. A. J., vol. 60, pp. 145-150 (Feb. 1949).*

In the opinion of this reader there cannot be too many reports of carbon tetrachloride poisoning. This reports the exposure of ten men in the course of a degreasing operation involving the use of the poison to clean a conveyor belt at three-week intervals. Three were in direct contact for a half a day, during which time 10 to 12 gallons of carbon tetrachloride were used, while seven were working in the same area but at an average of 25 feet away. The authors calculate that without change in air, the three men in direct contact were breathing 10,000 parts per million of carbon tetrachloride.

Of the three men engaged in cleaning the conveyor belt, one escaped with no symptoms, and one suffered nausea and vomiting for four days but no described after-effects. The third man became ill two days after the carbon tetrachloride exposure with headache, weakness and gastro-intestinal symptoms, and ten days later was hospitalized because of dyspnoea and a convulsive seizure. Five days later, the patient dies of pulmonary edema considered secondary to uremia. Autopsy revealed an interstitial pulmonary fibrosis, central hepatic lobular necrosis, as well as lower nephron nephrosis.

One of the workers 25 feet from the operation involving carbon tetrachloride had an episode of uremia with symptoms beginning the day after exposure, producing oliguria the second day. Marked disturbance in kidney function with nitrogen retention required hospitalization. Liver functions tests, except for hippuric acid synthesis, were normal. Six weeks after the onset of symptoms, the kidney function had returned to normal.

The authors are impressed with the predominance of renal disturbance in these two cases of carbon tetrachloride poisoning. They bring out the fact that on the basis of the single autopsy, the renal pathology is the "lower nephron nephrosis" of Lucké, "haemoglobinuric nephrosis" of Mallory, "the kidney of crush

syndrome," "kidney of traumatic uraemia," "tubulovascular syndrome" to quote synonymous names. This suggests an extrarenal cause of the renal picture seen in carbon tetrachloride poisoning. Perhaps the uremia is always present to a degree and is secondary to hepatic necrosis, but also perhaps Trueta's concept of changes in blood supply to the kidney is important.

The authors discuss the reasons for the variable response to apparently identical exposures to carbon tetrachloride. They also discuss the reversal of the uremia in one ill worker and the reasons for the lung findings at autopsy. There is no bibliography and no discussion of treatment.—*Harriet L. Hardy, M.D.*

URINARY EXCRETION OF MERCURY. *A. R. Smith and Samuel Moskowitz. Monthly Review (New York State Division of Industrial Hygiene & Safety Standards), vol. 27, pp. 45-47 (December, 1948).*

This article presents certain material accumulated in the study of workers in New York State, employed in a fur felt hat manufacturing establishment. The medical author divides 231 cases of chronic mercurialism discovered as follows: 87 had no signs of chronic mercurialism, 86 had positive signs and symptoms, and 58 made a group of suspicious cases of chronic mercury poisoning. Figures are given for mercury air exposure and urine content of mercury found in this same set of workers. The findings are correlated in an attempt to present evidence, if available, as to relationship between air and urine values and clinical status. The values obtained show that in a group, urinary mercury excretion is a rough index of exposure. The authors conclude that little can be learned about individual cases from this type of study, since correlation between exposure, excretion and medical diagnosis does not exist. By study of these data, Smith and Moskowitz believe there is suggestion that there may be some relationship between the failure to excrete mercury and the development of chronic mercury poisoning. The authors do not state how many air samples the figure given represents or how many urine samples per person were tested, nor is the length of exposure of these workers to mercury given. This

lymphocytes and plasma cells are also fairly numerous. In one case centrilobular necrosis of the liver has been seen. With chronic pulmonary fibrosis (granulomatosis), the lungs have been voluminous and emphysematous. Scattered diffusely throughout the lungs were many fine nodules. Enlargement of the right ventricle of the heart was constantly present in these cases. Microscopic characteristics include diffuse interstitial and nodular fibrosis of the lungs together with a peculiar granulomatous reaction. The granulomas which are typical of beryllium disease are formed in part within alveolar spaces by the organization of exudate, and in part within the septal and peritruncal connective tissues. They are comprised of central regions of fibrinoid material or granular debris, and the central regions are surrounded by a peripheral zone of fibrosis in which there is an infiltration of lymphocytes and plasma cells. The centers of the granulomas are occasionally occupied by giant cells of the Langhans' type. There are conchoidal bodies in most cases, sometimes within giant cells, sometimes lying free in debris or in fibrous tissue. 17 references and 15 plates.—*Nuclear Science Abstracts*.

**TROTYL AND TETRYL POISONING.** *Leo Novo. Wien. med. Wochschr., vol. 91, pp. 969-971 (1941).*

The symptoms of trotyl (trinitrotoluene) and tetryl (tetranitromethylaniline) poisoning were obtained in 147 munition factory workers examined clinically and in 491 others whose subjective symptoms were collected. The 2 compounds caused the same picture. The Takata reaction was positive in many cases, indicating the probability of liver parenchyma damage.—*Erich Hirschberg, Chem. Abstrs.*

**KEROSENE POISONING.** *Henry C. Thacher and Glidden L. Brooks. The J. of the Maine Med. Ass'n, vol. 39, 10, pp. 277-278 (October, 1948).*

The authors report four cases of kerosene ingestion in children. Three of the four showed x-ray evidence of pneumonitis and one of these also developed a slight pneumothorax. The authors note that signs of pulmonary involvement may not appear for twelve hours or more after kerosene ingestion. Prophylactic penicillin was used. All the patients recovered.—*Albert O. Seeler.*

**INDUSTRIAL FLUOROSIS OF FARM ANIMALS IN ENGLAND, ATTRIBUTABLE TO THE MANUFACTURE OF BRICKS, THE CALCINATING OF IRONSTONE, AND TO ENAMELLING PROCESSES.** *F. Blakemore, T. J. Barworth and H. H. Green, J. Comp. Path. Therap., vol. 58, pp. 267-301 (October, 1948).*

It is shown in this account that the contamination of vegetation is entirely superficial and not caused by fluorine compounds carried down into the soil by rain. The origin of the aerial fluorine is discussed in relation

to the industrial processes involved, and extent of damage related to distance from source and direction of prevailing wind. Representative clinical cases are recorded and the histology of skeletal changes is illustrated by microphotographs.

Urinary analysis was found to be a convenient way of diagnosing fluorosis, especially in subclinical cases where no dental signs or obvious skeletal changes were present, and the value of this simple method is emphasized for surveying areas of potential fluorine hazard in respect of either human or animal population. By correlating urinary excretion with fluorine values on rib bone removed surgically in an animal taken away from the source of fluorine ingestion, it was found that about half of the original skeletal fluorine still remained after 8 months and that, although marked clinical improvement occurred quite soon, urinary values remained far above normal (13 p.p.m.) in equilibrium with the very slow change of bone value in the later stages of skeletal reconstruction. A high urinary value may reflect either high current consumption of fluorine, or the skeletal storage of fluorine in the past, and close correlation in any given outbreak of disease between clinical symptoms, urinary output of fluorine and changing levels of pasture contamination can be expected.

Points of difference between fluorosis in its veterinary and medical aspects are briefly discussed in relation to social consequences and the hazards to human subjects and the economics of control of fluorine hazards. 28 references. 10 figures.—*Nuclear Science Abstracts*.

**BERYLLIOSIS.** *Stanley A. Wilson. Rhode Island Med. J., vol. 31, pp. 719-722 (December 1948).*

This paper is a brief resume of the disease known variously as "Pneumonitis of Be Workers," "Pulmonary Granulomatosis of Be Workers," and "Delayed Chemical Pneumonitis," but here called berylliosis. It includes a brief history, a listing of the most common symptoms, the pathological findings in acute cases, and a description of the lesion in so-called chronic pulmonary granulomata. Differential diagnosis is difficult and the conditions which may be confused with chronic berylliosis are many. No specific treatment has been found for the disease, though every known means has been tried to improve exchange of gases in the lungs.—*Nuclear Science Abstracts*.

**LEUKEMIAS FROM BENZENE.** *Enrico C. Vigliani and Giulio Saita. Med. lavoro, vol. 39, pp. 41-44 (1948).*

In one case of acute hemocytoblastic leukemia the worker had been exposed for 6 years to benzene vapors in a poorly ventilated workshop. The second case, a subacute leucopenic myeloid leukemia, occurred after 5 years in a rotogravure installation, where the ink contained 40 per cent benzene. Statistics showed that acute and subacute leukemias could be more convinc-

ingly ascribed to these working conditions than chronic leukemias. The contemporaneous existence of aplastic myelosis with this leukemia seems proven in many cases. Modern development of hematological study and especially the introduction of biopsy of hemopoietic organs have shown that acute leukemic cases from inhalation of benzene have a higher frequency in fatality than aplastic anemia. Many cases previously diagnosed as aplastic anemia in reality have been acute or subacute cryptoleukemic or leucopenic myelosis. Only 6 cases of chronic benzene leukemias have been described in the past 20 years as compared with several times this number of acute benzene leukemias. The number of statistical cases is still too small to come to definite conclusions as to the actual relations between benzene inhalation and leukemias.—*Chem. Abstrs.*

**A MUCH DELAYED CASE OF A PATHOLOGICAL CONDITION OF THE BLOOD, LYMPHOSARCOMATOUS IN TYPE, CAUSED BY BENZENE.** *J. Bousser, R. Neyde, and A. Fabre. Bull. et Mem. Soc. Med. Hopit. de Paris, Nos. 32/33, p. 1000, (1947).*

A most unusual fatal case is here reported. A man aged 42 had been at work for two years applying paint containing benzene, by a spray. Then he and 6 other workers were suspended from work at a periodic examination, when the blood picture was not considered normal. It was not, however, much affected and soon returned to normal on withdrawal to other work. He never resumed the exposure to benzene; but 3½ years later he was found to have generalized enlarged glands, without other signs or symptoms. After a biopsy a diagnosis was made of aleucaemic lymphomatosis, ascribed to benzene, since benzene was soon afterwards detected in the blood. There were 4 or 5 enlarged glands in both subclavicular spaces, in the axillae, and in the groins. The spleen was moderately enlarged; but not the liver. The man now complained of "indigestion" and could only take a little food, at frequent intervals, without vomiting. Still the blood picture was hardly disturbed. Rapid cachexia developed, with mild, but definite hyperglycaemia. The man became worse and died; in addition to the glands detected during life, a large lymphosarcomatous mass, invading the pancreas and stomach, was found in the abdomen. Benzene was found in appreciable amounts in both spleen and the glands. This persistence of benzene in the system for 3½ years, during which time it is initiating a pathological condition, is disquieting. Can it be found in others who have been exposed to risk after long periods? This case is the first instance of lymphosarcoma being ascribed to the action of benzene.—*E. L. Collis in Bull. of Hygiene.*

**CONCERNING THE INABILITY OF 2,3 DITHIOPROPNOL (BAL) TO PREVENT OR TO MODIFY THE ACUTE RENAL INJURY INDUCED BY URANIUM NITRATE.** *William de B. MacNider, Jesse C. Trott, Jr. and Margaret D.*

*Bruce. J. Pharmacol. and Exptl. Therap., vol. 94, pp. 262-273 (November 1948).*

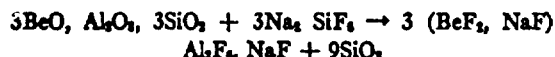
Uranium nitrate when given subcutaneously to dogs in the amount of 4 mg/kg induced a constant type of acute renal injury which was largely confined to the proximal convoluted segment of the renal nephron. However, no histologically demonstrable cell changes were found in the kidneys of dogs given 15 mg/kg of 2,3 dithiopropanol (BAL). But the use of BAL either as a prophylactic agent before uranium nitrate intoxication was experimentally produced or after, as an alleviating agent, increased the severity of renal injury. Furthermore, renal toxic action of uranium was greatest when BAL was used both before and after. It was concluded from this that BAL so influenced the epithelial tissue of the kidney as a sensitizing agent for uranium that the nephrotoxic action of this substance became more severe at its point of selective action and even spread to other segments of the renal nephron. 17 references.—*Nuclear Science Abstracts.*

**INTOXICATION BY BERYLLIUM.** *M. Ginabat. Arch. Mal. Prof., vol. 9, pp. 181-188, (1948).*

Eighty-five (85) cases of beryllium intoxication were observed over a period of 25 months. These cases resulted in a loss of 778 working days.

The manufacturing process involved consists of

1. Calcining a mixture of a double silicate of beryllium and aluminum, plus an equal weight of sodium fluosilicate at 750°C to form the double fluoride of beryllium and sodium—according to the following equation:



2. Later the double fluoride is selectively absorbed in water, crystallized out, and finally bagged. (No beryllium metal is manufactured.)

An extensive description of the cutaneous, ocular, respiratory, and general clinical manifestations of the alleged beryllium intoxication is given.

**Cutaneous.** Cutaneous manifestations were encountered in more than three fourths of the cases seen in the last two years. These lesions seemed to show a preference for the face, neck, and forearm, and varied in nature from a pale rose, papular dermatitis (often resembling measles) to small, indurated, chancre-like lesions which showed a tendency toward suppuration.

**Ocular.** Two-thirds of the ocular manifestations were characterized by hyperemic conjunctivitis and considerable edema of one or both eyelids.

**Respiration.** About 50 per cent of the cases exhibited some respiratory involvement. This involved especially an anterior rhinitis and tracheo-bronchitis. In contrast to the American observations, no cases of chemical pneumonitis were seen.

WITH RESPIRATORY COMPLAINTS. Hurley L. Malley, Leonard P. Long and Burgess Gordon. *Am. Rev. Tuberc.*, vol. 59, pp. 270-288 (March, 1949).

One hundred cases of anthracosilicosis with pulmonary symptoms and a long history of exposure to mine dust were studied. The degree of emphysema in these patients was estimated by comparing the residual air with the total lung volume. It was found that no relationship existed between the length of exposure and the degree of pulmonary emphysema. Likewise the degree of pulmonary emphysema could not be correlated with the stage of silicosis present as determined by x-ray.

It was found that the maximum breathing capacity gave a better indication of the degree of emphysema than did the vital capacity and the authors recommend the routine use of maximal breathing capacity measurements in the study of silicosis.

The authors discuss factors other than emphysema that may play a rôle in the impairment of pulmonary function in anthracosilicosis.—Albert O. Seeler, M.D.

ASBESTOSIS: VI. ANALYSIS OF FORTY NECROPSIED CASES. Kenneth M. Lynch and W. M. Cannon. *Dis. of Chest*, vol. 14, pp. 874-889 (Nov-Dec., 1948).

Findings in 40 autopsies on cases with asbestosis are reviewed. The authors believe that there is little progression of the disease following cessation of exposure to the dust. Asbestosis bodies persisted as long as twenty-seven years. Three of the forty cases of asbestosis had cancer of the lung. It is the impression of the authors that asbestosis does not predispose to tuberculosis.—Albert O. Seeler, M.D.

NEPHRITIS IN CHRONIC CARBON DISULFIDE POISONING, LEAD POISONING AND SILICOSIS. O. Gsell. *Ztschr. f. Unfallmed. u. Berufskrankh.*, vol. 41, pp. 55-65 (March, 1948).

Three cases are described in which the problem of a possible causal relationship between nephritis and an accompanying occupational disease is determined. The occupational diseases in the three cases reported were silicosis, carbon disulfide poisoning, and lead poisoning. In the first case it was decided that silicosis had neither caused nor aggravated the nephritis. The opinion in the second case is that the nephritis did not arise as a separate disease, but along with injury to other organs, as a result of severe poisoning by carbon disulfide. The chronic nephritis in the third case was regarded as the result of lead poisoning, especially in the absence of any other cause of renal injury.—*Indust. Hyg. Found., from Bull. Hyg.*

MANAGEMENT OF COMMON OCCUPATIONAL SKIN DISEASES. Nelson Paul Anderson. *J. A. M. A.*, vol. 139, pp. 912-916 (April 2, 1949).

The general practitioner faced with a dermatosis of possible industrial origin must be concerned not only

with the problem of treatment, but with the implications of the dermatosis for the employer and other workers, and with the medico-legal aspects. Criteria are cited for the recognition of the industrial origin of the dermatosis. Diagnosis is facilitated by a knowledge of dermatological hazards in specific industries, by a thorough physical examination, and by the appropriate and judicious application of patch tests. The clinical recognition of infectious eczematoid dermatitis as a concomitant of occupational injuries is discussed.

In the treatment of the industrial dermatosis, the importance of avoiding the use of irritant and sensitizing therapeutic agents is stressed; among these are the mercurials, sulfonamides, penicillin, local anesthetics, acridine dyes and various dyes and paints; phenol, resorcinol, and nitrofurazone ointments; and, probably, adhesive tape. Ultraviolet rays should be used only in suberythematous doses.

Prophylactic aids of value include protective clothing; mild soaps, detergents and cleansing agents; and protective ointments, which have some value.

Local topical remedies for dermatoses should be confined to soothing or, at least, innocuous agents, and overtreatment should be avoided. Specific therapy of acute, subacute and chronic dermatitis and of secondary pyodermias is discussed.—Bertrand C. Kriedte.

STANNOSIS. F. Barták, M. Tomáška, and O. Tomáček. *Časop. lkh. lask.*, vol. 87, pp. 915-920 (Sept. 3, 1948).

The authors have established stannosis as a pathological condition. Their claim is based upon the case of a workman aged 49 who had worked for 18 years in an enamel-ware factory, attending a furnace in which metallic tin was burnt to produce stannic oxide. Much dust was raised during the emptying of the furnace. He had suffered from asthma for many years, and the attacks had become worse before treatment was sought; his health was otherwise quite good. On X-ray examination of the lungs, many small sharply-defined shadows were seen, the appearance of which was unlike that of any recognized condition. Investigation of 16 other persons working in the same environment showed that 6 of them had a similar condition; they had been exposed to the dust of stannic oxide for periods ranging from 6 months to 25 years. Symptoms were absent. The original patient died of carcinoma of the stomach one year after coming under observation. Examination of sections of the lungs showed changes attributable to asthma, and also deposits of black pigment and infiltration with macrophages. Pigment was also found in the tracheal lymph glands. Tin was identified by chemical means in the lungs, lymph glands, liver and spleen. Deposits of stannic oxide were demonstrated in sections of lungs and lymph glands by means of micro-incineration: they were less in amount than those visible in stained preparations, the discrepancy probably being due to a deposit of carbon. The findings were confirmed by micro-incineration of tissues of rabbits and

guinea pigs inoculated with stannic oxide; the x-ray appearance could also be duplicated by placing stannic oxide upon the skin of the thorax.—D. J. Bauer, *Bull. Hyg.*

OCCUPATIONAL DISEASES OF THE LUNGS CAUSED BY INHALING BERYLLIUM. G. Corri re, P. Froiss , L. Richard, and L. Roche. *Arch. d. mal. profess.*, vol. 9, pp. 304-308 (1948).

The use of the rare metal beryllium is coming to the front for alloys with bronze, for glass permeably by X-rays, and in the manufacture of atomic piles. The fluorides and chlorides of beryllium freely form fine dust, and, if heated, emanate toxic fumes. The authors describe 9 workers exposed for 2 to 3 days to dust of a double fluoride of beryllium, and 2 exposed for a very short time to fumes of beryllium chloride. All experienced lung trouble and were admitted to hospital some months later, when their condition was most carefully studied. The symptoms included irritation of the eyes, nose and air passages, varying in intensity with individuals. The ocular and nasal irritation passed away in days or weeks; but then cough and dyspnoea appeared to get worse, keeping the patients from work. The dyspnoea followed effort and sometimes drifted into asthmatic attacks, sometimes at night. These troubles gradually diminished and disappeared, leaving behind a sensitivity of the air passages to vapours or fumes. In one or two cases x-ray showed shadows indicative of some discrete reticulo-nodular lesions, which in their turn tended to disappear in time. The distinctive features of these cases are the short exposure, the delayed onset of severe pulmonary symptoms, and final complete recovery after several months.—E. L. Collis, *Bull. Hyg.*

PULMONARY FIBROSIS DUE TO SILLIMANITE. H. G rtner and C. van Marwyck. *Deutsche med. Wochenschr.*, vol. 72, pp. 708-710 (1947).

This paper consists of a short record of the examination of portions of a lung obtained from a worker exposed to dust from the smelting ovens in the manufacture of corundum. A portion of lung about the size of a hazel nut was fixed in formalin, heated with 30 per cent. perhydrol, dried, and reduced to a fine powder. Another portion of lung was submitted to microscopical examination in the usual way. The powder from the dried lung was compared with the dust of the smelting oven by means of phase and r ntgenographic analysis and showed corundum, bauxite, amorphous silicic acid and mullite (a silicate allied to sillimanite). The features of the portion of lung examined histologically were those of a proliferating perivascular, peribronchial and perialveolar connective tissue formation together with the production, at these sites, of small irregular nodules which did not however, resemble the typical whorled nodules of silicosis.

Similar appearances were found in the lungs of rabbits experimentally infected with sillimanite, and the

authors conclude that the lesions in the human lung examined are attributable to this silicate, possibly associated with amorphous silicic acid. [This account suggests a histological lesion of the "dust reticulation" type described by Belt, Ferris, King and others in Parts I and III of Chronic Pulmonary Diseases in Coalminers, *Medical Research Council, Special Report Series Numbers 243 and 250* (this *Bulletin*, 1942, v. 17, 512; 1945, v. 20, 776).]—S. Roodhouse Gloyne, *Bull. Hyg.*

OBSERVATIONS ON THE CAUSES OF DYSPNOEA IN CHRONIC PULMONARY GRANULOMATOSIS IN BERYLLIUM WORKERS. Robert A. Bruce, Frank W. Lovejoy, Jr., George B. Brothers and Tulio Velasquez. *Am. Rev. Tuberc.*, vol. 59, pp. 364-390 (April, 1949).

See Abstract of the Literature, this Journal, vol. 31, p. 45, 1949.

THE PNEUMOCONIOSES. L. E. Hamlin. *J. A. M. A.*, vol. 139, pp. 909-912 (1949).

Hamlin emphasizes a point which is just beginning to become more widely recognized; namely, that all dusts inhaled into the lungs are not harmful. Many inert dusts appear to cause no lung damage at all even when inhaled in large concentrations over a long period of time. When such inert dusts are radiopaque, such as the oxides of barium, tin, and iron, they may cast nodular shadows on the chest roentgenogram, which to the unwary film interpreter mean "fibrosis." Increasing post-mortem evidence not only shows a complete absence of fibrosis with such an inert dust as iron oxide, but there is not even a mild inflammatory reaction resulting from its phagocytosis and deposition in the pulmonary lymphatics. The resulting change is nothing more than a pigmentation entirely comparable to the anthracotic pigmentation of all city dwellers. No functional damage has been done.

A classification of the pneumoconioses is presented by Hamlin which separates these inert dusts from those which are primarily allergic, irritative fibrogenic and systemically poisonous. This is not easy with some materials which may fit into several categories and others whose basic reaction is not well understood. For example, cadmium dust may be both irritative to the lungs and poisonous to the system after absorption into the general circulation. The byssinosis of cotton fibers and the bagassosis of sugar cane fibers may be allergic as well as infectious, the latter due to organisms inhaled with the fibers. Beryllium may be irritative to the respiratory tract as well as fibrogenic, or a combination of both. By and large, however, the classification appears sound and contributes materially to a better understanding of the pneumoconioses.

The classification reaffirms the generally recognized fact that only free silica and asbestos fibers, and possibly certain compounds of beryllium, are fibrogenic. Compounds of silica, other than asbestos, have not been proven to be reactive. Even asbestos now appears to be

less of a fibrogenic reaction in the productive silicotic sense than it is a bronchiolar obstruction and peripheral atelectasis due to a peribronchiolar inflammatory reaction. Short fibers which reach the alveoli appear to cause no fibroblastic cellular reaction after they have been phagocytized.

A recently recognized fact, that certain amorphous silicas may when superheated or calcined release free silica, was not discussed in this paper. A classical example is crude diatomaceous earth which has always been considered inert, but which when heated to 1,275° liberates a highly reactive free silica, cristobalite. Exposure to dust of the calcined product has caused widespread fibrosis in some workers so exposed. The little understood Shaver's disease due to the fusing of bauxite in the manufacture of the artificial abrasives may have a similar etiological basis.

Hamlin closes this excellent discussion of the pneumoconioses with the sound advice that the medical profession generally should inform itself of all phases of this complex problem before giving advice and opinions to workers with lung changes. Irreparable damage is being done every day by some physicians who by their well-intentioned but unstudied advice to patients are causing workers to leave their jobs which they can continue with complete safety. Modern dust control and hygienic practices have so reduced the dust hazard in most industries that it no longer is necessary to recommend change of employment in the majority of the so-called dusty trades. Firsthand investigation by these physicians in the plants involved is invited by most plant management, which will prove to be an enlightening and worth while experience.—O. A. Sander, M.D.

CHANGES IN THE LUNGS PRODUCED BY NATURAL GRAPHITE. H. E. Harding and G. B. Oliver. *Brit. J. Indust. Med.*, vol. 6, pp. 91-99 (April, 1949).

A brief description is given of the industrial uses of natural graphite, which is a crystalline carbon mixed with up to 10 per cent. of free silica and with other minerals. The greatest dust risk occurs in the grinding of natural graphite, and evidence is presented to show that this can lead to disabling and fatal pneumoconiosis. Marked changes in the lungs are, however, seldom seen in grinders who have less than twenty years' exposure. The results of three autopsies and some of the findings in other workmen are given, together with the results of some experiments on animals. The radiological and histological findings are similar to those found in South Wales coal workers.—Authors' summary.

SOLUBILITY OF INDUSTRIAL SILICEOUS DUSTS. P. F. Holl and H. V. A. Briscoe. *Nature*, vol. 163, pp. 334-336 (Feb. 26, 1949).

The solubility theory of silicosis has given impetus to the study of the solubility of siliceous dusts in water

and in various media. All the experiments have been made under conditions quite different from those in the body, especially in that in the body the liquid in contact with the dust is continually renewed. The results obtained have been quite variable, apparently depending upon small differences in the conditions of the experiment. The authors have studied some of these conditions and in one case have attempted to simulate the action of body fluids. They have demonstrated experimentally the following factors in the influence of carbonates on the solubility of silica, especially from flint: (1) calcium silicate is decomposed by carbon dioxide, resulting in a marked increase in the silica which passes into solution, shown by re-igniting a sample of calcined flint dust, converting much of the calcium sulfate into silicate; (2) carbon dioxide reduces the solubility of lime-free flint; and (3) calcium carbonate increases the solubility of flint. Under the authors' experimental conditions, the solubility of silica from purified flint was reduced from 8.6 to 0.2 mg. per 100 cc. by saturating with carbon dioxide, while from natural flint, the solubility was increased from 0.6 to 2.0 mg. They also obtained different results at different dilutions, which is to be expected from the above results. According to the solubility theory, cement dust is less harmful than flint dust because it is less soluble, as shown by several earlier investigators. However, the authors found that on repeated extraction of samples of calcined flint and of cement dusts with fresh portions of water saturated with carbon dioxide, flint shows a progressive decline in the rate of solution with each successive extraction, but the opposite is true of cement; on the fourth extraction, for instance, about four times as much silica is extracted from the cement as from the flint. In a future paper on experiments on dissolution of dusts in the body, a similar comparison will be reported.—*Indust. Hyg. Found.*

SILICOSIS AS VIEWED BY AN INTERNIST. W. Bernard Yegge. *Dis. of Chest*, vol. 14, pp. 550-567 (July-Aug., 1948).

Dr. Yegge reviews briefly the etiology, symptomatology, differential diagnosis, roentgenographic classification, complications and treatment of silicosis. Ten case histories serve to illustrate various aspects of silicosis.—Albert O. Seeler, M.D.

SILICOSIS AND PULMONARY FIBROSIS. A CONTRIBUTION TO THE TERMINOLOGY OF PULMONARY DISEASE DUE TO DUST. K. W. Jöllen and H. Gärtner. *Deutsche med. Wchnschr.*, vol. 72, pp. 531-534 (1947).

The authors discuss at length in the light of recent publications the present confusion in the terminology of the dust diseases, in the world's literature. Intensive methods of study, chemical, mineralogical and physical, have been introduced since the last international conference on silicosis met shortly before the war

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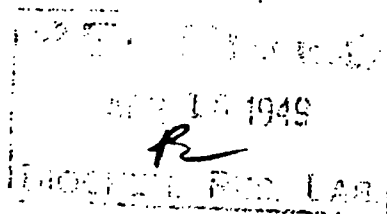
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