

November 1937

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4400 Fifth Avenue,
Pittsburgh, Pa.,
May 15, 1937

HWBB-0007829

A. News Items

236. BUREAU OF INDUSTRIAL HYGIENE FOR WISCONSIN. Surv. Labor Law Admin. 12, Mar. '37
A bureau of industrial hygiene is planned in the Wisconsin State Health Department, through cooperative efforts of State and Federal Health Departments. This bureau is to render technical service to the Industrial Commission, which is charged with the responsibility of enforcing rules and regulations, assuring safe and healthful working conditions in the state.
(The March, 1937, issue of the Survey of Labor Law Administration, including this and the following items with other material of interest, can be obtained on application to the Division of Labor Standards, U. S. Department of Labor, Washington, D. C.)
237. INDUSTRIAL HEALTH LEAFLETS. Surv. Labor Law Admin. 12, March 1937.
The Division of Labor Standards has prepared four additional Industrial Health Leaflets, dealing with cause and prevention of methyl alcohol, carbon tetrachloride and carbon bisulfide poisoning and carbon dioxide asphyxiation. Copies will be available for distribution in the near future. Those now obtainable include: (1) industrial skin diseases, (2) anthracosis, (3) arsenic, (4) carbon monoxide, (5) chromium, (6) mercury, (7) lead, (8) benzene poisoning and (9) silicosis. They are very brief, but present the extent of exposure and fundamental rules of prevention for employees and workers. They are obtainable from the Superintendent of Documents, Washington, D.C., at 5 cents each. The numbers given above are the numbers of the leaflets.
238. HEALTH OF GRANITE CUTTERS. Surv. Labor Law Admin. 12, March 1937.
The U. S. Public Health Service made an extensive study of health conditions among granite workers in Barre, Vermont, in 1928, and is now beginning a reexamination. The results of the present study should furnish additional important data concerning silicosis under controlled and uncontrolled conditions.
239. NATIONAL INSTITUTE OF LABOR, MEDICINE & HYGIENE FOR MEXICO. Surv. Labor Law Admin. 12-13, March 1937.
The Mexican Government has been requested by its Labor Department to appropriate funds for a National Institute of Labor, Medicine and Hygiene. A recent cooperative meeting has given impetus to a general movement to improve health conditions, including the establishment of that Institute.

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240. ILLINOIS CODES ON SAFETY AND HEALTH. *Surv. Labor Law Admin.* 14, March, 1937
The Industrial Commission has appointed committees representative of all interested agencies and is proceeding with the development of codes on safety and health. A policy committee has been formed to guide the work generally and to avoid duplication and overlapping of codes. The codes of other States and other safety information collected by organizations are being used freely as source material.
241. THE DUST STILL FLIES. *Eng. News Rec.* 118, 456 March 25, 1937
Last June New York State enacted a law to reduce the silicosis hazard in rock drilling and excavation, but the law is still inactive, awaiting action of the Industrial Commission in formulating a code for its application and administration. The main cause of delay is a protest by cement and gypsum interests over the technical definition of silica-bearing rock. The editor urges the end of quibbling over the definition, and action on the law, so that all concerned may know where they stand.
242. FIRST AIR CONDITIONED METAL MINE. *Mining Cong. J.*, 23, (March, 1937) p. 41
Installation of air conditioning with mechanical refrigeration in the Magma copper mines at Superior, Arizona, was announced by W. H. Carrier. The normal temperature at the depths of 3400 to 3600 is 95 to 100° with high humidity. A forced ventilating system is used.
243. MINERS' PHTHISIS IN SOUTH AFRICA. *Iron and Coal Trades Rev.*, 134, 454 (March 5, 1937), from *So. African Min. and Eng. J.*
The years since attention was first directed to silicosis and precautionary measures were undertaken have seen a continual decrease in its prevalence. Silicosis cases now number less than half of what they were in 1920 to 1923. There has been a slight upturn in the past year but it is no cause for alarm. One probable cause is the great increase in the number of miners. The increase amount of rock drilling is probably a factor. The reduction of dust from this source is now being studied.

B. General and Scientific Papers

In cases where we know or believe that reprints are obtainable from American and some other authors, their addresses are given.
A few papers under this heading and many under the next one are by French and German authors. In many cases reprints in the original language are probably available from the authors, but considerable delay will be involved, both in locating the author's address and in sending for the reprint. We suggest in order to avoid some of that delay, to order a copy of the periodical giving the name of the periodical (as abbreviated here), and the month or day of issue if given, or the pages included if not, from G. E. Stechert & Co., 31 East 10th St New York, N.Y. They will first quote prices if preferred, but it will probably average 50 cents or so for ordinary publications.

244. SILICA AND SILICATE SOLUBILITIES. Albert C. Titus. *J. Ind. Hyg. & Tox.*, 19, 138-146 (March, 1937).
In earlier investigations of the solubility of silica, the methods did not permit distinguishing the silica in true solution from that in the colloidal state, and the results were variable and often apparently too high. The author used a combination of earlier methods with ultra filtration and determined the silica by the colorimetric method of King and Stantial. He found 0.05 mg. silica in 100 cc. water when either pure silica or sericite was dissolved, and 0.1 mg. per 1 cc. from dolomitic chert containing 5% free silica. In blood serum, which contains normally 1 mg. silica per 100 cc., none of the materials mentioned dissolves sufficiently to increase the amount perceptibly. (Reprints are obtainable from the Division of Industrial Hygiene, Harvard School of Public Health, Boston, Mass.)

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245. THE OCCURRENCE OF SILICOSIS IN THE MANUFACTURE OF SILICON ALLOYS. Torsten Bruce, J. Ind. Hyg. and Tox. 19, 155-162, April 1937.
The author presents the results of an investigation of the presence of silicosis in two Swedish factories producing silicon alloys. In the process both silicon and silica vapors rise through the furnace and are condensed and given off as dust in considerable quantities. In one factory 8 out of 18 workmen examined, who had worked 4 to 8 years, were found to have silicosis, 3 being in the second stage. In the other factory, which was much older, 5 out of 26 examined had silicosis, only one being in the second stage, and the first clear case was after 14 years' employment. There were also two other suspected cases after 9 years' employment. The lower incidence in the older plant is evidently due to the use of an exhaust system which removes most of the dust which would otherwise settle in the plant. (In this survey and others made in Sweden, dust counts would have added much to the value of the reports. - Abstractor)
Reprints in English are obtainable from Dr. Bruce, Royal Seraphimer Hospital, Stockholm, Sweden.)
246. FURTHER STUDIES OF THE TOTAL PULMONARY CAPACITY AND ITS SUBDIVISIONS IN CASES OF PULMONARY FIBROSIS. Nolan L. Kaltreider, Walter W. Fray and H. van Zile Hyde. J. Ind. and Tox. 19, 163-176 (April 1937).
This paper supplements earlier work by some of the authors, showing the extent of the impairment of lung capacity in cases of fibrosis. In the present paper a correction for age is applied to the earlier results, and new cases are added. As fibrosis increases, the total and vital capacities are reduced, while the residual air and mid-capacity increase. If emphysema is present, the total capacity is not materially changed, but the vital capacity is reduced and the residual air is increased. The percentage of residual air referred to total capacity is proportional to the degree of dyspnea. Individuals with minimal fibrosis do not become worse when removed from the dusty occupation.
(Reprints may be obtained from Dr. Kaltreider, University of Rochester, School of Medicine, Rochester, N. Y.)
247. SILICOSIS AND RELATED CONDITIONS. Leroy U. Gardner. J. Ind. Hyg. & Tox., 19, 111-125 (March, 1937)
The first clarifying studies of silicosis in 1915 gave results that have been repeatedly confirmed by subsequent investigators, namely, that free silica is the only cause of silicosis, that the action of silica is chemical and not mechanical, and that pure silicosis is not infective, that it renders the patient more susceptible to tuberculosis, and that other dusts, including silicates (except asbestos) do not produce any type of fibrosis. The author defines silicosis as "a chronic disease resulting from the prolonged inhalation of particulate silica." The most distinguishing characteristic of silicosis is the presence of fibrotic nodules, found especially in the nodes of lymphatic vessels, and caused by irritation resulting from the presence of free silica. The only other cause of similar nodules is the tubercle bacillus; no other inorganic material (except asbestos) produces similar effects.
The complications that arise from silicosis include neither pneumonia, which seems to be retarded rather than assisted; nor heart and kidney diseases in spite of statements to the contrary based on isolated cases. Emphysema (inflammation of lung tissue) sometimes exists, but the most important complication is tuberculosis. It has been fully established, statistically and experimentally, that tuberculosis is much more common among silicotics than among normal persons. The tuberculosis of silicotics, however, exists in such a modified form that the distinguishing term silico-tuberculosis is generally applied. In most cases silico tuberculosis is difficult to diagnose, especially in the early stages, because the bacilli build up an extension of the fibrous tissue and other local reactions and are not expelled through the sputum, from which they are often absent until just before death. This increased susceptibility to tuberculosis has not been explained satisfactorily. Various theories

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have been presented, but none of them has been conclusively established, and much more work is necessary in that field. Diagnosis of silicosis, whether pure or complicated by tuberculosis, is difficult because there are few physical manifestations before the final stage. It must be based on x-ray examination and a complete occupational history, including that of exposure to dust and the concentration and composition of the dust. In a very general way it can be said that prolonged exposure to free silica dust in excess of 5 or perhaps 10 million particles of free silica is sufficient to produce silicosis, but the action of silica is so modified by the presence of other dusts that higher free silica concentrations may be harmless when other dusts are present. Much more experience will have to be acquired before any general standard of permissible dustiness can be established. The difficulty of recognizing both pure silicosis and silico-tuberculosis in their earlier stages, and therefore of beginning adequate treatment before it is too late, makes periodic and thorough examinations indispensable. The real solution of the problem is dust prevention. "The time is not far distant when dangerous concentrations of dust will no longer be permitted in American industries. They have been reduced to safe limits in some of them; they can be in all."

(Reprints may be obtained from The Saranac Laboratory for the Study of Tuberculosis, Saranac Lake, N. Y.)

248. QUARTZ IN INDUSTRIAL DUSTS AND DEPOSITS ON HUMAN LUNG TISSUES: X-RAY DIFFRACTION, CHEMICAL AND SPECTROGRAPHIC STUDIES. Victor Hicks, Olive McElroy and Mary E. Warga. J. Ind. Hyg. and Tox., 19, 177-186 (1937)

Several samples of industrial and atmospheric dusts were examined by x-ray diffraction and spectrographic methods. The methods gave somewhat different results, as they vary in the emphasis on different substances. Silicon was found by the spectrographic method in all the dusts, and quartz was identified by the diffraction method in most of them. Since quartz dust is so generally present in the air, it may be expected in lungs, whether silicotic or not. X-ray examination showed the presence of quartz in 5 out of 8 non-silicotic lung specimens examined, but an unidentified substance was present in others. The silicon content of some lungs in which quartz was thus found was as low as 1.4 mg. silica per gram of dried tissue.

(Reprints may be obtained by addressing this office.)

249. REPORT on 1936 SURVEYS OF MEDICAL SERVICE IN INDUSTRY BY THE AMERICAN COLLEGE OF SURGEONS. E. N. Newquist, Surg., Gynecol. and Obstet., vol 64, pp 403-406 (February 15, 1937).

This paper reviews 1,593 surveys of industrial establishments in the U. S. and Canada. The first point considered is the scope of adequate industrial service, with emphasis on medical examinations and supervision, proper care for industrial diseases, first aid and advice about occupational hazards. The second subject is the confidential nature of physical examination records. Civil courts have ruled that these are confidential. An industrial commissioner, however, may subpoena such information as has an important bearing on a claim for insurance. Only results of examinations expressed in codes should be filed with the worker's employment record. Finally, the problem of medical service for small plants is a special problem. These plants employed 5 1/2 million workers in 1929. The solution of the problem is "group service", i.e. a group of qualified physicians should provide service to groups of small plants.

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250. SPECIFIC TISSUE REACTION TO PHOSPHOLIPIDS. A SUGGESTED EXPLANATION FOR THE SIMILARITY OF THE LESIONS OF SILICOSIS AND PULMONARY TUBERCULOSIS. J. T. Fallon. Canadian Med. Assoc., J., vol 36, 223-228 (March, 1937)
The many similarities between the tubercle and the silicotic nodule led the author to search for a single substance that is produced in both silicosis and tuberculosis and causes the irritation leading to nodule formation. In both cases there are large accumulations of monocytes which degenerate with the production of phospholipids. There is therefore a probability that the phospholipids are responsible for the irritation and resulting fibrosis. This hypothesis was tested by several methods. After injection of silica into various tissues of rabbits the amount of phospholipids increased until the 16th week, when it began to decrease. Silicates introduced similarly produced only a moderate increase in phospholipid content of the lung and no fibrosis. Some of the phospholipid recovered from these animals was filtered to remove silica and then injected into other rabbits. In 4 weeks nodules very much like the silicotic nodules in the early stages were formed. They also simulated the lesions already demonstrated by Sabin, who similarly injected phospholipid from the tubercle bacillus. In the author's experiments the possibility remains that some silica in soluble form remained in the phospholipids injected. However, the theory is most attractive, and if fully substantiated answers one of the perplexing questions of the histogenesis and tuberculosis.
(Reprints are probably obtainable from the author, at Banting Institute, University of Toronto, Toronto, Ont.)

251. ETIOLOGY OF SILICOSIS. V. Reichmann. Med. Klin., 33, 226-229 (1937).
The author disagrees with other German investigators who claim to have observed silicotic changes resulting from inhalation of graphite and rust, on the ground that no roentgenological, pathological or histological proof has been presented. Of the 20,000 cases of silicotic lung changes he has seen, he found none that were not caused by several years' exposure to quartz dust. A check of a number of cases of silicosis, allegedly due to other causes, disclosed that there had been sufficient exposure to silica dust to produce the result. Arguments against the Jones sericite theory are also presented.

252. SILICOSIS IN SPAIN. A. Oller. Arch J. Gewerbepathol., 7, 383-387 (1936).
The author gives statistics on silicosis in the Spanish Rodalquilar gold mines, in which the gold is found in layers of quartz, but no sericite is present. The mines are dry drilled and have been in operation for 6 years. Ten workers died from silicosis in 1935, and 32 cases of silicosis were found in the 110 employees. In a ceramic factory 16 cases of silicosis were found among 350 workers. Hygienic improvements have been made in both industries.

253. ASBESTOSIS AND ITS PREVENTION. Windel. Arbeitsschutz, 9-16 (1937).
A survey of 11 German asbestos plants employing 385 workers disclosed 22 cases of asbestosis and 13 deaths. A short description of the clinical and x-ray pictures is followed by a careful description of the exhaust systems needed for certain processes with illustrations.

254. THE CARBON CONTENT OF ANTHRACOTIC LUNGS. E. Muller. Arch. f. Gewerbepathol., 7, 316-318 (1936).
A method for determining the carbon content of anthracotic lungs is presented. It consists primarily of pulverizing the tissue, extracting the fat with ether and alcohol, and then treating it repeatedly with concentrated hydrochloric acid. The method can be used also on other tissues.

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255. A GRAPHICAL METHOD FOR THE DESIGN OF EXHAUST SYSTEMS. A. Nutting. Heating, Piping and Air Conditioning, 9, 21-24 (January, 1937).
A series of charts has been prepared on which are plotted curves indicating basic relationships between air flow and resistance in exhaust systems. They are arranged in such a way as to solve any simple design problem directly and quickly. In any problem the static suction at the hood, the entrance loss into the hood, and the diameter of the exhaust pipes must be known.
(The above issue of the publication may be obtained from Keeny Publishing Co., 6 N. Michigan Ave., Chicago, price 50 cents or possibly 25 cents.)

256. INSURANCE COMPANY RELATIONSHIPS AND RESPONSIBILITIES IN COMPENSATION INSURANCE. A. D. Lazenby. Surg. Gynecol. and Obstet., 64, 393-396 (Feb. 15, 1937).
Insurance companies must undertake a definite program of accident and disease prevention. They must see that the disabled workman is restored to usefulness as quickly and completely as modern surgery and medicine can accomplish it, and that claims are justly and quickly paid. The importance of using the knowledge now available in industrial hygiene is stressed. The insurance company must cooperate actively if not actually participate in medical research and must apply the knowledge gained to the plants it insures. The author urges adequate training by medical schools in traumatic surgery and in occupational diseases, such as lead and arsenic poisoning and silicosis.

257. SILICOSIS: ITS CAUSE AND PREVENTION. C. O. Sappington. Mining J. Sept. 15, 1936
A brief review of the subject of silicosis, covering the nature of the disease, its detection and prevention, compensation laws, and preparation of defense of claims. (Reprints may be obtained by addressing Mine Safety Appliances Co., Braddock, Thomas & Moade Sts., Pittsburgh, Pa.)

258. NATIONAL SILICOSIS CONFERENCE. Summary reports submitted to the Secretary of Labor by conference committees, Feb. 3, 1937. U.S. Dept. of Labor, Division of Labor Standards, Bulletin No. 13.
The program of the Second National Silicosis Conference, at Washington on Feb. 3, attended by 150 representatives of the various interested groups, consisted of the presentation and discussion of the reports of the 4 committees on the following subjects:

- (1) Prevention of Silicosis through Medical Control;
- (2) Prevention of Silicosis through Engineering Control;
- (3) Economic, Legal and Insurance Phases of the Silicosis Problem, and
- (4) Regulatory and Administrative Phases of the Silicosis Problem.

This bulletin consists of a 51 page summary of the material presented in the reports, followed by a list of important references.
(The bulletin may be secured from the Superintendent of Documents, Washington, D. C., specifying the department, division and number as above, and enclosing 10 cents (no stamps).) A three page summary is also given in the Survey of Labor Law Administration, March, 1937, obtainable free from the Division of Labor Standards, U. S. Dept. of Labor.)

C. Scientific papers on industrial poisons and other subjects exclusive of dust.

259. A FURTHER NOTE ON INDUSTRIAL CANCER. W. Alwens and W. Jonas. Arch. f. Gewerbepathol., 7, 532-537 (1936).
A careful examination of the case history and autopsy of a chromate worker. Chemical examination of a piece of lung yielded 2 mg. of chromium in 300 g. of lung tissue

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260. THE BLOOD PICTURE IN X RAY AND RADIUM WORKERS. I. I. Kaplan and S. Rubinfeld. *Radiology*, 26, 47-55 (1936).
Blood counts on 22 technicians and clinicians working with x-rays and radium showed no grave effects of exposure. There was, however, some individual sensitivity. The most characteristic was a decrease in the polymorphonuclears and an increase in the lymphocytes, frequently accompanied by a sense of undue fatigue, and requiring greater protection or a vacation to restore the blood to normal.
261. ACUTE POISONING FROM COMBINED SOLVENTS. H. Schutz. *Arch. f. Gewerbepathol.*, 7, 452-467 (1936).
The author reports a number of cases investigated by the Berlin University Institute of Industrial Diseases, including poisoning by methylene chloride, benzene, benzine, butyl acetate, trichlorethylene and similar substances in mixtures. The symptoms were usually headache, dizziness, vomiting, loss of consciousness, watering of the eyes and sensation of choking. Cases of poisoning from lead dyes are mentioned in which xylene and methyl alcohol were used as solvents.
262. THE PATHOLOGY OF THE NEWER COMMERCIAL SOLVENTS. A. V. St. George. *Am. J. Clin. Pathol.*, 7, 69-77 (Jan. 1937).
A list of 23 solvents and the industries in which they are used, is given. It is difficult to determine the actual effect of each solvent in industrial use, on account of impurities that modify the effect of the pure substance. Most of these solvents irritate the mucous membrane of the conjunctival, the respiratory and gastro-intestinal tract, affect the central nervous system and sometimes the liver and kidneys. Practically all of them act on the hemopoietic system. In general the compounds of the aliphatic series usually produce a secondary anemia, while aromatic compounds generally effect more serious blood damage manifested by types of aplastic anemia with leucopenic reactions.
263. EXPERIMENTAL RESEARCH ON ACUTE POISONING FROM COMBINED SOLVENTS. F. Luce. *Arch. f. Gewerbepathol.*, 7, 437-451 (1936).
Experiments on mice to test the acute narcotic effect of mixed solvents showed that the effect is usually additive. In heavy concentrations, however, the effect of the mixture is somewhat less than calculated from the effect of the components. An exception is found in the case of mixtures of benzene and acetates, which are no more toxic in certain concentration ranges than the separate components. A graphic method for predicting the additive action is included.
264. METHYL ALCOHOL AND TOXIC METHYL COMPOUNDS. F. Flury and W. Wirth. *Arch. f. Gewerbepathol.*, 7, 221-226 (1936).
The special toxicity of methyl alcohol and derivatives from which it is liberated does not depend upon impurities, but is due rather to the slow oxidation of the alcohol into formaldehyde, which is a very active cell poison. The production of formic acid in the tissue is of only secondary importance.
265. FATAL OCCUPATIONAL BROMETHYL POISONING. W. Friemann. *Sam. v. Vergiftungs.*, 8, 31-32 (1937).
A case of bromethyl poisoning is reported in which a solderer repairing a tank for several hours, became stuporous and died after 4 days. The autopsy showed hemorrhages in the mucosa of the large intestine and bone marrow. The ganglion cells were also changed greatly, and bronchitis was present. The victim had evidently received a large dose in a very short period of inhalation.

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266. TWO CASES OF ANGINA PECTORIS RESULTING FROM INHALATION OF TRICHLOROETHYLENE. H. Gorbis. Arch. f. Gewerbepathol., 7, 421-426 (1936)
Two workers using trichloroethylene in a badly constructed degreasing plant fell ill with usual symptoms with heart pains as well. The elder one, 59, fell ill on the first day and died after 5 days. The younger man was disabled after 1½ years of work, but returned after 5 months and gradually improved.
267. PRACTICAL ANALYSIS OF SOLVENTS. SYSTEMATIC ANALYTIC METHODS FROM THE INDUSTRIAL HYGIENE VIEWPOINT. H. Weber. Schrift. des Reichgesundheitsamtes, No. 3. I. A. Barth, Leipzig, 1936. 40 pp.
The increasing industrial use of various solvents and recognition of the hazards involved in the use of many of them have led to the necessity of reliable methods for their recognition and analysis. Such methods are described in this book, including pre-examination to determine to what class of substances the particular solvent belongs, methods of analysis of each class, and tables of boiling point curves, refractive indices, characteristic reactions, limitations of methods, data on precipitation and other useful information.
268. LEAD HAZARD IN TYPESETTING. E. Lederer. Arch. f. Gewerbepathol., 7, 331-377 (1936).
Most of the lead hazard in printing shops arises from the fumes in the stereotyping department. In hand and machine typesetters the slight danger comes from dust rather than fumes. The lead in the urine is high, especially among the stereotypers, in 11 cases of the latter amounting to 30-60 micrograms per liter. Blood pressure was found to be slightly higher among printers than in the average population, but the small number of cases did not permit drawing fuller conclusions.
269. THE LIVER IN LEAD AND MIXED POISONINGS. H. Pernico. Arch. f. Gewerbepathol., 7, 538-593 (1936).
In persons unexposed to lead, the detoxifying action of the liver is shown by the difference in lead content of the blood entering and leaving the organ. About 2% of all cases of lead poisoning cause liver damage. The jaundice present in such cases is a hepatogenic one caused by injury to the liver parenchyma. The liver function test may be unsatisfactory since in one case all examinations were negative in spite of liver enlargement and jaundice. Experiments were carried out on guinea pigs poisoned with combined lead arsenic and lead trichloroethylene. In lead poisoned animals the action of both arsenic and trichloroethylene is very strong, especially the liver enlargement.
270. LIVER DAMAGE IN LEAD POISONING. K. Fellinger. Arch. f. Gewerbepathol., 7, 414-420 (1936).
In fresh cases of lead poisoning various functional disorders of the liver were shown. These injuries were found only rarely in old cases, indicating that they usually heal rapidly. However, the process still exists that a lasting liver damage takes place.
271. CONTRIBUTION TO THE STUDY OF THE TOXICOLOGY OF LEAD. CRITICAL STUDY OF LEAD CONTENT OF BIOLOGICAL MATERIAL BY THE DITHIZONE METHOD. F. Lom. 96 pp. Jouvo & Cie., Paris (1936).
The author presents his own modification of Fischer's dithizone method as the best for the determination of lead in biological material. The lead can be determined colorimetrically by comparison with known standards and is sensitive to 1/500 mg. Results of the application of the method to various animal organs are presented.

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272. HEINZ BODIES AND METHEMOGLOBIN FORMATION IN POISONING WITH AMIDO- AND NITRO-BENZOL COMPOUNDS. H. Freifeld, A. Schilova and R. Ludvinovsky. *Folia Haematol.*, 56, 333-342 (1937).
From the first development of methemoglobin anemia up to the appearance of the Heinz bodies takes only a few hours. Their number then falls rapidly over a few days. The authors advance the theory that the methemoglobin increases the combustion of carbohydrates, activates oxygen and strengthens the oxidizing power of tissue. If there is such a healing power, nothing is implied regarding the toxic action of these substances on the cells and on the nervous-vascular system.
273. DIAGNOSIS OF CHRONIC BENZENE POISONING. W. Freimann. *Arch. f. Gewerbepath.*, 7, 278-285 (December 1936).
The author calls attention to the difficulty of diagnosing chronic cases of benzene poisoning by the ordinary methods. He carried out estimations of vitamin C in the urine of men exposed to benzene and benzene vapors. The author hopes that future investigations will show the value of Vitamin C estimation in early diagnosis of benzene poisoning.
274. HEMATOLOGY IN BENZENE INJURY. R. Klima. *Wien. klin. Wochenschr.*, 50, 200-201 (1937).
The bone marrow and blood changes from benzene exposure are described. There is an early decrease in the number of leucocytes and thrombocytes and an increase in the lymphocytes and monocytes. The red blood cells also decrease in the absence of any signs of regeneration. The blood changes from x-rays and radium are also briefly described. Prevention is emphasized.
275. CARBON MONOXIDE POISONING. H. T. Berwald. *Military Surgeon.*, 79, 386-396 (1936).
The carbon monoxide hazard in army and navy planes, street traffic and garages is discussed. Street traffic is said to increase the carbon monoxide 2 to 4% at peak hours. Symptoms experienced in garage workers and in a well ventilated vehicular tunnel are described. Symptoms and treatment are discussed. The best treatment consists of oxygen-carbon dioxide inhalation with external heating. The author does not recommend methylene blue.
276. LIVER FUNCTION AMONGST MERCURY WORKERS. D. G. Tallenberg. *Arch. f. Gewerbepathol.*, 7, 305-315 (December, 1936).
Mouth diseases, mental disturbances, lymphocytosis, and presumptive evidence of liver pathology were noted in 17 otherwise apparently healthy hatters.
277. CHRONIC MERCURY AND AMALGAM POISONING. A. Stock. *Arch. f. Gewerbepathol.*, 7, 388-413 (1936).
The author presents data from earlier investigations on the very general occurrence of mercury in small quantities - in soil, water and food. The normal "mercury-free" person excretes 0.5 microgram daily in the urine and 10 micrograms in the feces. Animal research shows that after mercury inhalation ceases there is a rapid fall in the mercury content of the lung and the rest of the body but an extremely gradual one in the kidneys. After oral injection the storage by the kidneys is still more striking. Animal experiments also show that inhaled mercury is more harmful than when ingested. With the exception of the kidneys, the greatest quantities are found in the nose and in the olfactory and frontal lobes of the brain. A large part of the paper is devoted to clinical material on chronic poisoning, and on the development of hypersensitivity to mercury.

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278. INJURY FROM LIGHT METAL AND ITS SIGNIFICANCE IN INDUSTRIAL HYGIENE. W. Ehrlich Arch. f. Gewerbepathol., 7, 517-531 (1936).
The author has observed many cases of light metal injury, and attributes a large proportion of them to cutting by the sharp surfaces, causing infection from organisms adhering to the metal.
279. RESEARCH ON THE SIGNIFICANCE OF ALCOHOLISM IN THE DEVELOPMENT OF ARSENIC POISONING. E. Zimmerman and E. Remy. Arch. f. Gewerbepathol., 7, 486-496 (1936).
In animal experiments it was shown that larger quantities of arsenic are retained in the body when alcohol, in the form of beer, was also administered.
280. THE USE OF WHITE ARSENIC IN THE ENAMEL INDUSTRY. M. Dechigi. Arch. f. Gewerbepathol., 7, 468-476 (1936).
In the pottery industry arsenic trioxide is often used to produce a fine white glaze instead of the more expensive stannic oxide. Majolica tiles thus glazed give off arsine in the presence of mold when they are powdered or broken. The author urges protective measures against this source of poisoning.
281. ZINC VAPOR POISONING (METAL FUME FEVER), CLINICAL OBSERVATIONS AND THERAPY. F. Chrometzka. Deutsch. Arch. f. klin. Med., 179, 569-576 (1937).
The symptoms of zinc vapor poisoning include irritation of nasal mucosa, bronchitis, marked gastric irritation, and irritation of the mucosa of the urinary passages. Temporary nervous symptoms are frequent. The workers examined were electric welders, who are much more endangered than workers in zinc or metal foundries.
282. PROTECTION AGAINST TOXIC GASES IN INDUSTRY. J. D. Pratt. Paper to the Institute of Chemistry (South-Eastern Counties Section) February 19, 1936.
This is a preliminary report on an investigation made by the British Home Office and the chemical industry on the detection of toxic gases in industry. It has been customary to place mice in a closed vessel used for manufacturer or storage of dangerous products before a workman is allowed to enter, but this test is unsatisfactory because mice are not always more sensitive than men. In this investigation simple chemical tests were sought and tried for the most frequently used toxic gases, making the test quantitative whenever possible. For each gas it is necessary to determine the maximum allowable concentration into which the unprotected man can safely go, for a maximum period of 6 hours. Information on maximum permissible concentration and methods of testing for a number of gases is given. (Reprints are apparently not available, but when the official report appears in the near future this abstract list will give information as to how may be obtained.)
283. DETERMINATION OF SOLVENT VAPORS IN AIR. J. May. Arch. f. Gewerbepathol., 7, 510-516 (1936).
The interferometric method of determination of vapor concentrations in air is described.
284. A MICROCOLORIMETRIC METHOD FOR THE DETERMINATION OF TOLUENE. W. P. Yant, S. J. Pearce and H. H. Schrenk. U. S. Bur. Mines Rept. Investigations No. 3323, 1936.
Toluene in the liquid or vapor state is nitrated with fuming nitric acid, which is then diluted with water, neutralized and extracted with butanone. On addition of 50% potassium hydroxide a reddish-blue color develops in the butanone, proportional to the amount of toluene, which is estimated by comparison with standards. When benzene is also present, both substances can be estimated by using an already published method. An apparatus for preparing vapor air mixture is also described.

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(A free copy of this report can be obtained by addressing the Information Division, U. S. Bureau of Mines, Washington, D. C. Report of Investigations No. 3287, which described the method for benzene, can also be obtained from the same source, if a supply is still on hand.)

285. REPAIR OF DRUMS OR TANKS WHICH HAVE CONTAINED CELLULOSE SOLUTIONS OR INFLAMMABLE SOLVENTS. Home Office Form 1975, H.M. Stationery Office, London (November, 1936).

The purpose of this leaflet is to give warning of the dangers that arise in repairing vessels that have contained cellulose solutions or other inflammable liquids, and to describe the precautions that should be taken before repairs involving the use of heat is begun. These precautions include steaming and scraping out solid residues, or if scraping is not practicable, rinsing with a solvent until all solid residues have been removed, then steaming or immersing the vessel with boiling water for 20 minutes, while the interior is thoroughly swept out with air. In the case of large vessels, the inside air must be tested by a competent person, or, preferably, the person who enters must wear suitable breathing apparatus. Only tested and certified lamps should be used. Even in the case of trivial repairs these precautions should be taken, though they may take much more time than the repair itself. Caps or covers should be removed before heat is applied. A warning notice should be displayed in all places where repair work involving the use of heat is likely to be undertaken.

286. CONTROL OF CHROMIC ACID MISTS FROM PLATING TANKS. E. C. Riley and F. H. Goldman. U.S. Pub. Health Repts., vol 52, No. 6, 172-174 (February 5, 1937).

A cross-draft ventilation system has almost always been used to remove mists and fumes from chromium plating tanks, and this investigation was made to evaluate the control with reference to standards recommended in 1928 by Bloomfield and Blum. The kate thermometer, anemometer and the newer and more convenient Alnor velometer were used to measure air velocity at the face of the hood. Static suction was also measured, and air samples were taken with the impinger. Where only general ventilation was used even in large rooms, the chromic acid concentration was above the safe limit of 1 mg. per 10 cubic meters. With the cross draft local-exhaust system it is possible to keep it below that limit. Adequate control was maintained when velocities of about 1,500 and 2,000 feet per minute were created at the face of the hood.

(A copy of the above issue may be obtained from the Superintendent of Documents Washington, D. C. for 5 cents. Stamps are not accepted.)

287. TREATMENT OF HATTER'S FUR. E. Elod. Arbeitsschutz, pp. 16-17 (1937).

In the process of treating pelts for felt hats with mercury it is impossible to free the fur completely from mercury by heating. The vapor is liberated in all stages of hat fabrication and even in finished hats. In cooperation with the German gold and silver purifying association, a completely satisfactory bath has been developed, by means of which the mercury hazard can be eliminated from the hat industry.

288. APPLICATION OF ENGINEERING SURVEYS TO THE HATTERS' FUR CUTTING INDUSTRY.

J. J. Bloomfield and J. M. DallaValle. Am. J. Pub. Health, 27, 167-178 (February, 1937).

A detailed survey of 5 plants and a preliminary study of 36 others showed that the major hazard was the exposure to mercury vapor and dust. The General Electric selenium sulfide detector was used for determining mercury vapor and the impinger, followed by the Fraser electrolytic method, for mercury dust. The konimeter was used for determining total dust. The concentrations of mercury to which workers were exposed varied from 7.2 mg. per 10 cu. m. for shipper pliers, 5.4 mg., and blowers, 4.6 mg., down to 0.6 mg. for the least exposed miscellaneous employees. The methods now employed for controlling mercury vapor and dust are: (1) segregation, (2) local exhaust ventilation, and (3) general natural ventilation. It is suggested that by segregating the operations handling untreated pelts, approximately 23% of the workers may be removed entirely

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from the hazard. Proper local exhaust applied to some operations and good natural or mechanical ventilation applied to piling and shipping reduce the mercury exposure for those occupations. The removal of treated material from workrooms, good housekeeping and good general sanitation are recommended. (Reprints may be obtained from Mr. Bloomfield, U. S. Public Health Service, Washington, D. C.)

289. THE DETERMINATION OF MERCURY IN CARBONATED FUM. F. H. Goldman. U.S. Public Health Repts., vol. 52, No. 8, 221-223 (Feb. 19, 1937).
This brief paper describes an analytical method used in connection with the the survey reported by Bloomfield and DallaValle (preceding abstract). Ten samples of workroom dust ranged from 0.06 to 0.37 mg. mercury per cubic meter of air.
(The above volume and number may be obtained from the Superintendent of Document Washington, D. C. for 5 cents. Stamps are not accepted.)
290. THE ESCAPE OF MERCURIAL VAPORS IN WORKSHOPS. STUDY OF METHODS FOR CONTROL OF THESE VAPORS. A. N. Lvoff. Med. du. travail, 9, 4-25 (Jan. 1937).
In industries where mercury is employed, much of the hazard arises from mercury deposited on floors, in cracks, under linoleum and in many other places. Where the metal is accessible, the use of potassium permanganate and hydrochloric acid reduces the vapor concentration 30 to 60%. However, in workshops where mercury may be absorbed by walls or is in inaccessible places, the use of a penetrating gas that will react with mercury is necessary. The use of hydrogen sulfide in concentrations of 0.5 mg. per liter for 40 hours was found to reduce the mercury vapor approximately 82 to 90%. This treatment of course introduces another hazard and should be used with the utmost caution if at all. The concentration of hydrogen sulfide used does not produce acute poisoning, but chronic poisoning may follow prolonged use. At present it is used, not only in isolated places, but for entire floors and workshops, after work hours.

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AIR HYGIENE FOUNDATION OF AMERICA, INC.

4400 FIFTH AVENUE

PITTSBURGH, PA.

HW86-0007841

The Medical Bulletin which was somewhat delayed in printing has been completed and your copy will reach you with this. Additional copies for use in your own organization may be had at \$2.00 each.

Enclosed is a copy of the Proceedings of the Annual Meeting, and the recommended and approved Research Program. As a member of the Foundation you can be helpful in calling this to the attention of other firms of your acquaintance who should be affiliated. As you know, the Foundation needs a greatly increased membership in order to make up the sum necessary to support this research program. The current dues were set relatively low with the idea of interesting as many firms as possible. Any additional number of copies of this bulletin may be had upon request and without charge.

The supplement to Bulletin No. 1 of the Legal Series will be ready for distribution in a week or ten days. This bulletin brings the information relative to statutes and court decisions up to March 1, 1937.

Also enclosed will be found an announcement of the publication by the Journal of Industrial Hygiene and Toxicology of a series of lectures given at the Harvard School of Public Health in August, 1936, on THE ENVIRONMENT AND ITS EFFECT UPON MAN. These papers have been bound in book form and are now available at \$3.50 each. If you wish one or more copies, address the Journal of Industrial Hygiene & Toxicology, Harvard School of Public Health, 55 Shattuck Street, Boston, Mass.

A Symposium on Silicosis will be held at Saranac Lake, June 21-25, 1937. A copy of the proposed program is enclosed. It is thought that you may care to call this to the attention of your medical staff.

Also enclosed is a report on Comparison of Dust Counters, which was prepared at the request of several members.

H. B. Meller
Managing Director

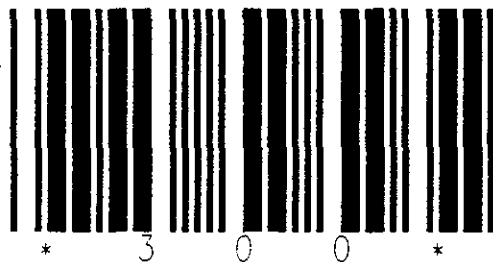
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UNITED STATES AIR ARMS

November 1992

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Third Silicosis Symposium Saranac Lake, N.Y. June 21 — 25, 1937

Monday, June 21:

- 9:00 a.m. Introductory Remarks. Dr. L. U. Gardner, Saranac Laboratory.
- 9:15 The Significance of the Silicotic Problem. Dr. A. J. Lanza, Metropolitan Life Insurance Company.
- 10:15 Etiology of Silicosis. Dr. R. R. Sayers, U. S. Public Health Service.
Discussion.
- 2:15 p.m. Dust Concentrations and Their Measurement. Prof. Philip Drinker, Harvard School of Public Health.
Discussion, led by Mr. M. T. Durkan, Saranac Laboratory.

Tuesday, June 22:

- 9:00 a.m. Pathology of the Pneumoconioses. Dr. L. U. Gardner, Saranac Laboratory.
Discussion.
- 2:15 p.m. Pathology of the Pneumoconioses. Correlation of the Pathologic and Roentgenologic Findings. Dr. L. U. Gardner, Saranac Laboratory.
Discussion.
- 4:00 Physiology of the Fibrotic Lung. Prof. W. S. McCann, University of Rochester, School of Medicine.
Discussion.

Wednesday, June 23:

- 9:00 a.m. Roentgenologic Aspects of the Normal and the Silicotic Lung. Dr. E. P. Pendergrass, University Hospital, University of Pennsylvania.
Discussion.
- 2:15 p.m. Roentgenologic Diagnosis of Silicosis. Dr. H. L. Sampson, Trudeau Sanatorium.
Discussion.

Thursday, June 24:

- 9:00 a.m. The Clinical Picture and Diagnosis of Silicosis with Consideration of Disability. Dr. A. R. Riddell, Ontario Department of Health.
Discussion.
- 12:00 The Occupational History. Mr. D. E. Cummings, Saranac Laboratory.
- 2:15 p.m. Control — Medical Aspects. Dr. A. R. Riddell, Ontario Department of Health.
- 3:15 Control — Engineering Aspects. Prof. Philip Drinker, Harvard School of Public Health.
Discussion.

Friday, June 25:

- 9:00 a.m. Administrative Aspects of Silicosis. Mr. D. E. Cummings, Saranac Laboratory.
Discussion.
- 2:15 p.m. Legislative Control and Compensation. Mr. T. E. Waters, Occupational Disease Commission, Baltimore, Md.

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THE ENVIRONMENT AND ITS EFFECT UPON MAN is comprised of the following lectures given at the Harvard School of Public Health, August, 1936.

1. The effects of the social environment. Lawrence J. Henderson and Elton Mayo.
2. Industrial Fatigue. David B. Dill.
3. Air-borne disease. H.D. Chope and Wilson G. Smillie.
4. Air-borne infection and sanitary air control. William F. Wells and Mildred Weeks Wells.
5. The effects of heat and humidity upon the human body. Cecil K. Drinker.
6. Abnormal air conditions in industry: their effects on workers and methods of control. C. P. Yaglou.
7. The physiological effects of high pressures. Louis A. Shaw.
8. Industrial operations in compressed air. Ole Singstad.
9. Carbon monoxide poisoning. Cecil K. Drinker.
10. The toxicology of organic vapors and gases. John H. Foulger.
11. The toxic dusts and fumes. Lawrence T. Fairhall.
12. The causation of pneumoconiosis. Philip Drinker.
13. Clinical aspects, diagnosis and prevention of pneumoconiosis. W. Irving Clark.
14. Design of exhaust hoods for dust-control systems. Theodore Hatch.
15. Respiratory protective devices. Carlton E. Brown.
16. The application of air conditioning in normal life. Philip Drinker.
17. Hospital air conditioning. C. P. Yaglou.
18. A laboratory of industrial toxicology. W. F. von Oettingen.
19. A laboratory of industrial hygiene. Warren F. Cook.

These papers have been bound in book form and will be available about April 20. Copies may be ordered direct from:

The Journal of Industrial Hygiene and Toxicology,
Harvard School of Public Health,
55 Shattuck St.
Boston, Mass.

Price, prepaid, \$3.50.

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NOVEMBER 1937

THE AIRBORNE

NW88-0007847

Air Hygiene Foundation of America, Inc.,
4400 Fifth Avenue,
Pittsburgh, Pa.,
May 1, 1937

COMPARISON OF DUST COUNTERS

As a result of inquiries from several members, the Foundation procured from Bausch & Lomb one of their new dust counters and made a limited number of parallel tests with that instrument, the Owens jet dust counter, and the Greenburg-Smith impinger. The comparison under varied conditions showed that no close correlation of the results can be made. The Bausch & Lomb instrument gave results lying between those obtained with the Owens counter and those with the impinger. In some cases the lightfield method with the Owens counter and the impinger was supplemented by some darkfield tests.

Experimental Results

Tests were made in which the instruments were compared under the usual conditions for each, i.e., with lightfield for the impinger and Owens counter. The results so obtained will be presented first in the following table. The numbers in parentheses indicate the number of reasonably agreeing tests averaged, if more than one:

Test			Impinger, 120 X	Bausch & Lomb	Owens, 1170 X
No.	Date	Character	Million particles	per cu. ft.	
1	1-13	Outside, fog	5.3 (2)	22.5 (2)	145 (2)
2	1-27	" "	6.0	15.3 (2)	
3	1-29	" slight fog	1.7	29.7 (2)	96
4	3- 2	Industrial	15.0 (2)	51.3 (2)	93 (2)
5	3- 2	"	8.4	46.3 (2)	142, 103
6	3- 2	"	1.45	11.1, 5.4	
7	3-10	Lab., clear	0.75	24.5	63.5
9	4- 2	" "	0.17	12.1	23.4

The value for the impinger in test 4 is an average of two poorly agreeing tests on the same sample.

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Following are the ratios for each pair of instruments:

Test No.	Bausch & Lomb & Impinger	Owens & Impinger	Owens & Bausch & Lomb
1	4.2	27.4	6.4
2	2.5		
3	17.5	56.5	3.2
4	3.4	6.2	1.8
5	5.5	17.0, 12.2	3.1, 2.2
6	7.6, 3.7		
7	32.6	85.0	2.6
9	63.5	136.0	1.9

These ratios illustrate the lack of correlation of counts. It will be noted especially that the ratios of the Bausch and Lomb and Owens results to those obtained with the impinger are much higher for atmospheric samples than for industrial samples.

A few impinger samples were counted in darkfield illumination. One difficulty was always encountered, in that fine scratches and pits on the cells, invisible in the lightfield, were so numerous as to give a high blank. Even when counts were made with new cells, the blank became rather high after using the cell a few times. The following comparisons were made with the same samples in light and darkfield illuminations:

Test No.	Light, 120X	Dark, 120X	Light, 230X	Dark, 230X
1	10.6	33.9		
2	9.5	19.3		
3	1.3	12.7		
4	0.75		1.3	3.3

The first attempts to use darkfield lighting with the Owens samples were unsuccessful, as the oblique rays did not cross the air space between the slide and the cover glass on which the dust was deposited. Finally that difficulty was overcome by inverting the slide, so that the cover glass made an oil contact with the darkfield condenser. The following results were obtained with that method:

Test No.	Light, 120X	Dark, 120X	Light, 230X	Dark, 230X	Light, 1170X
1	5.8	22.7	15.0	4.3	
2	2.7	15.6	8.6	19.3	23.4

The last test was the only one in which a direct comparison of all methods was successfully obtained, except the impinger with darkfield. The figures for that test are summarized here:

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Special Report on Comparison of Dust Counters - 3

Impinger, light, 120 X	0.17
" " 230 X	0.5
Bausch & Lomb, dark, 200 X	12.1
Owens, light, 120 X	2.7
" dark 120 X	15.6
" light 230 X	8.6
" dark 230 X	19.3
" light 1170 X	23.4

The exact magnifications were chosen so as to afford a simple ratio of the apparent width of stage micrometer divisions to that of the Whipple disk squares.

Discussion and Conclusion

The figures obtained, of course, do not constitute an accurate comparison. The number of tests was small, and errors, incidental to the beginning of a series of tests, occurred.

One source of error in all dust counting, especially in darkfield work, is lack of constancy of illumination. Ordinarily the maximum illumination, namely that given by an arc light, is used in darkfield work. The Bausch & Lomb procedure makes use of a substage incandescent light, which is satisfactory if the illumination is constant, but care must be taken to keep the lamp in the proper position. It is possible that the first results are low because that precaution was not observed, and the apparatus should be made foolproof in that respect. It is understood that the manufacturers are correcting that defect by providing for attaching the lamp housing to the counter.

These preliminary results lead to the following conclusions:

- (1) No correlation of any pair of the three instruments is possible for all kinds and sizes of dust.
- (2) Under the same illumination and magnification, the count in air is higher than in water, the divergence increasing with decreasing particle size.
- (3) The Bausch and Lomb counter gives results intermediate between those with the impinger and those with the Owens apparatus under the usual conditions of use.

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May 1937

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