

REFERENCES TO RECENT LITERATURE ON INDUSTRIAL DISEASE

59. CONNECTICUT STATE DEPARTMENT OF HEALTH, FOR YEAR ENDING JUNE 30, 1935. REPORT OF BUREAU OF OCCUPATIONAL DISEASES. A. S. Gray, Pp. 411-462, Hartford, 1936.
- During the year 405 inspections were made, involving the more serious industrial hazards, including especially dust in foundries, lead, solvent vapors and chlorinated naphthalene.
- It is aimed to encourage the reduction of not only silica-bearing dusts but all dusty atmospheres to moderate limits. The most fruitful appeal for control measures is based on compensation and economy. Ten pages are devoted to control of foundry dust with numerous figures on results found in practice, and detailed recommendations for reducing the silicosis. Konimeter "grab" samples have been added to impinger samples as a routine to supplement data on fluctuations in dust concentrations through time, location, operations and causative factors. Similar details are given from studies of rock drilling and miscellaneous dust exposures. In a long tunnel through rock with 10 to 15% quartz it was recommended that dust be reduced to 20 million particles per cu. ft., in part by use of traps on drills. In a cotton picking room with 3.5 changes of air hourly a surprise was met when samples showed excessive concentrations of dust, of which the settled portion contained 28% silica. Some street sweeping machine operators were likewise found to be exposed to a potential but controllable silicosis hazard.
- Lead hazards were found in some cases, especially where the temperature of melting pots exceeds 900 F. Dipping hot aluminum castings into nitric acid was found not to be hazardous in one case. Sodium fluosilicate as a flux for aluminum was found to yield too much hydrofluoric acid for safety. Fumes from the drying of lacquers caused symptoms of poisoning. Triethylene chloride used in degreasing constituted a hazard in one case. Preventive measures, often simple, were recommended in every case.
- Many laboratory methods are described fully, including determination of solvent vapors in air, determination and analysis of dust, and analysis of poisonous material. Educational activities are described. The total cost of the work for the year was 4 cents per worker in industries with potential health hazards.
60. INDUSTRIAL HYGIENE ACTIVITIES IN THE UNITED STATES. R. R. Sayers and J. J. Bloomfield. Am. J. Pub. Health, 26, pp. 1087-1096 (1936).
- During the first nine months of 1936 the number of State Departments of Health conducting industrial hygiene work was increased from 5 to 17, in addition to two states in which that work is conducted by the Department of Labor. The budgets vary from 7 to 77 mills per worker. The authors describe and emphasize the help which the federal Public Health Service can give states inaugurating such bureaus.

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References to Recent Literature - 2

61. RECENT CHANGES IN THE PAINTERS' TRADE. A. Hamilton. U. S. Dept. Labor, Div. Labor Statistics, Bull. no. 7 (1936).
The painters' trade has always been unhealthful above the average, as shown in various statistical records. The advances made in industrial hygiene during recent years have not as yet registered their effect on these statistics, although in many branches of the industry such advances have been striking. Naturally it will require a longer time before the improvement can be shown statistically. The improvements in the last twenty years include the displacement of lead pigments by harmless materials, especially in interior work and in mass production, and the substitution of mineral spirits for turpentine. On the other hand, the growing use of lacquers instead of paints, and the use of the spray gun, have introduced new hazards. The solid materials are harmless, but the liquid components are not always. Several of the new organic solvents are known to be poisonous, and their effect is aggravated by the use of the spray gun. As yet no thorough study has been made of these solvents, except benzol. Although most of the solvents may prove to be harmless, it is well to take all possible precautions, including the use of local exhausts and abundant ventilation, and if these fail, the use of respirators. However, the only efficient way of protecting the spray painter is to select coatings that are free from poisonous constituents.
62. LEAD ENCEPHALOPATHY AS CAUSE OF DEATH? E. Beintker. Samml. v. Vergift., vol. 7 (Gutachten) pp. 53-56 (1936).
Death of a worker in a storage battery foundry from pneumonia following severe exposure while in a state of mental confusion raised the question whether the lead had acted directly on the brain. The quantities of lead found in the various organs did not confirm that supposition, but indicated rather that the mental condition could be traced to an early progressive paralysis.
63. LEAD POISONING AS AN ETIOLOGICAL FACTOR IN CIRRHOSIS OF THE LIVER. S. Bazzano. Med. del Lav., vol 27, pp. 303-314 (1936).
Cirrhosis of the liver is not a common concomitant of lead poisoning and when present can be traced to other factors, especially alcoholism.
64. ANGINA PECTORIS AFTER FLUE GAS POISONING. C. Kroetz. Med. Klin. vol. 32, pp. 1521-1524 (1936).
A locomotive engineer suffered an attack of angina pectoris following poisoning by flue gas when his train was stalled in a tunnel. Seven years later, and again thirteen years later, he was poisoned in the same way and the illness increased. It was sometimes considered an accident neurosis, but the higher court ruled that there was no connection with the poisoning. The author shows from the literature and the case history that a connection exists between flue gas poisoning and angina pectoris and notes that heart injuries as a result of carbon monoxide poisoning receive too little attention.
65. NEW SYNDROME FROM INDUSTRIAL POISONING. C. Bollesini. Ponsiero Med., vol. 25, p. 269 (Sept., 1936).
A preliminary report on cases of industrial poisoning from absorption of diphenyl-arsinic acid through the skin, including one death. In other cases the symptoms regress after discontinuation of the work. The author emphasizes the importance of protecting workers by constant attention to cleanliness and the use of protective clothing.

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References to Recent Literature - 3

66. SECOND FATAL CASE OF LUNG FIBROSIS FROM INDUSTRIAL RADIUM POISONING. E. Tonges and H. H. Kalbfleisch. Frankfurt. Ztschr. f. Path., vol. 50, pp. 100-122. (1936).
The authors describe a fatal case of lung fibrosis in a woman working in a radium laboratory. After four years of work, she fell ill of grippe with inflammation of lungs and costal pleura, followed by difficult breathing and great loss of weight. She died two years later. Fibrosis of the lung was present several months earlier, and autopsy showed extensive damage to the lung, with radium deposits in various parts of the body, although less in the lungs than in some other sections. The lungs must be considered the chief means of entry of radioactive substances, both of radium salt particles and of the gaseous decomposition products of radium.
67. NEPHRITIC SYNDROMES CAUSED BY INDUSTRIAL POISONING WITH CARBON TETRACHLORIDE. S. Franco. N. Y. State J. Med., 36, pp. 1847-1853 (Dec. 1, 1936).
Two cases are presented, both of workmen exposed to the vapors without adequate ventilation. The kidneys were affected in both cases, and treatment was followed by recovery. A study of the literature showed two forms of kidney injury, represented in the two cases. Details of the two forms are presented, the principal difference being presence of albumen in the urine in one form and its absence in the other.
68. FATAL POISONING WITH NITROUS GASES. B. Both. Samml. v. Vergift., 7, (Vergiftungsfälle) pp. 177-178 (1936).
A typical case of nitrous gas poisoning resulted from breaking a flash of nitric acid. The notable fact was the appearance of dyspnea (shortness of breath) and cyanosis (blueness of skin) within 20 minutes of the inhalation of the fumes.
69. FATAL POISONING IN ACETYLENE WELDING (DISSONS GAS). R. Maeriske. Arbeitsschutz (Reichsarbeitsblatt III), pp. 283-285 (1936).
Two workers became ill after welding for 10 minutes in a large boiler with a small opening, and one died within a few hours of lung edema and other complications. Experiments under similar conditions showed that dangerous quantities of nitrogen tetroxide were produced. Phosphine and other substances were eliminated as possibilities.
70. ACUTE FLUORINE POISONING. W. Roholt. Deutsch. Ztschr. f. d. ges. gerichtl. Med., 27, pp. 174-198 (1936).
The author reviews 112 cases of fluorine poisoning with 60 deaths, mostly suicides or cases of mistake. Gaseous compounds are produced in superphosphate, aluminum, gas, enamel and chemical compounds; and fluorine compounds must have played a part in the big catastrophe in Belgium in 1930. The symptoms are acute irritation of the stomach and intestines, tetany-like cramps and paralysis.
71. RESEARCH IN LIGHT METAL DAMAGE TO EXPERIMENTAL ANIMALS. G. Gerlach. Bruns. Beitr. x. klin. Chir., 161, pp. 430-436 (1936).
Needles of various metals, pointed at both ends, were sunk into the liver and musculature of animals. Metals with a high percentage of magnesium have a damaging action resembling microscopic cuts. The author attributes the effects to the reaction between magnesium and water, liberating hydrogen in bubbles throughout the tissues, which breaks down and serves as a favorable medium for infection.

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72. COMPARATIVE HISTOLOGICAL AND QUANTITATIVE SPECTROGRAPHIC STUDIES IN EXPERIMENTAL MERCURY POISONING. R. Muller and E. Scheiner. Arch. expt. Path. Pharmacol., 180, pp. 718-730 (1936).
There is no correlation between the degree of damage to the intestinal mucosa of guinea pigs by mercury salts and its mercury content. The same is true in the kidneys when they are affected.
73. RESEARCHES ON THE TREATMENT OF HYDROCYANIC ACID POISONING CAUSED BY INHALATION W. Wirth. Arch. f. exper. Path. Pharm., 179, pp 558-902 (1935).
The study of hydrocyanic acid poisoning has become highly important on account of its increased technical use. The author has investigated 60 substances and mixtures to determine how far they can reduce the effect of hydrocyanic acid poisoning. The animals used - cats, rabbits, rats, mice and pigeons - were first poisoned until the respiration ceased while the heart continued to function, then put into chambers in which the various substances were introduced in vaporized form. Sodium carbonate was found the most effective of the substances used. Its effect was confirmed by experiments on the isolated frog's heart.
A characteristic fall of pH in rabbits treated with hydrocyanic acid was followed by an opposing rise when grape sugar and methylene blue were administered. No good results were found in experiments with substances producing methemoglobin. These extensive investigations show that the greatest reserve must be held regarding therapeutic measures for severe hydrocyanic poisoning. Only under particularly favorable conditions can sufficient of the poison be neutralized to save life.
74. METHEMOGLOBIN ANEMIA AFTER ETHYLENE NITRITE POISONING. K. ... Seggel. Deutsch. Arch. f. klin. Med., 179, pp 353-361 (1936).
Four workers who had breathed ethylene nitrite showed chocolate-brown-violet discoloration of the skin and mucous membrane, and a methemoglobin anemia. In all the color decreased markedly after a few days, and they were well in 10 days.
75. METHYLENE BLUE THERAPY IN NITROBENZENE POISONING. H. D. Leinoff. New England J. Med., 215, pp 191-193 (July 30, 1936)
Methylene blue, an antidote for nitrobenzene or cyanide poisoning, was administered intravenously to a man with an extremely cyanotic and dyspneic condition, characteristic of either of these poisons. Nitrobenzene was recovered from gastric washings, and recovery was startling in rapidity. A brief discussion of the use of methylene blue is included.
76. THE MANSFELD PNEUMOCONIOSES ON THE BASIS OF PATHOLOGICAL-ANATOMICAL RESEARCH. J. Wagon. Nova acta Leopoldina, 3, pp 477-592 (1936).
Pneumoconiosis has been studied in this locality with unusual thoroughness, and a number of papers have been published dealing especially with anatomical and pathological aspects. The present paper combines all the material collected, which is based on 119 autopsies of pneumoconiotic cases. Only a few points can be mentioned here: The dust of copper schist is black as a result of its bituminous content and this color is what appears in the lungs. The most severe forms occur without tubercular complications. Softening and cavity formation also occur without tuberculosis, evidently more often than with other dusts. This softening leads to dust mobilization and therewith to a progression of the process after cessation of exposure. The Mansfeld pneumoconiosis is undoubtedly a silicosis; it has one peculiarity - cavity formation. This paper is considered one of the most valuable publications on silicosis.

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77. THE PHYSIOLOGICAL RESPONSE OF PERITONEAL TISSUE TO CERTAIN INDUSTRIAL AND PURE MINERAL DUSTS. J. W. Miller and R. R. Sayers. U. S. Pub. Health Repts., 51, pp. 1677-1689 (Dec. 4, 1936).

Various dusts were injected into the peritoneum of guinea pigs. The response to each particular dust was constant in all the animals examined, and could be classified as an absorptive, proliferative or inert reaction.

In the absorptive reaction, the injected dust disappeared from the peritoneal cavity without the production of scar tissue. This reaction is given by calcite, limestone, precipitated chalk, gypsum and cement. Interpretation of this reaction is difficult, but it appears logical that dusts which tend to remain in the tissues should be considered potentially harmful, although not as dangerous as those causing a proliferative reaction.

In the proliferative reaction the nodules produced by the dust continually increase in size. Quartz, tripoli and siliceous chert (forms of free silica) produce this reaction, which corresponds, therefore, to lung fibrosis and is produced by the same substances.

In the inert reaction, given by anthracite and bituminous coal, precipitator ash, soapstone, talc, asbestos, feldspar, silicon carbide, hematite, calcium phosphate and sericite, the amount of injected dust remained the same in the peritoneal cavity, but the nodules become more flattened and fine dust particles are carried over extensive areas by phagocytes.

With this biological method of classification, which, in a number of instances, has been correlated with clinical observations and industrial surveys, it is quite possible to use intraperitoneal injection methods to determine the pneumoconiotic potentialities of a dust in a relatively short time, usually 60 days.

78. THE PNEUMOCONIOSES IN SOUTH WALES. S. L. Cummins. J. Hyg. Camb., 36, pp. 547-559 (Oct., 1936).

In the South Wales coalfield, many cases of lung disease are found which cannot be classified as silicosis, and are not compensable under present laws. The author describes some of these conditions and attempts to classify the pneumoconioses into four types: (1) silicosis and asbestosis; (2) silico-anthraco-sis and silico-siderosis; (3) anthracosis of coal trimmers and screeners, and (4) fibrotic aggregations of inert dust occasionally present in all types, especially in silico-anthraco-sis. In all types there are appearances suggesting damage to the elastic structure of the lung, which may be related to the emphysema (inflation of lung tissue) and dyspnea (shortness of breath) often found. The emphysema is not necessarily a compensatory effect of fibrosis as it often occurs with very little fibrosis. Bronchitis is common, probably as a result, rather than a cause of pneumoconiosis. The amount of fibrosis and lung damage in South Wales pneumoconioses varies directly with the intensity and duration of exposure to siliceous dust, as expressed in the silica content of the dried lung.

79. PATHOLOGY OF THE PNEUMOCONIOSES. L. T. Gardner, N. Y. State J. Med., 36, pp. 1377-1391 (Oct., 1936)

This paper is a brief summary of the anatomic changes produced by inhaled silica and other dusts and the complications due to infection. Simple silicosis, when manifested by uniform nodulation, does not produce symptoms or incapacitate for work. A person with this type of reaction may continue working, but severe dust exposure should be avoided and yearly x-ray examinations should be made. However, if there are massive areas of conglomerate fibrosis, usually associated with discrete nodulation, the patient will have dyspnea and limited capacity to work. He should be given lighter jobs, not discharged. No person with open tuberculosis should be allowed to work in silica dust. Persons over forty with well-healed adult tuberculosis can be safely employed where there is a silica hazard, provided means are taken to minimize it, but the younger the individual with a similar con-

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dition, the less safely can he be exposed to silica dust. A young employee with silico-tuberculosis should be given sanatorium treatment.

80. ASBESTOSIS: A ROENTGENOLOGIC REVIEW OF 71 CASES. H. R. Shull. Radiology, 27, pp. 279-292 (Sept., 1936).
A study of 71 cases of asbestosis is presented, with emphasis on roentgenologic appearances. The cases were classified as to the state of progression of the disease, which varied roughly with the length of exposure. Tuberculosis was found in about 12% of the cases, more frequently in the more advanced type. The author tabulates ten radiographic findings characteristic of asbestosis, and concludes that "while the roentgenogram is the most reliable diagnostic aid, the interpretation and correlation with clinical signs and symptoms is often difficult. Without the history of exposure a certain number of slightly advanced cases will not be recognized," that "a fair percentage of the slightly advanced and moderately advanced cases do tend to improve and the attendant disabilities to become lessened when removed from asbestos dust," that "asbestosis does not predispose to tuberculosis" and that from his observations asbestosis is not a progressive disease.
81. THE DIFFERENTIAL DIAGNOSIS OF SILICOSIS FROM OTHER PULMONARY DISEASE. G. G. Ornstein. N. Y. State J. Med., 36, pp 1382-1385 (Oct., 1936).
The author challenges the general belief that tuberculosis is more common in silicotic subjects and that that infection is the most common cause of their death. He believes that many of the lesions which are diagnosed as silico-tuberculosis in the roentgenogram are actually conglomerate manifestations of silicosis because of the absence of tubercle bacilli in the sputum and because the infection manifests itself so late in the course of the silicosis. He criticized the existing evidence for this belief in the case of the Barre granite cutters, the Picher lead and zinc miners and the N. Y. City rock drillers and points out that there may have been errors in diagnosis. He calls attention to possible errors in diagnosis and points out that "reported incidence of tuberculosis is too low (1.8 to 5%) to be compatible with the statement that "most silicotics die of pulmonary tuberculosis."
82. CLINICAL FEATURES AND SIGNIFICANCE OF SILICOSIS. A. J. Lanza. N. Y. State J. Med., 36, pp. 1386-1388 (Oct., 1936).
Diagnosis of silicosis depends upon a characteristic roentgenogram of the chest, a history of exposure to silica dust and a physical examination. Where the silica hazard is severe, the author believes that "more or less complete disability may develop from the resulting fibrosis without a complicating infection. The silicosis seen in American industry today rarely disables without superimposed infection. Silicotic patients with active tuberculosis must stop work." Lanza would advise the man with definite silicosis to avoid further exposure and thereby "hold his disease in check." He believes that if the employer takes measures to reduce the dust hazard the silicotic may remain at his regular work.
83. RESEARCH ON THE SILICA CONTENT OF THE BLOOD OF RABBITS AFTER PROLONGED INHALATION OF QUARTZ DUST. M. Schonfelder. Arch. f. Hyg., 117, pp. 44-52 (Oct., 1936).
Two rabbits were fed potassium aluminum silicate containing 20% silica, once a month for a year (quantity not given). The blood analyses at the end of this treatment indicated 1.63% silica, against contents of 2.3 to 3.9% in rabbits exposed daily for 9 months to 2.5 years. The two rabbits were then infested with tubercle bacilli. Six to eight weeks later the silica content remained the same, and the animals apparently had recovered from tuberculosis. The results again bring up the question of this type of therapy as an aid in the treatment of pulmonary tuberculosis.

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84. PSEUDOTUBERCULOM. SILICOTICUM. K. C. Eden and J. Herbert-Burns. Brit. J. Surgery, 24, pp. 346-349 (Oct., 1936).
A nodule was found in the arm of a young girl who had fallen on a flinty road. Sixteen years later the nodule was removed and found to have a microscopic appearance exactly like that of a tuberculoma, with clumps of giant cells, but no caseation or tubercle bacilli. Many doubly refracting crystalline particles were seen under a high magnification.
85. THE RESPONSE OF THE RABBIT'S CAROTID ARTERY TO INJURIES: Pt. 2. THE EFFECTS OF INJECTION OF STREPTOCOCCUS VIRIDANS AND OF SILICA. E. M. Ramsey and D. W. Gaiscr. Yale J. Biol. and Med., 9, pp. 43-44 (Oct., 1936).
A heavy suspension of finely divided sterile silica in sterile normal saline was injected into the ligated carotid arteries of 9 rabbits, which were killed at the end of various periods. Detailed appearances are given, indicating considerable damage to the tissues.
86. SILICOSIS AND TUBERCULOSIS. F. Capelli, Med. del Lav., 27, pp. 289-302 (Oct., 1936).
The author discusses the mechanism of the development of true silicosis, and of infections associated with it especially tuberculosis. The prognosis (expectation of results) of tuberculosis complicated with silicosis is less favorable than silicosis of the same degree. Eight cases are described.
87. FIRST REPORT ON AN INQUIRY INTO AFFECTIONS OF THE AIR PASSAGES AMONG COAL MINERS IN HOLLAND. Vossenaar and Doubrow. Bull. Lond. Med., 115, pp. 300-305 (1936).
Observations were made on 600 miners, all employed over 10 years, to determine the extent of injury from dust. Only one was found with fibrosis of the lungs, although 14 cases of respiratory troubles were included. The conclusions drawn are that the dust of the mines does not originate pulmonary fibrosis; no relation exists between exposure to the dust and the appearance of x-ray shadows; similar shadows may be met with in ordinary cases of tuberculosis; any infectious agent - but particularly tubercle bacilli - in the cases of those exposed to dusts may cause chronic troubles; hence industrial dusts are unhealthy factors. In the discussion which followed the usual French doubt is expressed as to the existence of any clinical entity to be known as silicosis, and advice is tendered to the Government not to ratify the International Convention listing silicosis as an industrial disease.
88. THE COLLECTION AND ANALYSIS OF AIR-BORNE DUST DURING THE DRIVING OF HARD HEADINGS. J. I. Graham and F. Lawrence. Trans. Inst. Mining Engrs. Lond., 92, pp. 1-11 (Oct., 1936).
The paper describes investigations of the average quantity of free silica suspended in hard headings and other underground places where mining operations are carried on in hard rock. Sufficient dust for analytical determinations was collected by means of a "sugar tube" method, modified by replacing the sugar by potassium nitrate. Several hundred cubic feet of air were usually necessary for a sample. The feldspathic material plus silica was first determined; the silica was then extracted, the feldspar determined, and the free silica then estimated by difference, with a correction for silica lost during analysis. The free silica contents ranged up to 44%. The quantities were determined as grains per cubic foot, but by use of estimated particle sizes, the results were converted into number of particles per cubic centimeter. A concentration of 15 grains per cc. corresponds to the safe limits as determined in South Africa. Whenever precautionary measures were taken, whether wet drilling with adequate ventilation or the use of the Hay dust-trap, the dust present was well below that figure.

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89. THE INDUSTRIAL ASPECTS OF DISPERSE SYSTEMS IN AIR AND GASES. R. Lessing. Trans. Faraday Soc., 32, pp. 1223-1233 (1936).
A general introductory paper on a symposium on the industrial aspects of aerosols, held by the Faraday Society last summer. It presents a review of present knowledge of dust, smoke, fog, flue gases, mining dusts, and methods used in separating these aerosols from air. It also points out directions in which future work should be carried out.
90. MODERN METHODS OF DETECTION AND DETERMINATION OF INDUSTRIAL GASES IN THE ATMOSPHERE. E. Leclerc and R. Haux. Rev. universelle mines., 12, pp. 293-298 (1936).
Methods of detection of various gaseous impurities in air are reviewed.
91. GRAIN ITCH. Med. du Travail, 8, pp. 90-113 (1936).
Grain itch has been declared a compensable disease in a French court. Dock laborers handling grain cargoes are especially exposed to it. Some men become inured to exposure, but others develop a sensitivity that may manifest itself as urticaria or asthma; immunity, however, is the exception. The exciting agent is a very small parasitic insect, the life history of which is given. Prevention lies in freedom from exposure to infection. Disinfection of the grain is difficult.
92. CALCIUM METABOLISM AND THERAPY IN DERMATOLOGY. E. S. Lain. Southern Med. J., 29, pp. 626 (1936).
The author finds that many so-called industrial dermatoses respond satisfactorily to calcium thiosulfate or gluconate solutions administered intravenously, supplemented with suitable doses of calcium gluconate or lactate taken in water. The danger involved in intravenous administration is stressed and suitable doses and technique are formulated.
93. OBSERVATION ON INDUSTRIAL DERMATOLOGY. H. R. Forster. J. A. M. A., 107, pp. 247-252 (July 25, 1936).
A general summary of the subject, describing compensation laws in the different states and the advantages and disadvantages of different types of such laws. Several groups of skin diseases are discussed. The author urges cooperation of dermatologists with industry and legislative bodies.
94. CHEMICAL BURNS OF THE EYE. O. W. Moss. New Orleans Med. Surg. J., 89, pp. 302-306 (Dec., 1936).
Copious irrigations with water are considered better than attempts at chemical neutralization. After irrigation, however, alkali burns should be neutralized with 1% acetic acid, and acid burns by 5-10% bicarbonate of soda. Glycerite of tannic acid is also useful for alkali burns, and a neutral solution of ammonium tartrate is of value in clearing corneal opacities after lime burns.
95. INDUSTRIAL EYE INJURIES AND THEIR PREVENTION. J. Winton. Brit. J. Ophthal., 20, pp. 673-682 (Dec., 1936).
The Royal Eye Hospital is in a highly industrialized section of London. About 6500 patients out of 10800 in the casualty department in 1935 had suffered industrial eye injuries. The ratio was the same in the first half of 1936. About half of these patients were emery wheel workers, and 30% more were engaged in turning, boring, soldering, drilling, welding, etc. The common types of eye injury are described and statistics are given. The high frequency of such accidents is due to lack of safety measures and indifference among the men. Safety devices are described.

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96. A NEW EYE SHIELD FOR USE IN INDUSTRY. C. G. K. Sharp. Brit. J. Ophthal., 20, p. 683 (Dec., 1936).
This shield has a flexible head band to which is attached a metal plate with a double ball and socket joint. On the end of this is a metal frame carrying laminated glass (for miners) or mica or gauze for other trades. As this frame stands out from the head, there is plenty of space for ventilation and it can be turned up when not in use or removed and put in a pocket.
97. FACTORS AFFECTING THE CONDITION OF MINE AIR. PROBLEMS ARISING OUT OF TEMPERATURE AND HUMIDITY IN DEEP MINING ON THE WITWATERSRAND. F. J. Tromp and J. P. Duminy. J. Chem. Met. Min. Soc. So. Africa, 37, pp. 89-93 (Aug., 1936).
A mathematical paper demonstrating that the final temperature of air humidified at the bottom of a dry shaft is the same as that of air humidified on the way down to the bottom of a wet shaft. The same amount of moisture is introduced in both cases. This confirms Tromp's theory as given in an earlier paper.
98. AIR FILTERS IN AIR-CONDITIONING SYSTEMS. P. B. Rowley. Refrig. Engin., 32, pp. 322-326 (Nov., 1936).
The author has compared both dry and viscous filters, using the standard A.S.H.V.E. procedure. Carbon black and ash were used in one series and lycopodium powder in another. One test demonstrates the longer life of a filter whose air passages become progressively smaller in the direction of air flow, as compared with a uniform filter. Several other tests show the higher dust-arresting power of a viscous or oil-coated filter having the same resistance as a similar dry filter. Lycopodium, typical of pollens, is easily filtered from the air.
99. FAN SELECTION AND NOMENCLATURE. A. A. Borstneff. Refrigerating Engin., 32, pp. 175-179 (1936).
This article is informative and valuable, not so much on the choice of fans as in clearing the nomenclature of fan engineering. It goes a long way in clearing up much of the confusion in the conception of static pressure at the inlet and outlet of a fan.
100. THE MORBID MINER. D. E. Dickson. Edinburgh Med. J., 43, pp. 696-705 (Nov., 1936).
Reports on miners' health in Scotland show a sickness rate among miners, equal to twice that of all other males in Scotland. The author, a medical practitioner in a mining town, seeks an explanation, and discusses various possible factors. He concludes that the root of the trouble lies in the rapid development of machine mining within the past 20 years. Machine-mining is accompanied by a speeding up of production, and the individual becomes just a cog in the wheel and has to keep up with the pace set by the machinery. Miners complain about this pressure of work. The feeling is not one of active discontent with conditions of work, but of passive dissatisfaction, almost going as far as being a repression. The men have not the same pleasure in their work as formerly, and they would, given an opportunity, stop work at a day's notice, simply for the sake of not having to go down the pit under present conditions. The cause appears to be that they are driven harder under present conditions; it is not a question of wages.

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101. DISABLING ILLNESS AMONG INDUSTRIAL EMPLOYEES IN 1935 AS COMPARED WITH EARLIER YEARS. D. K. Brundage. U. S. Pub. Health Service, B.-1700, pp. 11 (mimeographed) 1936.
The annual number of cases of sickness and nonindustrial accidents lasting 8 days or longer was higher in 1935 than in 1933 or 1934 but lower than for 1930, 1931 or 1932. Diseases of the circulatory system were more frequent in 1935 than in the preceding 5 year period. Influenza was more frequent in 1935 than in 1934, but less so than in the average for the five years. Other diseases changed but slightly. (The author's summary does not give percentages for the above). In female workers, nonindustrial accidents and cases of sickness decreased by about 2%.
102. SICKNESS AMONG MALE INDUSTRIAL EMPLOYEES DURING THE SECOND QUARTER AND THE FIRST HALF OF 1936. D. K. Brundage. U. S. Pub. Health Repts., 51, pp. 1675-1677 (Dec. 4, 1936).
This report gives statistics for the first half and second quarter of 1936, similar to the preceding abstract. For all respiratory diseases the rate for the second quarter was 2% higher than in 1935. Pneumonia increased 4%, and non-respiratory diseases increased 1.7%. A downward trend in new cases of tuberculosis was noted.
103. A STUDY OF TUBERCULOSIS DEATHS IN SILVER BOW COUNTY, MONTANA, AND OF OTHER IMPORTANT CAUSES OF DEATHS IN MINERS. J. H. Crouch. Am. Rev. Tuberculosis, 35, pp. 396-403 (1936).
In this locality the mortality rate for tuberculosis among copper miners was 755 per 100,000 over a period of 15 years, against 180 for other adult males, 43 for females and 19 for all under age 20. The effect of the industry is emphasized by comparison with similar rates for Indians, which were 530 per 100,000 for males, 592 for females and 537 for juveniles. The mortality rates among miners for accidents and for acute respiratory disease is also very high. The tuberculosis rate has declined approximately 50% during the 15 years under review. (The abstractor notes that no information on dust exposure is given, but that there can be little doubt that the high tuberculosis mortality represents occupational silicosis).
104. INVESTIGATION OF THE MINERALS THAT CAUSE SILICOSIS. B. Gossner. Sprechsaal 69, pp. 601-602, 616-617 (1936).
A study of dusts and silicosis was made in several porcelain factories, with special attention to the question whether sericite is a cause of silicosis, as propounded by Jones. Chemical analyses of raw materials and products were made. Microscopic examinations and x-ray powder spectra are inadequate. The analyses in one factory where silicosis occurred showed no mica in any form, including sericite. On the other hand, the dust in two coal mines was found to originate from a calcareous sandstone containing considerable muscovite. However, the workers were free of silicosis. The author concludes the mica is not the deleterious material.
Another factor in the causation of silicosis is the presence or absence of basic material. In the sandstone quarries in France and the Palatinate, where quartz and kaolin are abundant, silicosis is widespread. While it is rare in coal mines where the dust has similar composition. The author attributes the difference to the presence of lime in the mines, and similarly accounts for the absence of silicosis in basalt quarries and its rarity among granite workers (which does not check with American experience). He says: "The retardation of the effect of free silica by a base is not a mechanical action but prevents the chemical combination of the silica with the lung tissue. Mineralogy shows that silica is easily displaced from combination. In the same way, the presence of a base would prevent this combination from taking place." The question whether alumina has similar retarding effect is then discussed, and the author's answer is that the prevalence of silicosis in potteries where the alumina content of raw materials is high shows that it has not.

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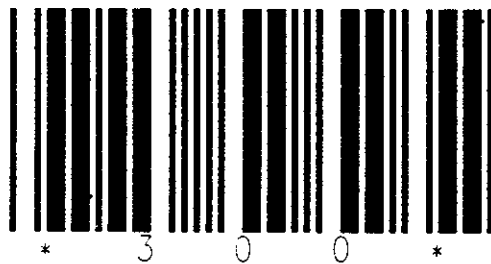
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