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tained in a second wash bottle. By passing a known quantity of air through these two solutions, sulphur dioxide and hydrogen sulphide at concentrations, respectively, of 0.017 to 0.6 mg. and 0.092 to 0.6 mg. per liter can be evaluated with satisfactory accuracy by determining the sulphuric acid formed.—P. D.

INJURIES FROM URSOL IN THE FUR INDUSTRY. *R. L. Mayer and M. Förster. Abstr. as follows from Zentralbl. f. Gewerbehyg., June, 1929, N. S. vol. 6, pp. 171–178, in Bull. Hyg., July, 1930, vol. 5, p. 571.*

Ursol is the commercial name given to metaphenylenediamine and is regarded as a frequent cause of dermatitis. The authors emphasize the importance of functional tests applied to the skin in addition to mere reliance on the family and occupational history. If such testing is applied, the number of cases alleged to be due to ursol falls considerably. The diagnosis "ursol poisoning" from the history is no more to be relied on than a diagnosis of lead poisoning from the presence of punctate basophilia.

Altogether 181 persons employed in the fur trade were examined, of whom 111 in the course of their work had suffered from dermatitis or asthma. Of these 111, 45 per cent. showed existence of eczema and 38.7 of asthma; a combination of the two was found in 16.2 per cent. On testing their skins with a small piece of 2 per cent. paraphenylenediamine vaseline, 44.1 per cent. showed hypersensitization to ursol. Forty-six per cent. of those with eczema gave positive reactions, 30.2 of the asthmatics, and 72.2 per cent. of those who showed presence of both conditions.

The authors regard 44.1 per cent. as directly due to ursol and 55.8 per cent. doubtfully so. The general question of sensitization of the skin is then discussed in particular relation to ursol.—T. M. L.

DUST HAZARDS AND THEIR EFFECTS

COAL-MINER'S LUNG. AN INVESTIGATION INTO THE ANTHRACOTIC LUNGS OF COAL-MINERS IN SOUTH WALES. *S. L. Cummins and A. F. Sladden. Jour. Path. and Bacteriol., 1930, vol. 33, pp. 1095–1132.*

This study of the black lung of coal miners is unusually thorough. It is based on an examination of lungs from thirty-eight cases, many of whom died accidentally. In addition to the pathologic examination, analysis of thirty-four of the lungs was carried out. Details of these analyses are given, and form the most extensive series of such analyses yet published. Eleven excellent illustrations are given of the macroscopic and microscopic conditions seen. The paper opens with a useful historical résumé of earlier references to anthracosis and other forms of dust diseases. The authors hold that the blackness of coal miners' lungs is due to coal; any iron present is quite in subsidiary amounts. Frequently fibrosis is present, caused by dusts other than coal, and particularly by silica from intervening rocks. Such fibrosis blocks the lymph flow of the lungs, and coal dust collects in abundance in such lungs. Normally coal dust is fairly rapidly removed from

healthy lungs, but it may so collect in a fibrotic lung as to form a nodule of coal. At times local necrosis takes place, with cavitation quite apart from any tuberculous infection. Discharge from such cavities accounts for the black sputum of miners. Wherever the coal particles were found most concentrated, analysis showed the content of silica to be particularly high. The conclusion is reached that anthracosis is not a simple condition, but exists only as a subdivision of lung fibrosis usually due to silicosis. Probably the lungs of miners interfered with by different dusts account for the high death rates from pulmonary diseases, particularly bronchitis, among coal miners on certain fields, even though their death rate from tuberculosis is usually low. Nevertheless, a condition of anthracosis may be pronounced without interfering with the general health.—E. L. C.

ASBESTOS AND PULMONARY ASBESTOSIS. *V. Dhers. Méd. du travail, July, 1930, vol. 2, pp. 147–172; Sept., 1930, pp. 187–209.*

In this long article the author usefully summarizes information relating to the occurrence of occupational asbestosis. A history of the use of this mineral and its properties is given, together with its chemical composition. Then a description follows of the industrial uses to which it is put, during which dust may be generated, particularly during weaving operations. The lungs of workers become affected in direct proportion to the length of time they have been exposed to it, until after twenty years of work 80 per cent. are affected. Statistics and information as to the occurrence of lung diseases among asbestos workers in all countries, but particularly in Great Britain, are brought together. Reference is made to experimental work carried out by exposing animals to the dust, the results of which have not all been published. The lung condition is characterized by dyspnea, cough, and the presence of asbestosis bodies, possibly in the sputum, but more certainly in the lungs. These bodies are due to organic material deposited upon fine spicules of dust. The fibrosis caused is general throughout the lungs, which are so damaged as to lead to a fatal issue without any secondary infection necessarily occurring. Such fibrosed lungs may be detected by radiography; but the shadows thrown are not so coarse as those of silicosis.

Diagnosis must depend largely on a knowledge of the occupation of the worker. Prognosis generally is unfavorable, since treatment is unsatisfactory. The only thing to do is to abolish the dangers by suppressing the hazard. Efforts made to this end are summarized. The article is accompanied by a full bibliography containing fifty-six references. It ought to be a useful contribution to which anyone interested in asbestosis will turn.—E. L. C.

PULMONARY ASBESTOSIS. A REPORT OF A CASE AND A REVIEW. *W. B. Soper. Am. Rev. Tuberc., Dec., 1930, vol. 22, pp. 571–584.*

A man of 30 years was admitted to the William Wirt Winchester Hospital, West Haven, Conn., on May 26, 1929, at which time he had been exposed to asbestos dust over a period of about thirteen years. No indications of the severity of the exposures (*i.e.*, dust

concentrations) are given. He was admitted to the hospital four months after he had stopped working.

At that time his weight was 142 pounds, or 8 pounds below his "best weight." Physical examination showed 5 cm. chest expansion, considerable dyspnea particularly on exertion, sputum occasionally blood streaked but negative for tuberculosis, occasional râles, burning pain over the whole chest, nervousness, irritability, and constipation of three months' standing. Several nontender "asbestos corns" were seen on the hands. Blood picture and urine showed nothing unusual. X-ray picture was distinctive of the cobweb appearance noted previously by Wood (see *Tubercle*, 1929, vol. 10, p. 353).

The patient was discharged on July 2, 1929 and reexamined on April 9, 1930. During the nine months' interval he had rested at home. Dyspnea had increased, the burning pain in the chest had been superseded by dull, drawing pain above the left breast, and weight had increased from 142 to 162 pounds. Respirations were 25 and "even ordinary speech produced shortness of breath"; the complexion was leaden, with noticeable cyanosis of the ears, hands, and skin on pressure; chest expansion was 1.5 cm. (as compared with 5 cm. nine months previously). Râles were considerably more definite and more widespread than nine months earlier.

Among the author's conclusions are the following:

"The most common single symptom is dyspnoea. This and the other symptoms are essentially those of a progressive generalised lung fibrosis.

"The physical signs of uncomplicated pulmonary asbestosis are substantially those of generalized fibrosis of both lungs and basal pleurisy.

"X-ray examination is of great value in establishing the diagnosis.

"Asbestos contains but a very small amount of free silica but probably conduces to a more hasty evolution of any accompanying tuberculosis, as in the better understood forms of silicosis.

"An immediate diagnosis at autopsy is said to be made possible by simply squeezing out upon a slide a drop of lung juice from the fibrosed tissue and covering with a cover-glass. The asbestosis bodies in large number are readily visible under the microscope." —P. D.

PNEUMONOCONIOSIS DUE TO FLUE DUST. *W. E. Cooke. Brit. Med. Jour., Nov. 15, 1930, vol. 2, pp. 816–817.*

Detailed analyses are given of boiler scale and flue dust, and also of dust particles found in the lungs of a man who had been a boiler cleaner for 113 months. He died from tuberculosis, while X-rays suggested advanced pneumonokoniosis. The lungs were fibrosed throughout, and dust and dust cells were found in excessive amount. Although the silicious matter in the pulmonary dust was proportionately less than in the flue dust, the occupational exposure is held to have certainly originated the pathologic condition. The process of flue cleaning is not favored; during twelve years twenty-seven men have been employed, and have given up the work. Excepting two of these, one of whom worked only one day and the other one week, the average period of employment was fifteen months, while the nearest approaching the period of the deceased man was thirty-six months.—E. L. C.

A STATISTICAL STUDY OF PULMONARY CHANGES FOUND AMONG MINERS WORKING IN ROCK DUST. *A. Policard and E. Martin. Bull. Acad. de méd., 1930, vol. 104, pp. 333–343.*

A clinical and radiographic examination was made of 175 men using rock drills in mines in the basin of the Loire, some being X-rayed on several occasions. As many men came for examination on account of illness, the percentage of lung trouble found cannot be held to be representative. Nevertheless, when divided according to length of work, nearly 50 per cent. were normal with less than one year of work, as compared with 25 per cent. with ten years or over of work. No information as to age is given, which might quite invalidate the findings. Two certain points emerge: one is that even ten years of exposure to mine dust does not necessarily cause pulmonary fibrosis; and the other, than an unusual number of cases of open tuberculosis were discovered. No X-ray picture diagnostic of silicosis was found; but progressive changes in the appearances were of value in detecting the silicotic condition. The authors hold pulmonary silicosis to be a particular instance of tuberculosis developing under special conditions.—E. L. C.

SOME ASPECTS OF SILICOSIS. *P. Heffernan. Tubercle, 1930, vol. 11, pp. 481–489.*

A discussion is presented, rather than new facts. The idea is advanced that silica may react by toughening lung tissue generally, while nodular fibrosis occurs at the site of lesions of which tuberculosis is the most common agent. Such nodules may be radiologic indicators of more extensive silicotic changes taking place, but not casting visible shadows. Silica enters into certain forms of life, and so may be expected to interact with protoplasm. The reaction may be expedited by such a medium as alkaline soap, with acute silicosis as a result; normally the reaction is a chronic process. Its development is associated with increasing dyspnea and dry pleurisy. Should tuberculosis not supervene, cardiac hypertrophy and failure ends the scene. Physical signs alone are not diagnostic; nor is a skiagram; a history of exposure to risk and symptoms must also be present. The fibrotic changes are progressive and cannot be inhibited. Coal and certain other dusts may facilitate the exit of silica; but silica has some power of bringing about the retention of dusts otherwise excreted. Prevention of the risk must be effected; it lies in keeping inhaled air free from a suspensoid system of silica-in-air, which behaves, physically, as a gas. The risk may be lessened by minimizing exposure to tuberculous infection.—E. L. C.

WOMEN AND CHILDREN IN INDUSTRY

WOMEN IN 5-AND-10-CENT STORES AND LIMITED-PRICE CHAIN DEPARTMENT STORES. *M. E. Pidgeon. U. S. Dept. Labor, Women's Bur., Bull. No. 76, 1930, pp. 56.*

This bulletin covers personal infor-

DUST HAZARDS AND THEIR EFFECTS

CONGRESS OF INDUSTRIAL MEDICINE. *Foreign Letter (Italy), Jour. Am. Med. Assn., Feb. 14, 1931, vol. 96, pp. 542–544.*

This letter contains an account of the Ninth Congress of Industrial Medicine held in Rome. The subject before the Congress—"The Problem of the Respiratory Pathology of Dusts"—was divided into several parts. Caccuri of Naples discussed the etiology and pathogenesis of respiratory diseases due to dust; Coppa of Naples, the pathologic anatomy and the clinical aspects of respiratory dust diseases; Pastena, also of Naples, radiologic research in pneumonokoniosis. Abstracts of these papers are included in the letter. Communications were also presented concerning the respiratory diseases of coal heavers and of workers in hemp.—K. R. D.

ASBESTOSIS BODIES IN THE SPUTUM: A STUDY OF SPECIMENS FROM FIFTY WORKERS IN AN ASBESTOS MILL. *F. W. Simson and A. S. Strachan. Jour. Path. and Bacteriol., Jan., 1931, vol. 34, pp. 1–4.*

The result is reported of examining sputum from fifty workers who were intensively exposed to asbestos dust in a mill in South Africa. Asbestosis bodies were easily demonstrated by direct thick films. They were found best by examining specimens taken early in the morning; in the evening the mouth and nasal pharynx were too loaded with dust for the bodies to be easily detected. The sputum was always mucoid in character, often resembling egg albumin. No tubercle bacilli were found. The asbestosis bodies were intracellular, and their study suggested that they were formed by the deposition on asbestos fibers of iron containing substance elaborated by the cell. The shortest exposure to be followed by the appearance of the bodies was five months.—E. L. C.

FURTHER OBSERVATIONS ON PULMONARY ASBESTOSIS, WITH SPECIAL REFERENCE TO ASBESTOS DUST AND THE CURIOUS BODIES FOUND IN THE LUNGS. *W. E. Cooke and C. F. Hill. Abstr. as follows from Jour. Roy. Micr. Soc., 1930, vol. 50, p. 15, in Physiol. Abstr., Feb., 1931, vol. 15, p. 626.*

In a case of suspected pulmonary asbestosis sections of the lung or macerations of the tissue revealed a large amount of fine granular black dust, but the fine spicules of fiber which make up the bulk of asbestos dust were not found. There were present, however, large fragments (5 to 360 microns) closely resembling in size, shape, and color various constituents of asbestos dust. These large fragments are found in the lung embedded in fibrotic or necrotic areas of which they are the cause. In addition, curious bodies were present which are believed to be diagnostic of pulmonary asbestosis. These bodies were eventually shown to consist of a central core of asbestos fiber enclosed in adsorbed débris.—C. K. D.

ANTHRACOSIS, SILICOSIS, AND TUBERCULOSIS IN COAL-MINERS. *S. L. Cummins. Lancet, Jan. 31, 1931, vol. 1, pp. 235–238.*

The claim is made that anthracosis is a dual condition, in which coal dust

is retained in the lungs owing to a state of fibrosis with lymph blockage. The pathologic condition arising from dust inhaled together with the coal and also chemical analyses are similar, so far as the content of silica, to those found in cases of pure silicosis. In certain cases of "hard heading" work, where rocks rather than coal are mainly tackled by the miners, silicosis develops which is indistinguishable from that seen in other cases of silica risk. But when coal has been the chief dust inhaled, no tendency to succumb from tuberculosis is found; and the suggestion has been offered that coal dust exerts a protective influence against tuberculosis. This idea is, however, not definitely accepted; at least, the suggestions to explain why the coal miner has a low mortality from tuberculosis, although his lungs show definite fibrosis, are not accepted. Professor Cummins now puts forward the possibility that carbon particles in the lungs adsorb the active principle of tuberculin. He points out that such adsorption occurs *in vitro*, since he has found that finely powdered dust of anthracite coal is capable of adsorbing a large proportion of the active principle from old tuberculin diluted with normal saline solution. Whether or not carbon dust particles bathed in the tissue juices retain this adsorbing power requires further investigation. Some clinical facts appear to favor the theory. Certainly, negative sputums are more common in clinically tuberculous coal miners than in other clinically tuberculous adult males. Although any excessive liability to tuberculosis is absent in the case of coal miners, a high mortality from bronchitis among old colliers is present, which, in all probability, is closely associated with the life of exposure, not only to coal dust but to stone dust as well.—E. L. C.

OBSERVATIONS ON THE DIFFERENTIAL DIAGNOSIS OF SILICOSIS BY MEANS OF THE ROENTGENOGRAM AND AN INVESTIGATION INTO THE RISKS OF SAND BLASTERS. *Lochtkemper. Abstr. from Arch. f. Gewerbepath. u. Gewerbehyg., 1930, vol. 1, pp. 271–302, in Ber. ü. d. ges. Physiol. u. exper. Pharmakol., Oct. 8, 1930, vol. 56, p. 182.*

The author distinguishes roentgenologically the various forms of silicosis, pneumonokoniosis caused by quartz dust. The first stage is characterized by enlarged hilum glands and by increased vascular markings; the second stage, by numerous infiltrated and isolated nodules and increased induration; the third stage, by the so-called "snowstorm" effect, which is partly an isolated and partly a confluent mottling.

The rate of dust inhalation and the concentration of the quartz content are important in the resulting roentgen picture. Among workers in industries whose dust contains free silicic acid—sandstone grinders, stonecutters, sand blasters, porcelain workers, and stone hewers—there are found in the X-ray picture of the lung, near the enlarged hilum, small, sharply defined shadows from pinhead to pea in size, some of which are isolated and others joined by fine streaks. The thickness of the nodules is greater in sandstone grinders and stonecutters than in porcelain workers and stone hewers, and in occupations in which a mixture of quartz and clay dust is inhaled. In the further development there is a gradual increase in the small shadows with an

incipient conglomeration to form larger connected shadows and a gradual coalescing, phenomena which are associated with stonecutters, wet sandstone grinders, and sand blasters. The circumscribed shadows appear less clearly in the lungs of stone hewers. Weak, diffuse, and cloudy shadows are characteristic of the coal dust lung; silicotic shadows are, however, for the most part, also evident. The X-ray picture of the schamotte stoneworkers shows less nodule formation, and a somewhat greater expanse of connected shadows.

In the second part of the paper are reported the results of the examination of twenty-five sand blasters, twenty of whom had indications of silicosis in different stages.

THE EFFECT OF PROLONGED EXPOSURE OF THE SILICEOUS SPICULES OF A WATER SPONGE (*Spongilla Fragilis*) TO THE ACTION OF ANIMAL TISSUES: A CONTRIBUTION TO THE PATHOGENESIS OF SILICOSIS IN MAN. *R. G. Mills. Am. Jour. Hyg., Jan., 1931, vol. 13, pp. 224–234.*

The spicules of the fresh-water sponge, *Spongilla fragilis*, are composed of opal, a form of hydrated silica, which is similar to quartz in its chemical reactions. These spicules, when introduced into the tissues of animals, are slowly but definitely dissolved, proving conclusively that silica is soluble in the tissue fluids of animals, and presumably of man. This confirms the assumption held by many students of silicosis that particles of silicious material are gradually converted into the colloidal state and as such exert a fibroplastic influence on the lungs of miners and other exposed industrial workers. Definite fibrosis of the lung of a dog into which the spicules had been introduced suggests that there is a concomitant injury attributable to the disappearance by solution of the silicious elements of the spicules.—*Author's Summary.*

OCCUPATIONAL AFFECTIONS OF THE SKIN AND SPECIAL SENSES

OCCUPATIONAL ECZEMA. *M. Oppenheim. Arch. Dermat. and Syph., Feb., 1931, vol. 23, p. 346.*

The dermatitides may be divided into the following groups: (1) In dermatitis, *sensu strictiori*, the skin is able to deal with the irritant; almost everyone's skin reacts to certain strong irritants (*e. g.*, croton oil); even if, with slight variation, the course is acute, healing soon follows the removal of the poison. (2) In toxicoderma there is a congenital or acquired idiosyncrasy. The slightest irritant evokes it. The incubation period is short. The effect is marked and the course acute. (3) In eczema the entire skin is sensitized. For this reason the intensity of the irritant may be slight. The development of dermatitis is gradual. The course is prolonged and healing delayed.

Occupational eczema is among the most frequent of eczemas, the most frequent of skin diseases, and the most frequent of all diseases. The predisposing factors are important: immersion of the hands in water, soap, alkali or acid, and the influence of heat, cold, and mechanical action. Occupational eczema is allergic, but the predisposing factors mentioned play an important part. Dermatitis artificialis acuta re-

absorption of the tar. On the other hand, removal of fat from the skin by preliminary treatment with petroleum ether produced an opposite effect, the number of tumors being decreased.

As the authors point out, other investigators have obtained results directly contrary to those described in this paper.

FURTHER INVESTIGATIONS ON THE TREATMENT OF TAR TUMORS IN MICE WITH CERTAIN AMINO-ACIDS. *F. Vlès, A. de Coulon, and J.-L. Nicod. Abstr. as follows from Compt. rend. Acad. d. sc., 1930, vol. 191, p. 350, in Am. Jour. Cancer, April, 1931, vol. 15, p. 898.*

One hundred and seven mice with tar cancer (verified by biopsy) were given an equimolecular mixture of alanine, cystine, and proline by mouth, subcutaneously, or by both routes. The dose was 0.03 to 0.075 gm. daily. In twenty-six cases the tumor disappeared entirely; in thirty-seven there was partial regression; in nine the tumor continued to grow, and in thirty-five animals it remained stationary. This last group, however, contained a number of mice that died less than fifteen days after treatment was begun and before it had had time to exert its full effect. Among forty-two controls not a single case of regression had been discovered.

A mixture made up in the proportion of two molecules of d-glutamic acid to one of l-cystine promised to be even more successful. Among twenty mice treated, the carcinoma disappeared in ten and regressed partially in six more; three tumors continued to grow, and one remained stationary.

DUST HAZARDS AND THEIR EFFECTS

CONTRIVANCES FOR DUSTLESS FILLING AND PACKING OF DUST PRODUCING MATERIALS. *Beihefte z. Zentralbl. f. Gewerbehyg., no. 19, 1930, pp. 48. Reviewed as follows in Bull. Hyg., Nov., 1930, vol. 5, p. 897.*

This publication on a question of very great hygienic importance deals with the engineering side of the subject and as such is too technical to be of direct service to the medical officer of health. The author lays stress on the dual aspect of the problem: first, the importance of catching up the dust as soon as it is formed and rendering it innocuous; and secondly, the method of dealing with it afterwards. Naturally, the treatment will differ according to the industry, the nature of the dust, its quantity, and whether it is of sufficient commercial value to recover, or whether it has to be got rid of as a waste product. The work is amply illustrated both by photographs, and diagrams, all of which are well and clearly reproduced.—H. H. S.

RECENT VIEWS ON PNEUMONOCONIOSES. *E. L. Collis. Proc. Roy. Soc. Med., March, 1931, vol. 24, pp. 531–540 (Epidemiology and State Medicine, pp. 13–22). (Abstract by author.)*

This article presents a summary of work done with regard to dust diseases since the author's Milroy Lectures, 1915. Studies have shown that silicosis may be advanced without impairing health; but it may itself end fatally. Generally superimposed

tuberculosis hastens the termination. Such infection may precipitate latent fibrosis. Other dusts may set up fine, generalized fibrosis, seriously impairing the pulmonary functions, but not predisposing to tuberculosis. Asbestos is particularly injurious; other silicates, such as basalt, are harmful. Pure coal dust seems to be quite neutral; but raw coal often contains sufficient minerals to originate fibrosis; while fibrosis may so retain coal particles in the lungs as to block the lymph passages and impair function. Radiography displays the presence of fibrosis, but gives no clue to the causative factor. When silicosis is present the shadows tend to be nodular, superimposed upon pronounced arborization. Reference is made to recent mortality records associated with different dust hazards. Clinical and pathologic studies are summarized, which are concerned with dusts of iron ore, cement, coal, asbestos, and marble.

THE PRESENCE OF ASBESTOSIS BODIES IN THE FAECES IN A CASE OF PULMONARY ASBESTOSIS. *S. R. Gloyne. Tubercle, April, 1931, vol. 12, pp. 158–159.*

This short note describes how asbestos bodies in cases of pulmonary asbestosis may be found by emulsifying the feces in four to five times their volume of 5 per cent. formalin in saline and then centrifuging the product, which will be found to contain a scanty number. The bodies, which have probably been swallowed in sputum, show less than usual of the typical deep-yellow coloring matter, as though the pigment were partially destroyed by intestinal juices.—E. L. C.

SILICOSIS AND COAL-MINING. *J. S. Haldane. Tr. Inst. Min. Eng., 1931, vol. 80, p. 415.*

In earlier papers[1] the author concluded (*a*) that no danger to health would arise from stone dusting, provided suitable shale or other material was employed; and (*b*) that the unusually high bronchitis mortality rate among colliers in the higher age groups is due mainly to the strain on the lungs caused by muscular exertion. In the present paper Dr. Haldane reviews his conclusions in the light of the full data contained in the Registrar-General's Decennial Supplement for 1921 to 1923.

The phthisis death rates for the various groups of underground workers are distinctly lower than those for all occupied and retired civilian males. Even in that group engaged in making and repairing roads, which includes the work of ripping stone roofs and driving roads in stone, the death rate from phthisis at all ages above 25 is lower than for the standard population. It is mentioned, however, that in exceptional cases great risk may occur among men driving roads in highly silicious rock, particularly when machine drills are used. The Mines Act of 1911 required certain precautions in the use of these drills, but the clause was practically ignored. As a result, cases of silicosis have been discovered in the Somerset and South Wales coal fields.

The thesis is advanced that the inhalation of coal dust and shale dust, as among coal miners, coal boat loaders, or cement workers, stimulates

[1] The Effects of Dust Inhalation. Tr. Inst. Min. Eng., 1917–1918, vol. 55, p. 264. The Health of Old Colliers. *Ibid.*, 1915–1916, vol. 51, p. 469.

the phagocytic activities of the lung, and thus confers a relative immunity from phthisis. As compared with coal, shale, or clay dust, the dusts of china clay, slate, and feldspar are very slowly eliminated, and it is suggested that this explains why neither the china clay used in potteries nor the feldspar in true granite protects against silica. Dr. Haldane dissents from the idea put forward at a recent Congress at Johannesburg, that the risk of silicosis is simply proportional to the proportion of free silica in the dust. Instead, he suggests that the action of free silica in paralyzing the phagocytes is commonly overcome by the action of other substances in eliminating them, but where these latter substances are absent or insufficient the silica action is predominant.

The view that the high bronchitis mortality is due to heavy muscular work is reasserted, although it is conceded that "it seems practically certain that excessive inhalation of coal-dust or shale-dust must cause bronchitis, and ought therefore to be avoided." In a subsequent discussion Professor E. L. Collis quoted comparative mortality figures for miners in different coal fields. It was shown that the comparative mortality from bronchitis in 1921 to 1923 for miners (hewers and getters) varied between the extremes of 33.8 for Leicestershire and 139.3 for South Wales, as compared with 49.6 for all occupied and retired males. It was suggested by Professor Collis that these differences hardly accord with differences in the work done, and do not support the muscular exertion theory of Dr. Haldane.—T. B.

Silica Content of Normal and of Tuberculous Lung. *R. Piazza. Riforma med., March 9, 1931, vol. 47, p. 369.*

OCCUPATIONAL AFFECTIONS OF THE SKIN AND SPECIAL SENSES

Boils and Carbuncles among Miners. *S. W. Fisher. Lancet, April 4, 1931, vol. 1, pp. 750–751.*

The occurrence of boils among miners at one colliery led to an inquiry which covered forty-nine collieries; about 200 men were examined. The main factor in the causation of the boils was found to be a wet bulb temperature of 75°F. or over. At such temperatures the men work stripped, *i.e.*, wearing shorts and boots. Boils occurred more often on men who sweated profusely; their bodies were covered with a fine wet film of coal dust. The condition seems to be analogous to prickly heat and boils which occur in India in the rainy season. The fact that no men working in mines which had pithead baths were found to have boils, points to the value of cleanliness as a preventive measure. Some evidence of infection from man to man was found; cases followed one another in a given working stall. The offending organism was the staphylococcus, which under warm, moist conditions gained access to the open sweat glands and hair follicles.—E. L. C.

the workingmen of the country.—H. H. S.

PULMONARY ASBESTOSIS II. INCLUDING THE REPORT OF A PURE CASE. *K. M. Lynch and W. A. Smith. Am. Rev. Tuberc., June, 1931, vol. 23, pp. 643–660.*

In a survey of all available literature on the subject up to the present time we have collected 172 cases of pulmonary asbestosis. There are references to this subject in one or two abstracts, notably those of Bridge and of Sir Thomas Oliver, but specific cases are not enumerated. In four cases belonging to Wood's series the diagnosis was doubtful, and in the majority of others the diagnosis was based entirely on clinical and roentgen findings. There were twenty-seven in which the diagnosis was confirmed by the finding of the asbestosis bodies in the sputum, in the lung juice by puncture, or by necropsy. Necropsy has been made on eighteen cases. In three of these the disease was complicated by pulmonary tuberculosis, three by lobar pneumonia, three by bronchopneumonia, and one was a traumatic death. In four the authors failed to give a complete report, stating only that the necropsy confirmed the diagnosis. Including the very first case recorded, that of Murray in the Charing Cross Gazette of 1900, which apparently received little attention until resurrected by Cooke, there are now four records of necropsy on uncomplicated pulmonary asbestosis. Except those reported by ourselves, and those by Pancoast and Pendergrass, and four others by Simson from South Africa, these cases have all developed in the British Isles.—*Authors' Summary.*

A CASE OF PULMONARY ASBESTOSIS. *H. E. Seiler and M. D. Gilmour. Brit. Med. Jour., June 27, 1931, vol. 1, pp. 1112–1114.*

The pathologic findings are reported of a fatal case of asbestosis uncomplicated by pulmonary tuberculosis. The man died, aged 42, after twenty-three and one-fourth years of occupational exposure to asbestos dust in a textile mill. Death was due to cardiac failure secondary to back pressure from massive generalized fibrosis. Before death, cyanosis was pronounced; and diffuse fine mottling of a silicotic type was detected on X-ray. The macroscopic appearances were typical of dense pulmonary fibrosis, with other organs secondarily affected—dilated heart, nutmeg liver, and indurated spleen and kidneys. The fibrosis was generalized, with no nodules or whorls, as in silicosis; no polymorphonuclear cells were to be seen in the bronchi or elsewhere. Asbestosis bodies were abundantly present, and also a great deal of yellow pigment in the form of granules and globular masses. No tuberculous lesions were found.—E. L. C.

THE FORMATION OF THE ASBESTOSIS BODY IN THE LUNG. *S. R. Gloyne. Tubercle, June, 1931, vol. 12, pp. 399–401.*

Pictorial presentation is given in this short note of the formation of asbestosis bodies. They appear to be formed by the deposition of some organic material around an asbestos fiber; this material then fissures or cracks, giving rise to the appearance of segments; finally, fragmentation from the asbestosis body may separate portions. There is evidence that iron

is a component of the material deposited on the inhaled fiber. During the reaction which takes place, solution of a fraction of asbestos occurs; but evidence is lacking as to how the long asbestos particles, which are far longer than the diameter of a dust cell, are introduced into the pulmonary tissues. —E. L. C.

PNEUMONOCONIOSIS. THE DELAYED DEVELOPMENT OF SYMPTOMS. *J. A. Britton and J. R. Head. Jour. Am. Med. Assn., June 6, 1931, vol. 96, pp. 1938–1939.*

Most clinical and statistical studies of silicosis have been made on groups of men still employed in dusty trades. From such statistics one cannot say what is the late effect of short exposures. In this paper we have reported four cases of silicosis or silicosis and tuberculosis which developed many years after relatively short exposures. These instances suggest the necessity of revising the conception of the length of exposure necessary to produce the disease and make it seem probable that after relatively short exposures sufficient dust must be deposited in the lungs to set up a progressive fibrosis, which only after many years becomes sufficiently extensive to produce symptoms. They tend to indicate that men in the work develop symptoms only after many years of exposure, not because that length of exposure is necessary but because it takes a long time for the disease to develop. —*Authors' Conclusions.*

SILICOSIS. *T. H. Belt. Canadian Med. Assn. Jour., Dec., 1930, vol. 23, pp. 802–804.*

This is a general discussion of silicosis and of the various theories as to the method of action of silica dust in the lungs.—M. C. S.

DIAGNOSIS OF PULMONARY SILICOSIS. *C. Garin. Abstr. as follows from Presse méd., April 18, 1931, vol. 39, p. 568, in Jour. Am. Med. Assn., July 11, 1931, vol. 97, pp. 143–144.*

Garin states that most of the authors cited by him reported cases of silicosis in which tuberculosis was present. None of them attempted to bring out the differences between the two conditions. He stresses mostly the roentgen side of the question. In his roentgen studies he found that about 85 per cent. of the cases are those of a granular silicosis. He believes that the granular aspect of chest roentgenograms is not positively that of pure silicosis and it should not serve as an exclusive base for diagnosis of silicosis. The diagnosis between simple silicosis with a granular picture and pulmonary tuberculosis is, however, easy: the patients with silicosis have no fever and the condition is progressive. On the other hand, diagnosis becomes almost impossible in cases of silicotuberculosis, in which the tubercle bacillus interferes with the development of a pure silicosis. In such cases it is impossible to pronounce the patients sick with silicosis or with pure tuberculosis. One can only make a diagnosis of silicotuberculosis.