

FILE NAME: Owens Illinois Library (OWL)

DATE: 1934

DOC#: OWL012

DOCUMENT DESCRIPTION: Abstracts from The Journal of Industrial Hygiene and Toxicology

THE JOURNAL OF "INDUSTRIAL HYGIENE

EDITORS

DAVID L. EDSALL, M. D., S. D., United States

EDGAR L. COLLIS, M. D., M. R. C. S., Great Britain

VOLUME XVI

JANUARY, 1934—NOVEMBER, 1934

PUBLISHED BY

THE WILLIAMS & WILKINS COMPANY

Baltimore, Md.

	PAGE		PAGE		PAGE
ARSENIC poisoning, chronic, studies in, arsenic content of "mosquito incense" (Li and Yang).....	50	BLADDER, aniline tumors of, symposium on (Ferguson, Gehrmann, Gay, Anderson, and Washburn).....	76	CARBON MONOXIDE intoxication, hazard and Mellissin poisoning (Barrett).....	
ARSENIURETTED HYDROGEN, industrial arsine poisoning (Schrader).....	123	BLOOD, fats and lipoids of, effect of benzol on (Nikulina and Getmann).....	121	poisoning, cyanide and, thionate and methy (Draize).....	
phosphine and arsine in air, reagent for detection of (Weber).....	103	lactic acid in man during rest (Cool and Hurst).....	17	poisoning, experimental (Fetzer and Weiland).....	
ARSINE, <i>see</i> Arseniuretted Hydrogen		pH of, after muscular work, changes in (Margaria and Talenti).....	37	surveys of two garages (bell and Farber).....	
ASBESTOSIS bodies (Berger).....	56	pressure of young men over a 7-year period, changes in (Diehl and Hesdorffer).....	37	CARBON TETRACHLORIDE effects of (Willcox and Desoille).....	
memorandum on (Merewether).....	56	BODY MASS and growth ratio in Viennese school boys and girls (Brezina).....	141	intoxication by (Duve and Desoille).....	
pulmonary (Stock).....	103	BURNS, medical treatment of men burned in colliery explosions.....	14	poisoning, acute, fatal (cini).....	
pulmonary, and silicosis; pneumoconiosis (Bromley, Wood, and Ellman).....	102	BUTCHERS, two pork, unusual ungual condition in (Oppenheim).....	63	poisoning, toxic amblyopia accompanying physiologic anes in (Wirtschauer).....	
ASPHYXIA, resuscitation (Henderson).....	118	BUTYLACETATE, chronic occupational toluol poisoning, sequelae? (Fühner and Pietrusky).....	45	CARCINOGENETIC agent tumors; chemistry and aspects (Gehrmann).....	
ASPHYXIA, <i>see also</i> under Carbon Monoxide poisoning, Gas poisoning		CADMIUM POISONING, industrial (Fühner and Blume).....	121	chemicals (Sannie and T oils, selection of nonfrom (Twort and Lyth).....	
ASTHMA, baker's, and allergic diathesis (Anton).....	137	CAISSON DISEASE, case in which hip joint was affected (Christ).....	107	CARCINOMA, bladder, etiology (guson).....	
pollen, air conditioning in treatment of (Nelson et al.).....	141	participation of eye in, especially cataract formation (Pfimlin).....	86	CARDIOVASCULAR SYSTEM (MAKARITSCHIEVA).....	
occupational (Moll).....	12	preventing illness in compressed air work (Oliver).....	138	CASTINGS CLEANERS and other dusty processes (Adler-Herzmark, Kleinstein).....	
ATMOSPHERE, <i>see</i> Air		prevention and therapy of (Sakai).....	137	silicosis among (Landau).....	
ATMOSPHERIC CONDITIONS, lighting and, in an Ontario public school (MacLean and Partridge).....	90	CALCIUM CYANAMIDE poisoning, case of (Linneberg).....	50	CATARACT formation, part the eye in caisson diseases (Pfimlin).....	
ATMOSPHERIC POLLUTION, report of Building Research Board for 1932.....	38	CALCIUM CYANIDE, ship fumigation at Port of Durban (Ross).....	50	glassmaker's, and relative types of cataracts (Mee).....	
BACTERIA, method of testing filters against (Drinker and Wells).....	139	CANCER, experimental production of, by dust obtained from tarred roads (Campbell).....	54	CELERY ITCH: dermatitis d in vegetable canning (H).....	
BAKER'S ASTHMA and allergic diathesis (Anton).....	137	mineral oil, and dermatitis, prevention of (Twort and Twort).....	62	CHAMOTTE PLANTS, sandstones and moulders in glass cosis in (Stetter).....	
BARIUM, pneumoconiosis from (Arigoni).....	55	occupational (Carozzi).....	94	CHEMICAL examination of tion to dermatitis (Cox industry, eyes of worker jutin).....	
BEE sugar worker's neuritis, symptoms of (Kroll).....	118	CANCEROUS or precancerous skin lesions in tar workers in New South Wales (Hunt).....	12	carcinogenic (Sannie and).....	
BELGIUM, first four years of insurance for occupational diseases in (Glibert).....	41	CARBON DIOXIDE in air, determination of (Kauko).....	11	CHEMIST and public health ().....	
BENZENE and toluene, inquiry into relative toxicity of (Ferguson, Harvey, and Hamilton).....	28	in production of oxygen poisoning, influence of (Hill).....	87	CHLORINE gas poisoning, mit after repeated (Pernice).....	
chronic action of, on organism (Vigdortschik).....	47	CARBON DISULFIDE poisoning, industrial, red cell resistance in (Germano).....	79	CHLOROSULFONIC ACID caution industrial accident ().....	
derivatives, lead and, examination of workers in (Serson, Meyer, Schneider, and Adler-Herzmark).....	48	CARBON IODINE as protection for mercury vapor poisoning (Stock).....	45	CHLOR-PICRIN in air, deteri (Blashek).....	
effect of, on fats and lipoids of blood (Nikulina and Getmann).....	121	CARBON MONOXIDE, animal experiments on chronic action of exhaust gases (Süpfle and May).....	119	CHROMIUM, pharmacologic ecological action of con (Mattuci).....	
hemopathies due to (Emile-Weil).....	5	hazard, another possible.....	98	CIRRHOsis of LIVER, can it lead poisoning (Bötttrich).....	
intoxication, chronic, prophylaxis of (Grünberg).....	121	hemoglobin concentration of workers connected with internal combustion engines (Jenkins).....	36	silicotic; reaction to fine sized quarts and alumi particles (Gardner and C toxic (Poindexter and Gree).....	
poisoning, chronic (Dassen).....	5	hygiene of (Süpfle, Hofmann, and May).....	80	CLIMATE, some psychop (Tyler).....	
poisoning, experimental, alterations in female generative system (Barzilai).....	120	in air, improved analyzer for (Frevert and Francis).....	98		
poisoning, experimental, gas chamber for (Lifschitz).....	133	in inspired air, acclimatization (?) of animals to 0.3 per cent. (Campbell).....	81		
toxicity of different (Lestschinskaja).....	46				
experimental investigation of (Baltotta).....	79				
BENZOL, <i>see</i> Benzene					
BENZOLISM in pregnancy, experimental studies in (Barzilai).....	120				
BERYLLIUM, action of fluor-beryllium on organism (Samachovskaja, Marzinkovsky, and Sirojetschkovsky).....	123				

The committee is, however, of opinion that the incidence of the disease in the Kolar Gold Field is much smaller, and that it takes a much longer time to develop signs and symptoms than in South Africa. This is evidently due to the fact that the percentage of free silica contained in Kolar rock is 5 to 20, as compared with the much larger percentage of 43 to 98 obtaining in South African samples. Further investigation is in progress. Under the circumstances I feel it necessary to contradict the statement made in the article referred to that "in the Kolar Gold Field, India, where the rock contains a large amount of quartz and sericite is absent, silicosis has hitherto inexplicably been unknown."

A COMPARISON OF THE DEVELOPMENT OF THE SPECIFIC NODULE OF SILICOSIS AND OF TUBERCULOSIS. W. S. Lemon and W. H. Feldman. *Arch. Intern. Med.*, March, 1934, vol. 53, pp. 367-378.

In order to obtain material for a comparative study of the respective morphologic reactions provoked in the lung by particulate silica and by bacilli of tuberculosis, intratracheal injections were given to two series of rabbits. The animals were killed at intervals of from 4 hours to 4 weeks after receiving the respective inoculums, and the course and character of the resultant cellular response were studied histologically. The results obtained appear to warrant the following conclusions:

1. The character of the cellular response to the irritative influences of particulate silica and to bacilli of tuberculosis is essentially the same; both promote the formation of characteristic tubercles.

The properties of the provocative agent responsible for the production of the silicotic tubercle preclude the formation of a structure characterized by continuous progression. This contrasts markedly with that formed as a consequence of the injection of bacilli of tuberculosis, which is usually of a progressive, destructive nature.

3. The similarity of the structural unit or tubercle invoked experimentally in response to particles of silica and to bacilli of tuberculosis is so striking as to make their certain differential identification impossible by ordinary morphologic criteria.

4. Although the pathologic characteristics of the two processes are practically identical during the early period of cellular progression, significant structural differences become evident as the duration of the diseases is extended.—*Authors' summary.*

MINERS' PHTHISIS, WITH DISCUSSION. L. G. Irvine. *Jour. Chem. Met., and Min. Soc. So. Africa*, Mar., 1934, vol. 34, pp. 304-316.

This is a concise review of the history of silicosis and the progress in its control in South Africa. Both medical and engineering measures for control are discussed. The importance of increased ventilation as an all-essential preventive of silicosis is emphasized. The high temperature-humidity problem in deep mines, and the alleviation of this problem by substituting dry drilling, with control of generated dust at the source for wet drilling processes, is considered.

Irvine considers both free silica and the silicates as possible causes of silicosis but refuses to judge their relative importance on the basis of existing data. He points with justifiable pride to the progressively decreasing incidence of silicosis in South African mines and predicts a continuation of this trend.

A comprehensive bibliography of the journal's publications dealing with miners' phthisis is found on page 316 of this issue.—*R. Page.*

PNEUMOCONIOSIS IN SULFUR MINE WORKERS AND THE ACTION OF SULFUR INTRODUCED IN THE RESPIRATORY TRACT. A. Cestari. *Abstr. as follows from Arch. intern. pharmacodynamie*, 1933, vol. 46, pp. 300-314, in *Chem. Abstr.*, June 20, 1934, vol. 28, p. 3795.

Intratracheal injection of sulfur suspensions brings about excretion of hydrogen sulfide in a period of 50 hrs. There are slight exudative and infiltrative reactions in the lungs. It is supposed that a substance similar to glutathione reduces the sulfur to hydrogen sulfide.

PNEUMOCONIOSIS.—A DISCUSSION. PART I. SILICOSIS. J. F. Bromley.

Sept., 1934]

AB

PART II. PULMONARY ASBESTOSIS. W. Wood.

PART III. PULMONARY ASBESTOSIS. Ellman. *Brit. Jour. Radiol.*, May, 1934, pp. 263-265.

In this discussion attention is paid particular to the radiological differences between silicosis and asbestosis. J. Bromley gives a short survey of silicosis stating the position as regards workmen's compensation and also with regard to the possibility of sericite being the causative factor rather than silica. He then describes the x-ray appearances which consist of characteristic discrete nodules spread over both lungs, but involving the upper and middle zones rather than the bases. At the earliest stage only the lung vessels are marked out as they divide and subdivide like the branches of a tree; next buds appear on the twigs; and lastly the leaves of the fibrous nodules—open out. Three cases drawn from a number of different occupations with exposure to dust are quoted, some of which are illustrated, and show various points. The same exposure to dust will cause different degrees of fibrosis in different persons; and similar x-ray shadows may be associated with different clinical stages. Asbestosis is characterized by innumerable fine striations and punctate stippling evenly distributed over the lower parts of both lungs, while the upper parts are left clear. There are no dense nodules. At first sight the condition looks less formidable than silicosis. In fact, a more serious clinical condition develops more rapidly than in silicosis, so that the average length of

DUST, SMOKE, FUMES, AND VAPOURS. PART I. ELIMINATION.

A NEW REAGENT FOR THE DETECTION OF PHOSPHENE AND ARSINE IN AIR. H. H. Weber. *Zentralbl. f. Gewerbehyg.*, Jan.-Feb., 1934, vol. 11, pp. 1-3.

At the instance of the International Board of Health a reagent which could be used by the layman for the determination of dangerous amounts of phosphene was sought for in order to avoid accidents from ferro-silicon. For this purpose the following was recommended: pieces of filter paper are soaked with a 5 per cent aqueous solution of mercury-cadmium iodide, dried and

✓ PART II. PULMONARY ASBESTOSIS. W. B. Wood.

PART III. PULMONARY ASBESTOSIS. P. Ellman. *Brit. Jour. Radiol.*, May, 1934, pp. 263-95.

In this discussion attention is paid in particular to the radiological differences between silicosis and asbestosis. J. F. Bromley gives a short survey of silicosis, stating the position as regards workmen's compensation and also with regard to the possibility of sericite being the causative factor rather than silica. He then describes the x-ray appearances which consist of characteristic discrete nodules spread over both lungs, but involving the upper and middle zones rather than the bases. At the earliest stage only the lung vessels are marked out as they divide and subdivide like the branches of a tree; next buds appear on the twigs; and lastly the leaves—the fibrous nodules—open out. Thirty cases drawn from a number of different occupations with exposure to dust are quoted, some of which are illustrated, to show various points. The same exposure to dust will cause different degrees of fibrosis in different persons; and similar x-ray shadows may be associated with different clinical stages. Asbestosis is characterised by innumerable fine striations and punctate stippling evenly distributed over the lower parts of both lungs, while the upper parts are left clear. There are no dense nodules. At first sight the condition looks less formidable than silicosis. In fact, a more serious clinical condition develops more rapidly than in silicosis, so that the average length of

dust exposure in fatal cases is only half as long. The disease is progressive with a bad prognosis, and state of the sufferer is more wretched, with troublesome dyspnea and emaciation. The third contribution is based on the author's paper in this Journal, 1933, 16, 165. He stresses the presence of asbestosis bodies and points out that if arranged in clumps in the sputum they indicate disintegration of lung tissue. E. L. Collis.

PULMONARY ASBESTOSIS. G. A. Stock. *Med. Bull. Veterans' Admin.*, 1934, vol. 10, pp. 126-129.

A case report.

A CONTRIBUTION TO THE STUDY OF SILICOSIS. Maché. *Bull. Acad. Méd.*, May, 1934, pp. 579-581.

The title is misleading as the subject reported is exposure to cement dust. Subjects not accustomed to such exposure were found to have their urinary pH disturbed; thus, after 10 minutes' exposure, an average normal pH of 5.1 became, 1 hour later, 5.6, and, 3 hours later, 5.8, returning to normal after 8 hours; after 30 minutes' exposure, it became, 1 hour later, 5.9, and, after 3 hours, 6.7, again returning to normal after 8 hours; after 1 hour's exposure, it became, 1 hour later, 7.1, and 3 hours later 7.0. Workmen employed in the manufacture of cement and so accustomed to prolonged exposure to the dust were found, on the other hand, to have become inured to its influence so that their urinary pH was quite unaffected.—E. L. Collis.

DUST, SMOKE, FUMES, AND VAPORS: THEIR DETERMINATION AND ELIMINATION

A NEW REAGENT FOR THE DETECTION OF PHOSPHENE AND ARSINE IN AIR. H. H. Weber. *Zentralbl. f. Gewerbehyg.*, Jan.-Feb., 1934, vol. 11, pp. 1-3.

At the instance of the International Board of Health a reagent which could be used by the layman for the determination of dangerous amounts of phosphene was sought for in order to avoid accidents from ferro-silicon. For this purpose the following was recommended: pieces of filter paper are soaked with a 5 per cent aqueous solution of mercury-cadmium iodide, dried $\frac{1}{2}$

hour in a drying oven at 80°, preserved dry in a test tube sealed with a rubber stopper, and moistened with a drop of anhydrous acetic acid before using. In the presence of .01 mg. per liter of phosphene in air, there results a distinct yellow reaction within 10 minutes, and in the presence of .002 mg. per liter of arsine the edges of the drops become a weak yellow. In the presence of both gases, the arsine changes the reaction more toward a brown.

According to the author, the physiological toxic threshold coincides with the

poses the name "baritinosi" (baritosis).—*L. T. Fairhall.*

✓ ASBESTOSIS BODIES. *P. I. Berger. Virchow's Arch., 1933, vol. 290, pp. 281-353.*

This paper gives a detailed study of the optical and histological nature of asbestosis bodies with excellent photomicrographs. Specimens from the lungs of a man who had been exposed to asbestos dust contained vast number of small needle-like particles identified petrologically as chrysotile-asbestos. Following Bragg's crystal lattice method, the author shows that the hydrated magnesium silicate of the original metallic atom is dissolved out by the acid body fluids leaving a shell of silicic acid. The form of the needles and their double refracting property remains unchanged. Asbestosis bodies form as a result of protein absorption by the needles and subsequent coagulation as a gel. Analogous substances are produced *in vitro* by introducing acid-treated asbestos into a solution of egg-white. The silicic acid shell of the asbestos needle is dispersed in the albuminous sheath of the asbestosis body, and gradually destroyed. Then follows resorption of the silicic acid-containing albuminous mass by the body fluids. Granular streaks of ferric oxide stain the bodies a brownish tint.—*L. Teleky.*

✓ A MEMORANDUM ON ASBESTOSIS. *E. R. A. Merewether. Tubercle, Nov., Dec., 1933, and Jan., 1934, pp. 69-81, 109-118, 152-159.*

This series of articles summarises present knowledge on the subject of asbestosis, upon which the author has had unique opportunities of acquiring information. The nature and uses of asbestos with the early recognition of asbestosis are first sketched. The disease is then described as a generalised pulmonary fibrosis, insidious in onset, irregular in course, and variable in mode of termination which is to a fatal issue, often from the onset of some acute infection; but, while the presence of asbestosis seriously impairs the chance of recovery, its existence does not predispose to the onset of such infections of which tuberculosis is one. Left to itself, ultimately the strain of maintaining the circulation through the partially strangled lungs becomes insupportable, general dropsy with an enlarged liver appears

and death ensues from slow heart failure. Many cases complain of cough and dyspnea and show duskiness or slight blueness of the lips. Both physical signs and symptoms are unobtrusive; and the fine pin-head mottled ground-glass appearance on X-ray contrasts with the more definite localisation of the "snow-storm" of silicosis; in fact a radiographic picture may be not only inconclusive but most misleading.

Asbestosis cannot be diagnosed on either physical examination or radiological alone; but with both a fair conclusion can be drawn. Yellowish elongated bodies with bulbous ends, of microscopic size, occur in the lungs of asbestos workers; they are indicative of exposure to asbestos dust; and when found in clumps, instead of individually, they point to disintegration of lung tissue and the presence of definite asbestosis. Exposure to asbestos dust for only 5 years has resulted in fatal asbestosis, but no steep rise in the incidence rate occurs until after 10 years' employment; after 20 years more than one-fourth have been found affected. The dust, once it is trapped in the lungs, remains to exert its fibrosis-producing powers for many years. While increasing concentrations of dust in the air reduce the period before asbestosis occurs, further increase in the dust does not further reduce the period after a certain high concentration has been reached; on the other hand, below a certain concentration disabling asbestosis will not occur. In the more dusty processes the fibrosis-producing period is commonly 11 years. Death from the disease is inevitable sooner or later, if life is not cut short by accident or some unconnected disease. The added risk to asbestos workers from pulmonary tuberculosis is limited and definitely less than for workers exposed to the dust of free silica. Details are given of the preventive measures now adopted to minimize the dust hazard and also of the scheme under which in Great Britain compensation is awarded to those who develop asbestosis. Evidence goes to show that the dust produced in the dusty processes can be kept below the threshold of danger; if this standard is conscientiously maintained, the disease should not occur; then the outlook for the industry is bright.—*E. L. Collis.*

THE FUNCTION OF THE HUMAN NOSE AS DUST FILTER. *G. Lehmann. Arb. physiol., 1933, vol. 7, pp. 167-175.*

The author blew into the noses of 26 experimental subjects dusty air at the rate 3 to 5 L. per minute, and allowed the air to come out through a tube held in the mouth. The dust content of the ingoing and outgoing air was determined by means of an apparatus constructed on the principle of the nimeter. The assumption was made that for the examination, the palate would remain in a passive position.

It was found that from 8.3 to 73.7 per cent of the dust was retained in the nose. Two of the subjects retained more than 50 per cent in the nose ("good noses"), six less than 25 per cent. In retaining dust, not merely the extent of the air passages but also the form as well as the quantity and nature of the secretions play an important part. The speed at which the dust goes through the passages and the dust concentration do not seem important. A dust high in silica was used.

Subsequent experiments were carried out on 62 miners most of whom had worked more than 10 years in rock. The 33 who were healthy averaged a dust retention of 50 per cent whereas the silicotics retained only 22 per cent. Thus, the opinion that the dust retaining power of the nose is of great importance in the origin of pneumoconiosis is confirmed.—*L. Teleky.*

THE PROGNOSIS OF SILICOSIS. *A. Böhm. Bochum. Beitr. z. Klin. d. Tbc., Dec. 1933, vol. 84, pp. 119-139.*

The author has observed 200 cases of silicotic stone cutters. All 200 had been out of this hazardous occupation for at least 5 years, half of them had given up their trade 5 years previously, and in some cases 10 years had elapsed since exposure. Silicosis progresses in 20 per cent of the cases having silicosis Grade 1, after giving up the profession; in 40 per cent of those having Grade 2, and practically always in those with Grade 3. The late stages of Grade 2 are correspondingly dangerous. The younger the worker, the more unfavorable is the course of silicosis; of 11 cases who had Grade 1 when they gave up the work, but which had advanced to Grade 3, ten were

THE FUNCTION OF THE HUMAN NOSE AS A DUST FILTER. G. Lehmann. *Arbeitsphysiol.*, 1933, vol. 7, pp. 167-176.

The author blew into the noses of 26 experimental subjects dusty air at the rate of 3 to 5 L. per minute, and allowed the air to come out through a tube held in the mouth. The dust content of the ingoing and outgoing air was determined by means of an apparatus constructed on the principle of the koinimeter. The assumption was made that for the examination, the palate would remain in a passive position.

It was found that from 8.3 to 73.7 per cent of the dust was retained in the nose. Twelve of the subjects retained more than 50 per cent in the nose ("good noses"), six less than 25 per cent. In retaining dust, not merely the extent of the air passages but also the form as well as the quantity and nature of the secretions play an important part. The speed at which the dust goes through the passages and the dust concentration do not seem important. A dust high in silica was used.

Subsequent experiments were carried out on 62 miners most of whom had worked more than 10 years in rock. The 33 who were healthy averaged a dust retention of 50 per cent whereas the silicotics retained only 22 per cent. Thus, the opinion that the dust retaining power of the nose is of great importance in the origin of pneumoconiosis, is confirmed.—L. Teleky.

THE PROGNOSIS OF SILICOSIS. A. Böhm-Bochum. *Beitr. z. Klin. d. Tbk.*, Dec., 1933, vol. 84, pp. 119-139.

The author has observed 200 cases of silicotic stone cutters. All 200 had been out of this hazardous occupation for at least 2 years, half of them had given up their trade 5 years previously, and in some cases 11 years had elapsed since exposure. Silicosis progresses in 20 per cent of the cases having silicosis Grade 1, after giving up the profession; in 40 per cent of those having Grade 2, and practically always in those with Grade 3. The late stages of Grade 2 are correspondingly dangerous. The younger the worker, the more unfavorable is the course of silicosis; of 11 cases who had Grade 1 when they gave up the work, but which had advanced to Grade 3, ten were

under 50 years of age. In the older men, the disease was not infrequently completely stationary. If the disease does not advance markedly in the first few years after giving up a dusty trade, the subsequent progression is gradual or comes to a standstill. Silicosis of Grades 2 and 3 point to a high disposition to tuberculosis; a progressive tuberculosis develops in 23 per cent of cases of Grade 2, and in 72 per cent of those with Grade 3. Usually this tuberculosis runs a cirrhotic course for a long time but finally is fatal.—L. Teleky.

THE CAUSE OF SILICOSIS: A DISCUSSION.

W. R. Jones, J. S. Haldane, E. H. Kettle, E. L. Middleton and J. Flett. *Brit. Med. Jour.*, Feb. 10th, 1934, p. 254.

The discussion was opened by Dr. W. R. Jones who put forward his contention (already noted in these Abstracts, Nov., 1933, 15, p. 129) that silica in the uncombined state was not the chief cause of silicosis, and that the chief cause was minute acicular fibers of sericite, a mineral which is abundantly present in all rocks known to have originated silicosis. Prof. J. S. Haldane strongly dissented from this suggestion; he held that in each of the instances quoted a mistake had been made. Prof. E. H. Kettle was not impressed with the negative evidence adduced; moreover, he had ample experimental proof that free silica and substances other than sericite were capable of producing lesions characteristic of the silica reaction. Dr. E. L. Middleton placed little reliance on the hypothesis advanced. Sir J. Flett held that the reaction to dust particles was due to their physical irritation rather than to chemical action. The discussion was adjourned.—E. L. Collis.

SILICOSIS AMONG CASTINGS CLEANERS. W.

Landau. *Abstr. as follows from Arch. f. Gewerbepath. u. Gewerbehyg.*, 1933, vol. 4, pp. 516-523, in *Bull. Hyg.*, Nov., 1933, vol. 8, p. 727.

A systematic examination of metal casting cleaners using pneumatic tools and not the sand blast has shown the great danger to health of this work. Of 126 men employed 66 per cent were found to have silicosis and 8 per cent had open pulmonary tuberculosis. Men employed in cleaning steel castings