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tory, that silica mixed with an inert dust causes a modified proliferative reaction.

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.—*Authors' summary and conclusions.*

THE PNEUMONOCONIOSES IN SOUTH WALES. *S. L. Cummins. J. Hyg. Camb., vol. 36, pp. 547–558 (Oct., 1936).*

In the South Wales coalfield, cases of more or less disabling lung conditions are frequently met with which cannot conscientiously be fitted into the category of "silicosis" as defined at the International Conference at Johannesburg in 1930. These cases cannot be certified for compensation under existing Statutes. In this paper the author indicates some of the pathological and histological features of lung specimens examined by him at Cardiff. An attempt is made to classify the pneumoconioses met with in South Wales into four types. Examples of Type 1 are silicosis and asbestosis; Type 2 includes silico-anthracosis and silico-siderosis; in Type 3 comes the anthracosis of coal trimmers and screeners; and Type 4 includes the fibrotic aggregations of inert dust occasionally present in individual cases of all types of pneumoconiosis, but especially frequent in silico-anthracotic persons. All three main types show some degree of lung injury attributable to dust accumulation, though the amount of fibrosis is minimal in pure anthracosis. Pneumoconiotic lungs of all types show appearances suggesting damage to the elastic structure of the lung, a finding which perhaps may have a bearing on the emphysema and dyspnœa met with. The observations appear to justify the conclusion that the emphysema associated with pneumoconiosis in South Wales is not necessarily only a compensatory effect of lung fibrosis, since it may be well marked in persons suffering from pure anthracosis with only minimal fibrosis. The precise etiology of this type of emphysema invites further investigation. Bronchitis is common in silicotic and silico-anthracotic cases, but seems to be the result rather than the cause of the pneumoconiotic state which renders the lungs specially liable to catarrhal infections. Ciliated epithelium is usually retained to the end, even in the worst cases of pneumoconiosis. The amount of fibrosis and lung damage in the pneumoconioses of South Wales varies directly with the intensity and duration of exposure to siliceous dust as expressed in the silica content on analysis of dried lung, and the number of "bright particles" seen with crossed prisms. (The paper is illustrated with 11 plates.)—*T. Bedford.*

ASBESTOSIS: A ROENTGENOLOGIC REVIEW OF 71 CASES. *H. R. Shull. Radiology, vol. 27, pp. 279–292 (Sept., 1936).*

The major emphasis is on roentgenologic appearances although reports of two autopsies are included in the study. The group consisted of 56 white males, 6 white females and 9 negro males. The average age was 34.4 years. Of the total number of 71 cases 16 were classified as "slightly advanced", 35 as "moderately advanced" and 20 as "markedly advanced". In the first group the exposure period varied between 1½ and 13 years with an average of 8+ years, and in the third group exposures varied between 1½ to 20 years with an average of 8.7 years (9.1 years if one short exposure of 1½ years is omitted).

Tuberculosis was not common; it was diagnosed in some degree, type not stated, in the following frequence: slightly advanced group 20%; moderately advanced, 11.4%; markedly advanced, 5%. The author does not believe that the operation played a significant rôle in the development or prognosis of asbestosis. Twenty-one of the group were re-examined 15 months after discharge from the industry. In the first group there was little tendency to progression, in the second, there was some in 2 of 7 cases re-examined; in the third group 4 of 5 cases showed no improvement.

The author tabulates the radiographic findings characteristic of asbestosis as follows:

1. Emphysematous type of chest.
2. Flaring of lower ribs.
3. Trachea not displaced.

4. Diffuse fine to coarse fibrosis reaching the periphery, interstitial in character, differentiating from the roentgenologic appearance of silicosis.

5. Hazy ground-glass appearance of lung fields.

6. Increased density of all pleural markings.

7. Shaggy appearance of the cardiac outline.

8. Tendency toward right-sided cardiac enlargement.

9. Disproportionate rise of left diaphragm.

10. Degree of cardiac involvement is not consistent with that of pulmonary involvement.

In his conclusions he states 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.—*Leroy U. Gardner.*

PATHOLOGY OF THE PNEUMOCONIOSES. *L. U. Gardner. N. Y. State J. Med., vol. 36, pp. 1377–1381 (Oct., 1936).*

The paper summarizes the anatomic changes produced by inhaled silica and other dusts which give rise to various roentgenologic manifestations and the complications resulting from coexistent infection. The clinical manifestations in each stage are also discussed. The reactions are discussed on the basis of the following classification which was published in the U. S. Public Health Reports for August 2, 1935.

Simple silicosis.

1. Discrete nodulation.

2. Conglomerate disease associated with discrete nodulation.

Silicosis with infection.

The following manifestations of infection are present upon a background of [illegible] nodulation:

3. The primary complex of [illegible] tuberculosis.

4. Localized foci of healed adult [illegible] tuberculosis situated in the apex [illegible] portions of the lung.

5. Aspiration or bronchogenic [illegible] losis, either productive or exudative [illegible]

6. Miliary tuberculosis.

7. Perinodular tuberculosis.

8. Chronic silico-tuberculosis.

The author's summary follows:

Accentuation of the linear markings in a pulmonary roentgenogram does not constitute a basis for a diagnosis of [illegible] The change is non-specific and even in persons with a history of prolonged exposure to silica, it may be due to other causes.

Simple silicosis is manifested by nodulation uniformly distributed throughout the lungs. This condition does not incapacitate for work; it does not produce symptoms. It is progressive to only a limited degree. A person with this type of reaction may continue in his regular employment although recognition of his condition supposes that every effort will be made to reduce the dust in which he works to a minimum concentration. He should be subjected to roentgenographic examination once a year to be certain that no infection is developing in his lungs.

Simple silicosis may also be manifested by massive areas of conglomerate disease usually associated with generalized discrete nodulation. Such cases usually have dyspnea and are definitely limited in their capacity to work. They should be given lighter jobs commensurate with their abilities. To discharge them will only make a hypochondriac and a possible litigant.

Uncomplicated silicosis is not fatal. Its predisposition to infection, in most instances tuberculosis, constitutes the real danger from this condition.

No person with open tuberculosis should be permitted to work in an industry where silica dust is created. An old employee with closed silico-tuberculosis that does not incapacitate him may be allowed to do light work in departments where no dust is generated. Sanatorium treatment may be tried but it is often ineffective. A young

employee with silico-tuberculosis [illegible] given the benefit of sanatorium t[illegible] Persons over forty with roentge[illegible] evidence of well-healed adult typ[illegible] lesions can be safely employed in a[illegible] with a silica hazard. Their only d[illegible] in the development of a massive c[illegible] nite type of fibrosis. But it is [illegible] that an industry that is having p[illegible] exam. examinations is cognizan[illegible] hazard and is making every effort [illegible] the dust concentrations in its pla[illegible] The younger the individual w[illegible] evidence of healed adult type tu[illegible] the less safely can he be expose[illegible] silicosis.

Roentgenographic evidence o[illegible] primary complex in a person ov[illegible] of age does not constitute groun[illegible] barring him from exposure to sil[illegible] Gardner.

THE DIFFERENTIAL DIAGNOSIS O[illegible] FROM OTHER PULMONARY DISE[illegible] Orenstein. *N. Y. State J. M[illegible] pp. 1388–1385 (Oct., 1936).*

Orenstein challenges the g[illegible] cepted thesis of increased i[illegible] tuberculosis in silicotic subje[illegible] this infection is the most comm[illegible] their death. He believes that [illegible] lesions which are diagnosed [illegible] berculosis in the roentgenogram[illegible] conglomerate manifestations o[illegible] cause of the absence of tuber[illegible] the sputum and because the in[illegible] feels itself so late in the c[illegible] silicosis. He criticizes the [illegible] dence for this belief in the cas[illegible] granite cutters, the Picher l[illegible] miners and the N. Y. City roc[illegible] points out that there may hav[illegible] in diagnosis and that the repor[illegible] of tuberculosis is too low (1.S[illegible] compatible with the statemen[illegible] silicotics die of pulmonary tu[illegible] *L. U. Gardner.*

CLINICAL FEATURES AND SIG[illegible] SILICOSIS. *A. J. Lanza. J. Med., vol. 36, pp. 1386–13[illegible]* The diagnosis of silicosis [illegible] characteristic roentgenogram[illegible] history of adequate exposure[illegible]

f these pulmonary condi-
nple silicosis; (2) infected
ulous or non-tuberculous);
licoses (silico-anthracosis,
etc.); (4) silicatoses (e.g.
iminitosis," "sericitosis");
ic pneumoconioses (e.g.
ase," anthracosis, etc.);
fibrosis not due to dust
is, syphilitic fibrosis, and
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ed only 38% of free silica.
ntains no more free silica
s shale dust used for dust-
.—*T. Bedford.*

ITALIAN INQUIRY INTO
Glibert. Bruxelles Méd.,
434 (1937).
a general discussion by
e Italian journal "Secondo

tas" (Oct.-Nov., 1936) on silicosis and its incidence in Italy.—*L. Teleky.*

PNEUMOCONIOSIS CAUSED BY TITANIUM. *L. Lenzi. Rass. di med. appl. lavoro indust., vol. 7, pp. 301–318 (Oct. 1936).*

The author subjected 28 guinea pigs to artificially produced titanium oxide clouds. Various groups of these animals were exposed for periods ranging from 2 hrs. to 4 months. Pathological examination of the lung tissue indicated that titanium oxide penetrates deeply, causing an inflammatory reaction which continues for some time. The author states that the pathology noted resembles that caused by exposure to pneumoconiosis-producing dusts, and proposes the name "titaniosis."—*J. M. Dalla-Valle.*

PATHOLOGY OF PNEUMOCONIOSIS: SILICOSIS; SILICOSIS AND TUBERCULOSIS; ASBESTOSIS; SIDEROSIS; ANTHRACOSIS. *O. Auerbach. Indust. Med., vol. 6, pp. 39–48, (Jan. 1937).*

In this article the author reviews briefly some of the literature on pneumoconiosis. He considers first the etiological factors, presenting the pros and cons of the controversial phases including the significance of sericite in silicosis. The evidence for the mechanical versus the physicochemical mechanism in producing the injury giving rise to the progressive fibrosis is discussed. Figures are quoted which indicate that the length of time of exposure may vary from 21 months to 20 years. Following such exposure, a number of years may elapse before clinical symptoms appear. One case referred to was exposed for 4 months and developed the first signs of silicosis 22 years later. Ten million particles per cubic foot of dust containing 35% free silica may be tolerated for a short time without danger. The size of particles which are dangerous range from 1–5μ, the smaller sizes being most effective. A table of statistics relating to the prevalence of silicosis and silicosis and tuberculosis in various industries is given. The gross and microscopic pathology of the lung is described, in which the author points out the spread of the dust by way of the phagocytic cells and the lymph system with resultant fibrosis. This fibrosis tends to be nodular. Numerous photographs illustrate the lesion. A very brief description of the more diffuse asbestosis fibrosis and the character of asbestos bodies is given. In the consideration of siderosis and anthracosis, the possibility of a silica complication in the fibrotic process is considered.—*R. Z. Schulz.*

SANDBLASTING AND SILICOSIS. *W. Bergerhoff. Arch. f. Gewerbepath. u. Gewerbehyg., vol. 7, pp. 156–181 (1936).*

The author has made clinical and x-ray examinations of 175 workers employed on sandblasting and found 100 fit for the work and 75 unfit. In 24 definite pulmonary tuberculosis was diagnosed. The workers were divided into two large groups, those who had only been employed on sandblasting and those who had previously been employed on other dusty work. On account of the ill effects from sandblasting most of the workers give up the work after a few years: estimation of the dust hazard is therefore difficult compared with grinders and stone hewers who work for longer periods. There is no doubt that sandblasting is the dustiest of all occupations and methods for efficiently removing the dust have so far not been very successful.

Effective protection can be obtained by masks and special helmets that are supplied with fresh air.

The medium and severe degrees of silicosis are far more frequently associated with tuberculosis than the early and slight cases. The diagnosis of silicosis caused by sandblasting is difficult. In the early stage it may be confused with diffuse fibrosis which is often found in quite healthy people. The physical signs of silicosis are surprisingly small. The x-ray picture shows extensive diffuse fibrosis and only coarse thickenings occur in severe cases. Simultaneous pulmonary tuberculosis changes the whole picture as in other kinds of silicosis. The blood picture affords no reliable grounds for differential diagnosis of silico-tuberculosis.

Susceptibility to silicosis has been shown to depend in large measure on the action of the nose. The worse the nasal filter is, so much the more silicosis develops, but observations on sandblasters have not confirmed this dependence; this may be

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. 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 that 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 agree 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.—*Air Hygiene Foundation.*

ETIOLOGY OF SILICOSIS. *V. Reichmann. Med. Klin., vol. 33, pp. 226-229 (1937).*

The author challenges the statements of Hollmann and Lochtkemper on silicotic changes resulting from inhalation of graphite and rust dust since to date no roentgenological, pathological or histological proof has been presented. Also [illegible] agrees with the view of Jones [illegible] Of the 20,000 cases of silicotic lung [illegible] he has seen, he found none that [illegible] caused by several years' exposure to [illegible] dust. Since he has for years held [illegible] that only quartz causes the disease [illegible] have been sent to him from all parts of Germany pictures of persons who [illegible] allegedly never exposed industrially to quartz. Silicosis was seen in a machinist, but careful questioning revealed that 20 years earlier he had had charge of unloading [illegible]. A bath attendant with silicosis had given sand baths, and part of his work involved straining the sand. A zinc galvanizer worked in a room in which sandblasting was done.

The decisive factors in a dust hazard are the quantity, free silica content, and fineness of the inhaled dust.—*L. Teleky.*

SILICOSIS IN SPAIN. *A. Oller. Arch. f. Gewerbepathol., vol. 7, pp. 383-387 (1936).*

In the Spanish Rodalquilar gold mines the gold is found in layers of quartz [illegible] though no sericite is present. These [illegible] are dry drilled and have been in use [illegible] for 6 years. In 1935, 10 workers died [illegible] silico-tuberculosis. Of the entire 110 workers there were found 12 cases of silicosis [illegible] Stage 1, 11 cases of Stage 2, and 9 of Stage 3. In a ceramic plant that employed 300 workers, 16 cases of silicosis were seen. Hygienic improvements have been made in both industries.—*L. Teleky.*

ASBESTOSIS AND ITS PREVENTION. [illegible] *Arbeitsschutz, pp. 9-16 (1937).*

On behalf of the North German Textile Association, a survey was made of 11 asbestos factories employing 385 workers. An incomplete table (as the author points out) shows 22 cases of asbestosis and 13 deaths. After a short description of the clinical and x-ray picture there follows a careful description with many illustrations of the exhaust systems needed for certain processes and machines.—*L. Teleky.*

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

PROTECTION AGAINST TOXIC GASES IN INDUSTRY. *J. D. Pratt. Paper to the Institute of Chemistry (South-Eastern Counties Section) February, 19, 1936.*

[illegible] *19, no. 5]*

The paper deals m[illegible] of toxic gases in ind[illegible] mic that before u[illegible] closed vessels that [illegible] manufacture or [illegible] products, a respons[illegible] ist a test by lowerin[illegible] into the vessel. B[illegible] ways more sensitiv[illegible] ple of gases with[illegible] amage will be done[illegible] ms. The Home [illegible] dustry sponsored [illegible] been underta[illegible] Defence Research [illegible] bject of finding sim[illegible] ould be easily app[illegible] ost commonly en[illegible] Tests were require[illegible] gen; arseniuretted [illegible] xide, nitrous fu[illegible] compounds, such [illegible] chlorbenzene; ani[illegible] carbon monoxide; [illegible] nitrobenzene. Th[illegible] pletion, and the [illegible] published. For ex[illegible] determine the ma[illegible] tration into which [illegible] safely go. This r[illegible] period of exposure [illegible] that the maximum [illegible] 8 hours. No man [illegible] into a closed vess[illegible] taining a concent[illegible] in excess of the 6 [illegible] centration. It h[illegible] make the tests qu[illegible] tative, and where [illegible] change color on [illegible] been developed, [illegible] color change giv[illegible] of the concentra[illegible] paratus will be u[illegible] ume of air can b[illegible] paper, and the to[illegible] pared with as set [illegible] methods for det[illegible] examined and th[illegible] regarded as the [illegible] tative estimati[illegible] For men doing [illegible] permissible con[illegible] the estimation

, no further use was made of nt.—*L. Teleky.*

NGES IN AN AUTOMOBILE DRIVER. N THE QUESTION OF KOILONYCHIA. *Dermatol. Zt., vol. 75, pp. 1–6*

ient cleaned cars with petroleum, , benzene and benzine. In this leveloped a marked sensation of oss of feeling in the finger tips. s were edematous, blue colored, tic, cold, and the nails were soft like. The author considers the d mechanical pressure to be the . *Teleky.*

TAL RADIUM POISONING. I. RROW AND LYMPH NODE CHANGES ITS PRODUCED BY ORAL ADMIN- N OF RADIUM SULFATE. *M. Ros- d E. J. Grace. Am. J. Med. 191, pp. 607–618 (May, 1936).* GES IN THE TEETH OF RABBITS D BY ORAL ADMINISTRATION OF ULFATE. *M. Rosenthal. Ibid., pp. 495–501 (Apr. 1937).*

bits were given several drops of n of radium sulfate orally three for 90 consecutive days, at the ch period each rabbit had re- it 100 μmg. of radium sulfate. osis can be induced by radium sothorium. Hyperplasia of the tic elements takes place in the w, later a decreased maturation lasts. Fibrous tissue replaces plastic marrow. There is an ulation of stem cells in the s and subsequently depletion of the lymph tissue. Absolute a is seen in the circulating

e third month the lower incisors s showed brown mottling which d spread to the other teeth. ppeared and tenderness in the animal that lived longest the lower incisors were worn gum margin. Abrasion also the molars. Radium was present in the teeth. Other are described.—*Helen Lawson.*

CHRONIC ZINC INTOXICATION. *E. S. du Bray. J. A. M. A., vol. 108, pp. 383–385 (Jan. 30, 1937).*

This is a report of the case of a man who, it is contended, developed a certain degree of anemia together with gastric symptoms while exposed to the possibility of absorbing a weak solution of zinc chloride through the skin. Since such exposure is not followed by the absorption of zinc and since no laboratory data are presented which to any degree substantiate the claims of the author, the contention that the case in question is one of chronic zinc intoxication is quite unfounded.—*C. K. Drinker.*

INJURIES FROM LIGHT METALS. *Kötzing. Arbeitsschutz, pp. 77–78 (1937).*

The author who writes on the basis of reviewing over 16,700 cases of light metal injury, adds nothing to what has already been frequently described in the literature. He recommends purification of the flesh around the wound with benzine and binding with a "bacteria killing" salve or sterile material.—*L. Teleky.*

FATAL PHOSPHINE POISONING IN FUMIGATING FOR CORN WEEVIL. *O. Gessner. Samml. v. Vergift., vol. 8 (Gutachten), pp. 13–18 (1937).*

Bags with 30 gm. of 60–75% aluminium phosphide (trade name "Delicia Al P") were distributed throughout a granary. Phosphine in quite high concentrations was liberated by the humidity of the air and of the grain. In a house adjoining the warehouse 12 persons fell ill with nausea, of whom one died.—*L. Teleky.*

DUST HAZARDS AND THEIR EFFECTS

DISCUSSION OF INDUSTRIAL ACCIDENTS AND DISEASES. 1935 CONVENTION OF THE INTERNATIONAL ASSOCIATION OF INDUSTRIAL ACCIDENT BOARDS AND COMMISSIONS, ASHEVILLE, NORTH CAROLINA. *U. S. Dept. Labor, Div. Labor Standards, Bull. no. 4, 1936.*

Essentials in the Diagnosis of Silicosis and Tuberculosis

L. U. Gardner (pp. 63–70)

Gardner begins by defining pneumoconiosis as "a condition of the lungs resulting from the prolonged inhalation of dust." Only when serious changes producing symptoms occur can the condition be considered as a disease.

Gardner then gives an outline of the types of dust that have been studied, emphasizes the point of "adequate exposure," and the importance of a good occupational history. The value of the x-ray is briefly discussed. Following this Gardner, in his usual concise and brilliant manner reviews the pathology and the roentgenological features of silicosis and of asbestosis, the two known pneumoconioses producing disability. A discussion of symptoms and prevention follows.

Relationship of Asbestosis and Silicosis to Disability

R. R. Sayers (pp. 70–75)

Sayers after giving the standard definition of silicosis, as drawn up by the American Public Health Association, gives a personal definition of asbestosis. He then defines disability, including in the definition, "an increased susceptibility to respiratory infection." He shows that there is increased lost time due to respiratory infections among workers exposed to dusty, as compared to workers in a non-dusty atmosphere. In the granite industry it was found that the severity, when measured by the duration of cases lasting 8 days and longer, for the group of hand pneumatic cutters exposed to high concentrations of silica dust, was about four times as great as the same for a group exposed to the minimum amounts of industrial dust. He gives interesting tables of anthraco-silicosis and respiratory infection and mortality from respiratory tuberculosis.

In discussing the physical examination of suspected fibrotics, he suggests an exercise test such as is used in determining heart function. In considering the stages of silicosis he feels that there is no dis-

ability until the second stage, when the disability is usually not great. In the third stage the worker is usually seriously and permanently crippled.

Pneumoconiosis

R. Hunt (pp. 83–91)

Hunt explains "pneumoconiosis" as being a general term very broad in scope and referring to presence of any dust in the lungs. After reviewing the pathology of the pneumoconioses as a whole he gives the pathology peculiar to silicosis. He states that "silicon dioxide is soluble in the weak body alkalies, and such reaction produces a toxin or poison." The "poisonous chemical reagent so alters the immediate surrounding tissue that it loses its resistance and becomes incapable of protecting itself against the invasion of foreign bacteria."

In considering the exposure necessary to produce silicosis he says: "roughly speaking, it might be stated that an atmosphere containing from 5 to 25 million dust particles per cubic foot may produce silicosis in from 5 to 25 years."

He says that "it has been estimated that 25% of all dust workers will eventually contract the disease." The history, physical examination, x-ray examination and dust counts are briefly discussed.

After speaking of the progress of silicosis after exposure has ceased, he speaks of the amount of silica found in the lungs at autopsy. Incineration of the lungs may yield a silica content varying from 2 to 50% in relation to the total ash of the lungs. He quotes authorities as hesitating to call any amount less than 15% abnormal.—*W. I. Clark.*

These papers are followed by discussions of several aspects of pneumoconiosis; at the end of this section (p. 94) Dr. Lanza gives a careful and concise "Discussion Memorandum."

The remainder of the 200-page bulletin is taken up with several papers on accident prevention and workmen's compensation. Dr. Kesser gives an excellent paper on "The Curative Workshop and the Determination of Capacity to Work" (pp. 142–150).—*Helen Lawson.*

THE HEALTH OF CEMENT WORKE
Curtis. Pit and Quarr
1937).

After giving a short rev
investigations, the auth
results of several years'
plants of the Portland Cemen
The study included three
of illness: (1) respiratory,
and digestive, and (3) mis
cluding rheumatism, arthritis
nary, eyes and dermatitis.

Owing to the very low conte
silica (average about 2%), in
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pated, and physical examinat
x-rays made by Dr. Gardner
showed little evidence of it.
rate from tuberculosis amo
workers was found to be
that for all males in the U. S.
Area.

Minor respiratory diseases
fluenza, rhinitis, cloudy nasal
and pneumonia were comm
be associated with dust ex
matism and arthritis are the
miscellaneous illnesses. Brief
made to eye troubles, dermat
neuritis, neuralgia, muscle cra
trouble, cancer, etc.—*A. D. Lu*

CONDITIONS OF WORK AND
AMONG WORKERS AT A THOM
A. G. Kalashian. Gigiena
bezopasnosti, no. 6, pp. 33-

The author describes cond
Thomas slag mill of the metallur
at Kerch. He found dust conc
very high—72.6–106.6 mg./L. a
ers; 78.2–83.7 mg./L. at the ele
37.8–42.1 mg./L. in the depart
spiratory diseases among the
are much more frequent than a
workers at the plant. He poin
the dustiness must be controlled
gests that workers should be
selected, and supervised medi
transferred at times to other
work.—*From the author's Engli*

MANGANESE PNEUMONIA. *E. W.*
Aerztlich. Sachverständ. Zt., *vol.*
75–81 (1937).

[vol. 19, no. 6]

A man engaged in
making dry cells di
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great amount of bibli
Russian, Brazilian
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DISCUSSION, *vol. 1*
(Dec. 4, 1936).

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ten with pure lead oxide. Other cases were caused by smoking opium pipes which had pewter mouth pieces and bowl supports. Another group had warmed their wine in pewter jugs. Because of the widespread use in China of low quality pewter, the authors suspect that there is a good deal of lead poisoning which remains undiagnosed. Another of their patients was exposed to lead in a lead foundry.—*Helen Lawson.*

DUST HAZARDS AND THEIR EFFECTS

NEW DOCUMENTS ON SILICOSIS. *E. Martin. Méd. du. Travail, vol. 9, pp. 149-156 (July, 1937).*

INQUIRY REGARDING SILICOSIS IN BELGIUM ORGANIZED BY THE INDUSTRIAL MEDICAL SERVICE. *Pp. 157-164.*

SILICOSIS: NEW RADIOGRAPHS AND COMMENTS. *M. Conrozier and J. Magnin. Pp. 165-209.*

The whole July issue of *La Médecine du Travail* is devoted to silicosis. The French and Belgian experts and industrial inspectors have been compelled for years to wage a stiff fight against a small but powerful group of mining companies and their physicians to get recognized the existence or the specificity of silicosis or at least its appearance in certain mining districts. This has delayed the adoption of protective measures and compensation of silicosis as an industrial disease. Martin in an introductory article tells the story of the long fight and presents an order of the French Under-Secretary of State for Mines who orders the industrial inspectors to require technical precautions against the dust hazard and medical examination of workers on the part of the owners.

The second paper gives a short review of the results of the Belgian inquiry, the investigation made of many different dusty industries and the more or less great number of cases of pneumoconiosis seen in each.

Conrozier and Magnin present 23 x-rays and short case histories on the basis of which they state the view, held by all experts, that silicosis is a separate disease from tuberculosis and has a different picture. It is found only where quartz containing dust is inhaled. If the confidential physicians of the mining owners do not see this, it is only because "they have evidently not looked for it or because they are unable to recognize it."—*L. Teleky.*

ANIMAL EXPERIMENTS ON PATHOLOGIC TISSUE CHANGES FOLLOWING INTRAVENOUS INJECTIONS OF VARIOUS DUSTS. *K. H. Stoeckel. Arch. f. Hyg., vol. 118, pp. 111-125 (1937).*

The author injected various dusts intravenously in rabbits at intervals of from a few weeks up to several months. The materials included steel-shot, quartz, lime, Thomas slag, argillite stone, chamotte, quartzite and argillite dusts. The tissue changes observed in different organs are described and illustrated. Different dusts, however, never showed a really characteristic or specific action. Unfortunately data are lacking on the chemical constituents of the steel-shot dust, argillite stone dust and argillite dust; only some on particle size are given from which it is seen that various dusts divide into different degrees of fineness, a condition which, according to the author, explains many of the differences in his results.—*L. Teleky.*

DUST AND TUBERCULOSIS. EXPERIMENTAL WORK ON THE EFFECT OF SERICITE AND GRINDING WHEEL DUST ON TUBERCULAR GUINEA PIGS. *H. Selter and P. Weiland. Arch. f. Gewerbepathol. u. Gewerbehyg., vol. 8, pp. 71-82 (1937).*

Interested by Jones's assertions (already almost disposed of), the authors have investigated the effect on tuberculous infection in animals that inhaled sericite and others exposed to "steel polishing dust" (steel and emery dust). Some animals were infected with weakly virulent bacilli, others with virulent ones; other animals were previously immunized with a weakly virulent bacilli and still others were super-infected with virulent material. They found that sericite is quite as damaging as the grinding dust.

In order to prevent misunderstanding, the abstractor adds that these experiments supply no contribution to the question of sericitosis-silicatosis since in these animals no decided pneumoconiosis developed, still less silicosis. And on the other hand, the sericite effect is not compared to that of

quartz, which is what the steel grinders in mining countries and in Sheffield breath. Here the sericite is compared only with the steel and emery dust that causes no silicosis or severe pneumoconiosis in men.—*L. Teleky.*

PATHOLOGICAL AND ANATOMICAL QUESTIONS IN CASES OF PNEUMOCONIOSIS: SEVERE ASBESTOSIS. *W. di Biasi. Med. Welt, vol. 11, pp. 1234-1237 (1937).*

The author gives a clear discussion of pathological and anatomical changes in the development of a case of pneumoconiosis (silicosis) and emphasizes the fact that up to now real pneumoconiotic changes—nodule and scar tissue formation—have been seen only after exposure to quartz. In many hundreds of cases (the author lives in the Ruhr coal district) he has never found connective tissue growth in lungs completely blackened by coal dust unless rock dust was also present. Of the cases where coal dust or soot is said to have caused scar tissue formation (Lochtkemper, Hollmann), no autopsy findings have hitherto been shown. Sometimes in judging the degree of silicosis, one finds a macroscopic examination at autopsy is inadequate—only a microscopic examination of the lungs will suffice.

In asbestosis, the growth of diffuse connective tissue is greater in the lower part of the lung than in the upper. According to the author, this growth is caused by the mechanical action of asbestos.—*L. Teleky.*

ZINC FEVER. PROPHYLACTIC RESULTS. *H. S. Natvig. Tidsskr. f. d. Norske Laegeforening, vol. 57, pp. 456-465 (1937).*

Natvig, who is attached to the University Hygiene Institute in Oslo, gives an account of his observations during the past 2½ years of zinc fever among workers employed in brass foundries, bicycle factories and other workshops in which zinc is fused with copper. He had no difficulty in finding more than 100 workers with a clinical history of typical zinc fever, which in 36 cases had recurred once a week or oftener for a long period. After giving an account of the clinical picture and noting that it is quite transitory and comparatively harmless, however disagreeable, he discusses its genesis. In his opinion it is due to the deposit of particles of zinc oxide in the lungs. These particles are so small that they penetrate to the alveoli of the lungs on inhalation. The boiling point of zinc being 930°, and the melting point of copper being 1180°, it follows that when the two are melted together the zinc gives off large quantities of zinc vapor, which is at once converted into zinc oxide on coming into contact with the oxygen in the air.

The prophylactic measures hitherto taken in the industries involved have proved disappointing; no system of ventilation with fans, and no mask has afforded adequate protection. Natvig is convinced that the only rational and effective prophylactic measure is to prevent the zinc vapor from escaping into the air inspired by the workers. The problem is complicated by the fact that the apparatus generating zinc vapor may have to be in operation in many different parts of a workshop; a fixed system of artificial ventilation would, therefore, not serve its purpose. Accordingly, a movable device was contrived, with a funnel-shaped opening and a forced draught for the aspiration of zinc vapor at the point of its generation. In a factory provided with this device were 15 workers who, without exception, had some time or another suffered from zinc fever. There has been no case of zinc fever since this contrivance was installed about 2 years ago, except on one occasion when it was not used.—*Bull. Hyg.*

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

DUST CONTENT OF AN ASBESTOSIS LUNG AND THE NATURE OF ASBESTOSIS BODIES. *N. Sundius and A. Bygden. Arch. f. Gewerbepathol. u. Gewerbehyg., vol. 8, pp. 26-70 (1937).*

Very careful chemical experiments on the asbestos in the lungs and asbestosis bodies. Only the following points can be mentioned here: although in industry much more serpentine asbestosis is used than

hornblende, the latter was found exclusively in the lungs examined. The asbestosis body sheaths are formed not only around asbestos but about rutile needles and also around a silica-free oxide with a similar type of crystal. No support has been made available to show that asbestos causes a chemical decomposition in the lung. The gel substance of the sheath consists of iron oxide (average 40%) phosphoric acid and an organic nitrogen and sulfur-containing substance (31%) which must be considered as a protein (Beger). This gel substance originates entirely from the body fluids and since the iron content is high, the most plausible suggestion is the blood. The cause of the sheath formation is not related to the chemical nature but to the rigid and sharp needle form. The authors also believe that the fibrosis can be blamed to this.—*L. Teleky.*

ULTRAMICROSCOPIC SILICA IN LUNG TISSUE. *O. M. Faber. Staub, no. 3, pp. 372-383, (Oct., 1936).*

In a previous article the author showed the possibility of determining the presence of silica in portions of hardened lung by means of x-ray spectroscopy, and of forming some idea of the quantity by the degree of blackening caused by the deflected rays. He now points out some of the sources of error which may occur in this very delicate work. The formalin used for hardening the portion of lung will by itself cause a certain amount of deflection, which has to be allowed for and portions of lung treated by aqua fortis or incineration give variable results.

The x-ray spectrum from pieces of lung showed that the silica was present in such exceedingly minute particles (from 100 to 10 mμ) as to suggest that it was in colloid form. The theory is put forward that silica becomes converted in the body into the colloid form and that it is in that form the most injurious.

It is now felt that it is necessary to know if silica is actually inhaled in the colloid form in any considerable quantity, or if the colloid silica found in the lung tissue is the result of a breaking down and dissolving of large particles. Further investigations are to be made on this point.—*Bull. Hyg.*

THE SILICOSIS PROBLEM AND THE NECESSITY OF RELIABLE METHODS OF DUST MEASUREMENT. *G. Hass. Zentrlbl. f. Gewerbehyg., vol. 24, pp. 175-177 (1937).*

The author lists the dust problems still to be solved (some of which are solved already.—*L.T.*) and notes the need for exact determination of dust concentrations and chemical constitution. Concerning the former he mentions the konimeter, Owens jet counter and the tyndall-meter. For determining the chemical character of the dust, he suggests the use of x-rays. Further working out of the methods are necessary.—*L. Teleky.*

IMPROVED METHOD FOR DETERMINING NASAL FILTRATION OF DUST. *G. Lehmann. Arbeitsphysiol., vol. 9, pp. 569-571 (1937).*

The former method was bothersome because of the excessive time used for counting the konimeter samples. The author now gives a photometric method that depends on the measurement of light reflected by the dust. For this he employs the Zeiss Pulfrich photometer and a special stage. The apparatus must be calibrated for each kind of dust.—*L. Teleky.*

THE SIGNIFICANCE OF NASAL DUST FILTRATION IN ASBESTOSIS. *G. Lehmann. Arbeitsphysiol., vol. 9, pp. 572-578 (1937).*

The author examined 73 workers in an asbestos factory. Since most of them had not worked long, he found only one medium to severe case, 17 light ones, and 24 incipient ones. After determining their nasal filtering efficiencies, he concludes that, as in silicosis, this efficiency is significant in asbestosis. [There are, however, quite a few exceptions to this relationship.]—*L. Teleky.*

SOME NEW CHARACTERISTIC PROPERTIES OF CERTAIN INDUSTRIAL DUSTS. *H. V. A. Briscoe, J. W. Matthews, P. F. Holt and P. M. Sanderson. Bull. Inst. Min. & Metall., No. 393 (June, 1937).*

This paper describes a continuation of the work referred to by these authors in an earlier note (Bull. Inst. Min. & Metall. No. 391, Apr., 1937, these Abstracts, vol. 19, p. 176). The effect of varying conditions—time, temperature, particle size, and the

ratio of dust to solute—on the apparent solubility of quartz dust have been investigated. It appears that a long period—100 days or more—would be required to attain a constant concentration of silica in solution at room temperature. At 80°C. the solvent action of water on quartz dust has about as much effect in 5 hours as that attained in a month at 20°C. Particle size has a marked effect. Solubility increases markedly with the proportion of dust subjected to the action of the solvent; this was found to be the case also with asbestos dust.

The solubilities of some dusts were examined. Asbestos on extraction with water may yield soluble silica greatly in excess of that given by quartz, and normally gives up alkali also, approximately in proportion to the silica extracted. Calcined flint dust increases in solubility with decreasing particle size; it yields alkali the amount of which may be taken as a measure of the lime dissolved from the dust. The solubilities of the comparatively harmless cement and sillimanite dusts are of a lower order than those of flint and asbestos. The solubility of calcined flint is much lower than that of raw flint and is similar to that of quartz. When asbestos or quartz dust is mixed with an equal weight of lime or cement its solubility is greatly decreased. Calcium borate also depresses the solubility of asbestos considerably, calcium lactate and citrate do so to a small extent, but calcium carbonate has no effect. The presence of added sugar charcoal reduces the yield of soluble silica from quartz. Magnesia has an action similar to that of lime. Magnesium carbonate, alumina and zinc oxide have no apparent effect. Cryolite diminishes the yield of soluble silica from flint and asbestos, but increases the yield from kaolin, felspar, cement and quartz.—*T. Bedford.*

THE SAMPLING OF INDUSTRIAL DUSTS BY MEANS OF THE "LABYRINTH." *H. V. A. Briscoe, J. W. Matthews, P. F. Holt and P. M. Sanderson. Bull. Inst. Min. & Metall., No. 393 (June, 1937).*

In recent papers these authors have described the salicylic acid filter for sampling dusts. The filter has been used under various conditions and its design is now settled. A scale drawing is given. The filter will pass 40 to 50 cu. m. of air in about 3 hrs. There are two practical difficulties in its use. Firstly, in factories it is often difficult or impossible to arrange for the necessary suction. Secondly, the recent experiments by the authors (Bull. Inst. Min. & Metall. No. 391, Apr., 1937, these Abstracts, vol. 19, p. 176) have demonstrated the great reactivity and solubility of dust solids, so that even the solution of the salicylic acid in alcohol may bring about considerable changes in the collected dust. In addition, the study of solubilities requires a fairly large sample of dust (at least 1-5 gm.). Hence some purely mechanical method of dust collection was indicated.

To this end the labyrinth was designed. The baffles are a series of polished copper plates spaced and located by lengths of copper tube, and held together in a single assembly by long brass bolts and nuts. The assembly is contained in a casing, which is rectangular in cross-section in one form of the instrument, while another form is cylindrical. The labyrinth is 36 in. long, containing 32 baffles spaced 1 in. apart. It is connected to a suction duct and the dust is collected by impingement on the baffles. The bulk of the duct collects in the first 3 or 4 sections.

In one test, made in a flint-grinding plant, the labyrinth was run for 25 days, say about 500 working hours, and the total air flow was about half a million cubic meters. On dismantling the instrument was found to have collected 100 gm. of dust. Microscopic examination of the dust from the several sections showed a distinct grading according to particle size, the larger particles being deposited near to the inlet end. The form of the mass-distribution curve suggests that the mass of dust escaping collection cannot be much more than 5%. Similar efficiencies were found in cement and sillimanite works, but with asbestos dust the efficiency was much less.

For the measurement of the particle size distribution of the dust in various sections, specimens were mounted on slides and the image of the dust was projected at a magnification of 500 diameters on a white screen ruled in squares with a side of 1 cm. = 20μ. A slight change of focus enabled aggregates