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**The PNEUMONOKONIOSES
(SILICOSIS)**

**Literature and Laws
of
1934**

**International Abstracts, Extracts and Reviews of
the Pneumonokonioses and Their Associated
Diseases and Subjects**

By

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SEP 23 1935

To
E. R. Le Count, M. D.
in Recognition
of His
Eminence as a Pathologist
and
Influence as a Teacher

cinoma which had metastasized to the right lung and also to both kidneys.

7. Amendola, O.

Pulmonary Asbestosis.

Morgagni. 76: 16-18, Jan. 7, 1934.

This review abstracts P. Ellman's paper read recently before the London Tuberculosis Society. Ellman brought out clearly the clinical, radiological, and anatomic-pathological characters of pulmonary asbestosis, as well as the dangers attending a tuberculous complication. Pulmonary asbestosis should now be considered a clearly distinct clinical entity. The diffuse nature of the resulting sclerosis much more seriously impedes the respiratory function than in equally advanced cases of silicosis; asbestosis is thus a more serious disease. Initial symptoms and their importance depend on the kind of asbestos work and the quantity of the dust inhaled. Some symptoms may be wholly lacking, in spite of clinical and pathological signs. Ellman thinks dyspnea the first symptom, setting in sometimes with such force and advancing so fast it becomes serious and persistent, being transformed eventually into a true orthopnea. A coarse dry cough and chest pain end the first period or accompany it. Expectoration frequently lacking; if present, is scanty, dense, mucous; worse in winter, often purulent; occasionally bloody discoloration present. Sometimes there are night sweats and dizziness. Anorexia can be stabilized in the beginning but is worse in advanced cases; the same with the cyanosis. Profound emaciation is a very distinctive sign. Cutaneous lesions sometimes occur on the hands and legs. Thorax is much restricted in expansibility, especially low down, where the sclerotic process generally originates. The apices are more retracted. Auscultation reveals lessened vesicular murmur and eventually fine rales, dry and crackling are present. Radiological examination shows the signs of the diffuse sclerotic process, originating and

prevailing chiefly in the lung base. Macroscopically the pleura and lungs show hardening, with pleural cavity partially or completely wiped out by the adhesions. Microscopically, there is chronic interstitial lung sclerosis. Asbestos particles occur in the alveoli or in separate masses in the sclerotic tissue. Interstitial tissue may be increased; in some cases the alveolar structure has completely disappeared; in other pulmonary zones there is compensatory emphysema. In all external and internal sclerotic zones are found the asbestos bodies, tending toward radial grouping. In the connective cells are found the relapsing giant cells, first described by S. R. Gloyne. Such cells are larger than the typical Langhans; their protoplasm is granular and colored quite differently from the structureless tuberculous giant cells. In all asbestosis autopsies are found the characteristic asbestos bodies, yellow as gold, found also in the sputa of asbestos workers. Their isolation as well as groupings in the sputa show the disintegrative process in the lung tissue, whether it be the simple bronchial pulmonitis, or a secondary tuberculous infection. Yet the mere presence of such bodies in the sputa does not justify the diagnosis of pulmonary asbestosis, even if this individual has been exposed to asbestos dust. Clinical signs of lung sclerosis and radiological examination have to confirm this diagnosis fully. Asbestos bodies can be encountered a long time in the sputa especially if the possibility of inhaling asbestos dust is decidedly short. In one case the person was exposed to asbestos dust for ten weeks and five years after asbestos bodies were found in the sputa. One inclines to think from this case that once in the lung these bodies continue more or less permanently in its tissue.

The disease progresses, but slowly; its gravity depends on the quantity of asbestos inhaled. A long period generally occurs between the inhalations occurring and the first symptoms. From numerous and complete observations of author

ities, it seems doubtful just now whether asbestosis can be decided to be a tuberculous process. Further research is needed to clear up the relations between the changes characteristic of asbestosis and the inception of a tuberculous process.

8. Amendola, O.

Silico-Tuberculosis in Gord Miners.

Morgagni. 76: 406, April 1, 1934.

This brief abstract of Magnin's paper read Jan. 13, 1934, before the Paris Society for Scientific Study of Tuberculosis, defines silicosis as lung fibrosis due to prolonged inhalation of silicious dust. Authoritative conclusions follow. The diagnosis of silicosis is not impossible or even difficult, given radiological and etiological data. Inhalation of dust from Gold rocks (which contain 90 per cent free silica) is as injurious as the dusts from gold-bearing quartz. Five years, even three, are enough to give a miner the disease. Inhalation of silica dust alone may cause lung fibrosis in an already tuberculous subject. Likewise pulmonary fibrosis of silicious origin may awaken in a worker a tuberculous infection previously contracted.

9. American Public Health Association.

Standard Practices in the Compensation of Occupational Diseases.

Yearbook, Am. Pub. Health Assn. 1934-1935; pp. 102-107.

Changes in the laws of certain states in United States are noted, as in Kentucky, New York, and Wisconsin, with reference to occupational disease compensation. In the United States as a whole there is a trend toward blanket coverage, for free choice of physician by employees, and the setting-up of medical control boards. Both the first two points are still controversial. As for the third no medical control board has yet been set up unless in Illinois, where the Industrial Commission has created a medical depart

ment with a Director and a list of consultants in the various branches of medicine and surgery. Mention is made of various co-operative boards, commissions, in the several states, whose work and powers obviate, at least temporarily, enactments at law, difficult to secure for a variety of local reasons. Marked emphasis on medical control has occurred and aroused interest in medical practice revision, due to undesirable or unforeseen developments in actions brought either under the common law or compensation laws.

A section records, Changes in Foreign Laws. 32 countries have now ratified the 1925 convention on occupational disease compensation. 6 countries newly establishing occupational disease laws, are: Australia, Ceylon, China, Cuba, Roumania and Spain. 4 countries have extensively revised their occupational disease laws. The recent affiliation of the United States with the International Labor Organization is called "an event of great significance." The United States thus aligns itself with 58 other nations for the improvement of world-wide conditions, and promotion of progressive standards of social hygiene and legislation.

10. Amor, A. J. and Evans, R. G. P.

Some Observations on the Diagnosis of Silicosis
Practitioner. 132: 700-708, June 1934.

The diagnosis of silicosis can only be arrived at with any degree of certainty by considering exhaustively the history, physical signs, radiological, pathological and chemical examinations. Physical signs should be interpreted in terms of pathology so that they will convey some idea of the morbid changes present in the lungs. It appears that the silica particle, as such, is not solely responsible for the causative lesion, and recent work by Jones brings forward evidence that a hydrate of potassium aluminum silicate or "sericite" is the causative factor of the pulmonary lesions of these workers. However, the evidence

as it stands at present is not convincing. The lesion appearing in the South Wales coal-miners is unique in many respects.

The authors have endeavored in this investigation to express some clinical, radiological and pathological views on the diagnosis of silicosis as it occurs in the South Wales coal-miners, with some notes on the differential diagnosis of the super-imposed tuberculous infiltration, frequently found in these workers.

(1) Brief details of the macroscopical and microscopical appearances of the condition are described, together with a series of silica determinations made by the colorimetric method noted in the text.

(2) Clinical and radiological examinations have been described in each of the three recognizable stages, and it has been shown that the correlation of these factors with the industrial history is essential for a correct diagnosis.

11. **Angerstein, T. C.**

Should Illinois have Legislation such as to Place Occupational Diseases under Compensation? Talk made before the 12th Annual Safety Conference Chicago, May 11, 1934, at the Dust in Industry Session.

In a 20 pp. reprint this Attorney advocates legal compensation for all occupational diseases, with provision for a proper and adequate medical department or medical board of review. The present "epidemic" of law suits involving \$50,000 and more for occupational diseases, with the confusion, racketeering and other attendant undesirable circumstances, are reviewed as a preliminary to the much greater justice and definiteness obtaining from such a law as might be enacted. The several statutory provisions under which a large number of law suits have been brought the last few years, together with the recorded decisions of courts appertaining thereto, are presented in condensed and usable form.

12. Anthracite Dust.

Report of the Industrial Pulmonary Disease Committee of the Medical Research Council.

Brit. M. J. 1: 198-199, Feb. 3, 1934.

In 1931 this inquiry was begun, on request of the Mines Department and the Home Office, as to whether the working health and capacity of coal miners had been impaired by inhalation of anthracite dust. The Committee found no evidence that such inhalation of anthracite or other dust had caused fibrosis of the lungs. The circumstances under which the inquiry was conducted, the conditions of miners working underground as well as above ground are carefully set forth. A very detailed tabulation of 51 patients studied is given with comments and interpretations.

13. Anti-Silicosis Equipment.

Rock Prod. 37: 69, May 1934.

A line of new respirators for protecting workers against inhaling silica, lead and similar dusts, has been announced by the Pulmosan Safety Equipment Corporation of Brooklyn, New York. Dust control engineers cooperated in developing features of these new appliances, such as: increased filter size, direct breathing intake, coarser wire screens to permit easier breathing. Screens are bound with aluminum and a gas mask type of exhaust valve is used. A new refinement on old patterns is an all-rubber head band adjustable to permit longer wear. The appliance does not obstruct the worker's vision.

14. Asbestos.

Indust. Bull. 13: 95, April 1934.

What is asbestos? It is a unique substance of rocky formation which resists heat, water and wear. It can be spun, woven or molded into any necessary form. There are several varieties containing various proportions of silicate of magnesia. Breathing the dust of this substance is bad for the health just as the inhaling of almost

any other foreign substance is deleterious. Workers who have to handle asbestos fiber or material should avoid, as far as possible, breathing the dust, and employers should take care to have suitable ventilation in places where asbestos dust is generated.

The disease resulting from the inhaling of asbestos dust will probably be declared similar to that resulting from silica. Manufacturers or handlers of asbestos products might then begin here and now to prevent trouble because the disease, like silicosis, can be prevented—there is no cure.

15. Asbestos Dusts.

Safety Eng. 68: 74, Aug. 1934.

It is established that asbestosis may cause disability and death but it is much less understood than silicosis, and the pathology of each is different. In asbestosis the damage is to the bronchioles rather than to the lung alveoli. The symptoms are similar to those of silicosis. Our present knowledge of asbestosis leaves much to be desired. Studies are now going on, the results of which should be of prime importance.

16. Barber, D. N.

Silicosis.

West Virginia M. J. 30: 310-313, July 1934.

Silicosis or "miner's phthisis" has been known to occur among miners in certain districts for centuries, especially among metal miners in New Zealand, Australia, South Africa, Great Britain and in many of the states of our own country. It is a form of pneumoconiosis caused by the inhalation of large amounts of extremely fine silica (SiO_2) or silicate dust. It is usually slow in development, the rate depending upon the character of the dust (silicates being not as active as free silica), the amount inhaled, length of exposure and physical condition of the man. The period of exposure before development of the disease in the gold miners of South Africa is from

2½ to 7½ years. It is characterized anatomically, by generalized fibrotic changes and the development of miliary nodulation in both lungs; clinically, by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis, and by characteristic x-ray findings. This process may progress for years after exposure has ceased. In the following industries it constitutes a definite occupational hazard: the mining of metals, coal, asbestos, etc., grinding and abrasive industries, glass-making, excavating, quarrying, stone cutting, sand pulverizing, sand blasting, furrace brick manufacture, making of vitreous enamel, slate manufacture, cement and pottery industries.

It is becoming an increasing cause of industrial court actions and compensation adjustments. At present the states have varying laws regarding silicosis and usually inadequate means for diagnosing it. Competent federal control would seem a better system, but if each state handed over all cases irrevocably to a properly constituted medical board the present chaos would be lessened.

Race and habits have no significance. Most cases develop between 20 and 30 years, but the length and type of exposure is more important than age. ✓ Silica may do harm mechanically, owing to its needle-like particles, but due to its partial solubility, it probably injures the lung tissue by its poisonous action.

Subjective symptoms are—dyspnea, pain in chest of a fleeting and vague nature and not disabling, cough, which is usually not productive, occasional slight hemoptysis, night sweats, loss of strength, and gastro-intestinal symptoms suggestive of indigestion.

Change of occupation to an open air work is necessary. Exercise is usually not restricted in uncomplicated cases. A high dry, warm climate is best.

It should be emphasized that while the cardinal

superior to any number of dust separators of the cyclone type, no matter with how many gadgets added. Alfalfa meal dust proving a formidable difficulty to one manufacturer, he applied to the Artisan for help, being rewarded by the five very informative, clear drawings of Engineer Baybutt's own cloth separating device together with his no less intelligible and simple accompanying description. A heavy cotton flannel bag was suggested for clearing out the clouds of alfalfa dust, with another bag inside it, if one bag proved insufficient.

19. **Beger, P. J.**

Asbestosis: Particle-Construction.

Deutsche Med. Wchnschr. 60: 231, Feb. 9, 1934.

Silicosis in all its forms is very like asbestosis. The typical difference appears to lie in the out-crop of asbestos particles. They are constructed as follows: asbestos dust is inhaled, is dissolved in the body humors; it is readily decomposed without its needles losing their form. Carbon-dioxide in the lungs frees the asbestos crystalline lattice work of its magnesium and iron ions. Thus the negative valence of the silicic acid supports in the lattice-work, is set free. Absorption of albumen from the body humors takes place. Röntgen pictures reveal the construction of the particles, step by step, through covering the needles with albumen. Since the albumen molecule, now too much enlarged for the lattice-work, cannot replace the mg. atom, it has to place itself on a needle where electric tension exists. That leads to dissociation of the silicic acid framework of the needle and so to the building up of the particle. The original compactly filled particles become wrinkled. Ferric oxide grains are distinguishable; in the final stage of construction rows of these grains remain in which the form of the asbestos particles can hardly be said to continue. Also the asbestos needle itself has disappeared. The albumen ions of the covering can work with a valence of 20

or more on the silicic acid tetrahedrons. Consequently the tetrahedrons will be torn out of the silicic chain and scattered through the albuminous covering. After saturation of the free valence, electrostatic equilibrium is accomplished; no further reason exists for the agglomerated mass. The albumen is carried off, loaded with its added and probably abundant silicic acid. The ferric oxide remains behind (as a mechanical impurity of the albumen).

20. Beger, P. J.

The Destructive Factor in Asbestosis and in Silicosis.

Med. Klin. 30: 1222-1227, Sept. 14, 1934: 1258-1261, Sept. 21, 1934.

This detailed discussion is accompanied by 4 Röntgen pictures of lung sections and 2 diagrams of the tetrahedrons of silicic acid and the structure of chrysotile, involved in the basic destructiveness of these two lung-affections, together with illustrative chemical formulae and reference to 21 authorities.

(1) Asbestosis in both theory and practice shows that the silicic acid framework of the asbestos needles goes into solution. The damage to the lungs is due to this solution of the silicic acid. The degree of severity of the fibrosis increases with the mass of the silicic acid dissolved, in connection with the time involved in this solution process. According to authorities on occupations the disease will develop further the longer the silicate is deposited in the lung.

(2) In consequence of the co-operation of their covering power and their valence power, the asbestos particles help to dissolve the silicious framework of the asbestos needles. Solution of the silicic acid as a purely surface-limited process of slow course, results from delay or omissions in the structure of the particles. Hence asbestosis comes into existence after silicosis.

(3) Dissolved silicic acid is the destructive factor in silicosis as in its analogue, asbestosis.

Kinds of quartz as well as total silicates, and the exclusively silicate artificial products, like glass, porcelain and so on, serve as sources for silicic acid. The degree of injury depends on the silicic acid content and its solubility. Solution depends on the surface limits involved. It is generally slower to appear than in asbestosis. The disease extends over a longer period of time while for degree of severity, irresistibility or other circumstances it is the same as asbestosis.

(4) Personal factors are as much concerned in the development of both diseases as the actual predisposing conditions derived from clouds of dust and work carried on therein. The clarification of details in both diseases must be left to further experimental research.

21. **Beger, P. J.**

Further Discussion of the Particles in Asbestosis. *Virchows Arch. f. Path. Anat.* 293: 530-539, Oct. 1934.

Discussion of controverted points from current views of several writers on the subject, together with experimental results secured by the author, form the body of this article. The following conclusions appear:

(1) By tests of opposing statements, it has been ascertained that all particles recognizable in asbestosis, have like properties.

(2) Mechanical irritation of the lungs from breathing asbestos dust does not appear to be serious if the symptoms break out after a mildly stimulating action or after the dust has been removed.

(3) The injury in asbestosis, as in the ordinary silicosis, is due to silicic acid solution.

(4) The asbestos particles speed up the solution of the silicic acid framework of the asbestos fibres. After this the asbestosis advances more quickly and severely than the ordinary silicosis.

(5) In cases where asbestos particles are not constructed or are delayed, the solution of as-

bestos dust at the limiting surfaces is enough to release disease symptoms. The asbestosis then goes on like a normal silicosis.

22. B  nassy, M. P.

Tuberculous Infection and Silicosis.

Bull. M  d. Paris, 4: 588-593, Sept. 29, 1934.

Current views and authorities on most questions involved in this title, are clearly discussed by this writer. The rare silicotic nodule characteristic of pure silicosis is compared with the common "mixed" silico-tubercular nodule, in respect of anatomic-pathologic characters, pathogeny, the development of their lesions, etiology and clinical signs.

In conclusion, the views of Louis Irwine, developed in 1929, are sustained in part, as follows: miners' phthisis or silicosis is produced by dust and by tuberculosis. Simple silicosis is a visible fibrosis not accompanied by tuberculosis clinically discernible, but in a majority of the cases of silicosis apparently simple, there is a precocious association of latent tuberculous infection. Certain unmodified infections occur in a silicotic lung; others, like tuberculosis, take on a silico-tubercular aspect. The naturally progressive character of simple silicosis may be attributed to an infectious factor, more particularly tuberculous.

These conclusions are categorically opposed when Irwine adds: "Initial modifications are due to dust and although by itself dust can cause lung fibrosis, it is chiefly important in predisposing lungs to tuberculous infection."

On the contrary, the French school, headed by Rist and Policard, think that in order to develop the initial silicotic lesion, the lung must be already tuberculous (more or less apparently so); or, at least, that dust inhalation and the infection are simultaneous (Policard). This opposition of view is by no means in vain, since medico-legal consequences of marked importance are involved. Rist, Mazel and Doubrow have very lately in-

sisted on the dangers presented by adoption of the proposed International Labor Bureau Convention, which in several respects is incompatible with French legislation. The convention announces mere hypotheses as if established facts: *loc. cit.* in respect of Articles 1 and 3; and the convention considers it impractical to cite industries or operations bearing silicosis risk. The example of Germany shows tuberculosis as rapidly becoming an official disease of the most onerous character, only 26 out of a 100 cases being verifiable. Social demoralization can only ensue when 75 per cent of cases filed are lost. In South Africa where both silicosis and tuberculosis are indemnified, 5,000 are pensioners. 40 items of Bibliography close this discussion.

23. Berger, W. (Münster).

Experimental Injury to Nasal Mucosa from Dust. *Ztschr. f. Laryng., Rhin., Otol. Theil I: Folio otolaryng.* 25: 60-66, March 1934.

This original and experimental contribution to respiratory dangers from dust first reviews the recent work of Lehmann on the interception of dust particles by the human nose. He investigated the disease relations between the different quantities of dust lasting out to reach the lungs and the disposition to silicosis. Great individual differences were ascertained, many noses retained 80 per cent of inhaled dust; others, only 5-20 per cent. In the same person investigated he found a value-constant independent of the dust concentration or lessened stream of air. He also found an astonishing agreement between lung-dust, and bad dust-interception in the nose. Old miners without dust in their lungs showed a nasal dust-laying power on an average of 50 per cent; those with "dusty-lungs" averaged only 22 per cent dust-laying power. Lehmann concluded that the nose is the dust-intercepting factor, irrespective of dust activity in the lung. Dust interception over 40 per cent protects against silicosis;

under 30 leads sooner or later, with positive certainty to lung disease from dust.

Before the Institute for Medicine and Natural Science, Münster, carried the matter further: whether the outcome of dust interception tests could depend on the occupation itself—the rhinologists asked: how the nose itself reacted to the dust? To what extent was the mucosa injured? Is there an individual predisposing factor here? Also could one resist dust successfully for 20 years, then suddenly succumb in a few years? How long does an average dust interceptor stay sound? To answer these queries animal experimentation was needed, since exact pathologic-anatomic grounds as to the influence of dust on the human nose was relatively lacking, also the effect of air heavily laden with dust on the nasal mucosa was not scientifically established.

In the Münster Industrial Hygienic Laboratory research on rabbits for dust injuries, also industrial gas injury to the whole organism, have been going on for years. A series of blood serum experiments in chronic CO poisoning has been carried out. The Laboratory Director, Dr. Jötten, induced the writer to investigate the upper respiratory air-ways for dust injuries.

Up to this time Dr. Berger has studied some 20 animals for pathological changes in nasal mucosa, as well as pharyngeal and laryngeal. Some animals had inhaled cement dust, others porcelain dust; still others, powdered white lead; some inhaled oat-husk dust, and a number CO (carbon monoxide) gas. The inhalation had lasted several months for some animals; others had inhaled for a year and more. Daily inhalation averaged 1/10 grams in 200 liters per hour of streaming air; the CO content of 200 liters was 0.5 of flowing air.

The results as yet only show macroscopic and histologic changes in the dust-interception powers of the nose. Healthy controls used, did no inhaling; such animals showed no characteristic

42. Cetrángola, A. A. and Trebino Ferrari, O.

Pulmonary Silicosis.

Semana méd. 1: 901-906, March 22, 1934.

The author discusses a case in which the clinical and radiographic findings did not permit of any other diagnosis than that of pulmonary silicosis. Man 26. Discusses literature affecting diagnosis of this condition. 3 illust., 2 of them histologic; one a radiogram of the thorax. He says that there is much similarity between tuberculous granulation and the silicotic pulmonary nodule.

43. Chamberlain, C. W.

An Ancient Occupational Disease in Modern Industry.

Radio Talk No. 207. Feb. 23, 1934. Illinois State Dept. Pub. Health.

Ramazzini's book was the first publication of its kind in the history of occupational diseases. Published in 1700, it made special reference to one of the most ancient of workingmen's maladies, the disease of stonecutters, who "oftentimes suck in by inspiration, the sharp, rough, and corner'd small Splinters of Particles that fly off; so that they are usually troubled with a cough, and some of 'em turn Asthmatic and Consumptive . . . and in dissecting the corpse of such Artificers, lungs have been stuck with little Stones. Diemerbroek gives a Curious Relation of several Stone-cutters that dy'd Asthmatic and were opened by him; in whose lungs he found such pieces of Sand, that in running the knife through the Pulmonary Vesicles, he thought he was cutting some Sandy Body."

Among the occupations which contribute sufferers from silicosis may be mentioned: mining, cement making, sand blasting, stone cutting, blast furnace work, soap making and particularly the manufacture of abrasive soaps and powders, paper making where agalite is used in coating paper, the working of fire clay and other clays, the spray coating of plumbing fixtures, rock drill-

ing, stone and metal polishing and grinding, sculpting, work in the cotton and textile trades, the making of monuments and tombstones, the manufacture of sand-paper, gem cutting and polishing, the glass industry, the pottery and porcelain trade, and the construction of railways, highways and tunnels.

The literature on silicosis includes the following as contributing a silica dust responsible for the disease: bituminous and anthracite coal, gold, silver, zinc, lead, copper, iron, sand, granite, sandstone, shale, silica flour and sand, feldspar, lava, silicon, carbide, silundum, fibrox, French chalk, tremolite, talc dust, pumice, mica, talc, asbestos, willemite, meerscham, sodium silicate (water glass used as a filler in soaps), hydraulic lime, soap stone, serpentine building stone, aillmanite, topaz, Fuller's earth, kaolin, clays, ultramarine, agalite, rock dust in coal mines, cotton and textile dusts, garnet (used as watch bearing, in gem cutting, polishing and grinding), permutite (used in water softening), slate, glass and many others. Some authorities make a differentiation between silicosis and silicatosis, the one being caused by combined forms and the other by free silica.

Recent investigations have shown that the dust particles in order to be absorbed into the lung tissue must be much too small to produce a direct mechanical injury. Furthermore it has been shown that silica is not the totally insoluble substance it was once believed to be.

44. Chamberlain, C. W.

Asbestosis as an Industrial Disease.

Radio Talk No. 209. March 2, 1934. Illinois State Dept. Pub. Health.

Although the exact differences between silicosis and asbestosis are not well-defined, anatomic and radiologic observations have shown that the appearance of asbestos fibrosis seems to be sufficiently distinctive to avoid confusion, asbestos causing a softer, more delicate, and more diffuse type of fibrotic changes; the importance of a full

medical and industrial history is emphasized as an aid to differential diagnosis.

The first American study of silicosis was made in 1914 and 1915 by Lanza and Higgins while the first study of asbestosis in this country appears to have been made by Soper in 1930. Early in 1924 a fatal case of asbestosis was reported, the postmortem examination revealing a fibrosis of the lungs which was said to be due to the inhalation of asbestos dust.

The principle of preventive medicine is of vital consideration to industry during and subsequent to business recovery; the role of the physician, in the prevention and treatment of sickness among the workers is becoming more and more recognized as the medical man points out that any industrial efficiency scheme which does not consider the health of the worker can be only partly successful.

45. Church, L.

Easy to Get Rid of Dust.

Factory Management. 92: 460-461, Oct. 1934; 512-513, Nov. 1934.

Various methods are in use for the disposal of dust. Receptacles or conveyances of the container type are commonly used, perfectly dust-tight except for an opening in the top, with a sleeve which can be connected to the dust hopper valves of the collector. Screw conveyors often take the dust away from the hoppers, mix it with water at the time of discharge from the collector hoppers; the wet mixture is then pumped into a territory near the plant where it may be allowed to settle, into conveyances with tanks for hauling it away.

If dust is discharged and carried away in dry form, this operation should be performed during hours when the plant itself is not in operation in order to minimize the danger of exposure to any dust that may escape.

The cost of a suitable dust system is never high

if, first, it is properly engineered: second, considered in the light of the benefits to be derived. These benefits are manifold, but cannot always be expressed in dollars and cents. They may consist of one or more or all of the following: (1) reduction in accident hazard, (2) reduced labor turnover, (3) greater efficiency of employees resulting in reduced production cost, (4) reduction in wear and tear on machinery, particularly where dusts of an abrasive nature are involved, (5) the saving of large quantities of a product, when the dust collected is the finest part of the product and has in many instances the greatest value, (6) reduction in labor by the elimination of sweeping and cleaning (7) elimination of danger to health of employees (8) elimination of a nuisance to the industrial establishment itself as well as to its neighbors (9) the aesthetic value of a clean plant (10) prima facie evidence that the employer is using every reasonable measure to protect the health of his workmen therefore providing a defense against the legal depredations of the dust racketeer or ambulance-chasing lawyer.

46. "Comfo" Respirator.

Industrial Finishing. 11: 34-35, Dec. 1934.

The Mine Safety Appliances Co., Pittsburgh, Pa., has introduced a new low-priced dust respirator that departs radically from old ideas of design. The new protector, called the MSA Comfo-Respirator (patented), has twin filters, one being carried on each cheek. This results in: (1) the filters are removed from the path of the moisture-laden exhaled air; (2) the filter area is greatly increased. The unusually large filter area—combined with the use of special filter material, scientifically treated to stop microscopically-fine dust—gives the Comfo exceptionally high protective efficiency, low breathing resistance and long filter life.

The Comfo-Respirator is especially designed to remove workmen's characteristic objection to

67. Deferred Liability for Silicosis Causes Alarm.

Insur. Field, 63: 13, May 3, 1934.

Recent alarming increases in the number and amount of damages allowed for silicosis claims have directed attention to the deferred liability under the compensation insurance. A jury at Belleville, Ill., on April 23 awarded \$15,000 damages against the Sterling Steel Casting Co., to William Blake, who claimed that he had contracted silicosis while working for it from 1921 to 1931. Many larger judgments have been awarded on equally old claims, and in numerous cases companies which have been off a line for years are held liable on the claim that the dust which caused the silicosis was inhaled years before, although its effects did not become evident until much later. The compensation companies have not been able to collect enough to pay their current claims, to say nothing of this enormous deferred liability now developing and which is being encouraged by ambulance-chasing lawyers.

68. Desoille, H.

Pneumonoconioses Problem.

Médecine, Monograph, supplement, 15: 1-56, Oct. 1934.

French experts generally do not recognize silicosis apart from tuberculosis—at best they call it pulmonary fibrosis, not compensable in France, not even an occupational disease. So it is not surprising to find M. Desoille in his extensive monograph, opening with the question "Do pneumonoconioses exist or not as disease entities?" His closing summary answers his query: "Many unknown factors persist in connection with pneumonoconioses—of which it seems certain they have no individuality at all clear."

With or without an identity true French precision requires this authority to begin his exposition with a definition: spelled with either c or k, "pneumonoconioses are pulmonary manifestations due to dust." The term itself was invented

by Zenker in 1866. Taken thus literally wholly disparate diseases are included. Dusts are (1) toxic (lead, mercury, manganese); (2) caustic (slag of dephosphorization); (3) infectious (coal, mycoses, etc.); (4) flabby and flexible (cotton, wool, feathers); (5) hard and wounding (saw-dust, silica dust).

Those infectious in character, toxic, cancerous (cobalt or arsenic mines), or allergic (asthma, due to dust), as well as the acute pneumopathies, whose most important variety is pseudo-pneumonia, due to inhaling slag-dusts, lime and phosphoric acid dusts—are eliminated. Also, not entered under the pneumoconioses are "pulmonary changes of color" and pulmonary sclerosis, due to prolonged dust inhalations, which contrast with the *nosoconioses* (Layet) comprising every disease caused by dust (pulmonary, cutaneous, and so on).

The author proposes for study: anthracosis (coal dust inhalation); silicosis (inhaled silica dust); asbestosis (asbestos dust); siderosis, schistosis, gypsosis, etc. (of mineral origin); tabacosis, byssinosis, etc. (of vegetable origin, tobacco, cotton, etc.); of animal origin. The paging devoted to these in turn gives an idea of the relative importance the author assigns them; thus: anthracosis which to him is hardly a disease at all, but more of a "pulmonary discoloration," "just a depot," or "lung-tattoo," has 4 full pages, while silicosis requires 23 before the author concludes that it actually does exist as the particular affliction of workers in silicious districts—MM. Rist and Doubrow to the contrary, who deny it as a disease entity. Asbestosis has $2\frac{1}{4}$ pages; the remaining 10 kinds of pneumoconioses comprise $8\frac{1}{2}$ pp. Legislation upon these conditions the world around is given a half-page, just listing the name of the country, the disease, and the contracting industrials.

Enough historical perspective occurs in the sections devoted to major dust diseases, with cita-

tion of experts and opinions along their evolutionary way, to interest and make definite the general reader; clinical, anatomical and diagnostic signs of these conditions, together with radiograph descriptions and a few illustrative reproductions, appeal to the medical student with their inevitable French clearness and discerning selection; conflicting opinions are judicially set out and often decision rendered either by the author or by the era involved. An eminently quiet exposition, one might term this monograph, dispassionate, unemotional; one is afar from suffocating lungs, or hearts that beat with "fetal rhythm, deceptive dextrocardia, due to the sounds on the rhythm of the right predominating over those of the left." The descriptions of South African authors are sometimes "terribly confusing"—why not? asks a tender-hearted reader, when "simple silicosis" cases were 29 to every 100 there; and tuberculosis cases were 49 to every 100 there; and tuberculo-silicosis cases were 89 to every 100 there in the last 26 years. Disconcerting too, to learn that in the silicotic autopsies, 2 out of every 3 are associated with tuberculosis. Of course, in South Africa, thanks to better ventilation, what was a fatal silicotic ratio of 8.94 to every 100 in 1917, was reduced to 2.55 by 1929. In some French mines 10 traveling employes who were among the strongest and best workers, were all dead in 10 years from chronic lung troubles. Eight workers, all young and hearty in 1905 were all dead by 1915.

In concluding the author cautions against confounding silicosis and its variants with the silicatoses, as from talc and asbestos. Silicosis is plainly confessed to be a dangerous disease even though its associations with tuberculosis are not entirely disentangled. "Thanks to hygienic progress the pneumoconioses are in retrogression and certain of disappearance." No preventive devices or appliances are treated in this article, though they should be "rigorously" present and in use. As Rist and Doubrow insist, all

the general measures recognized for tuberculosis should also be in force. Prudence too demands that workers showing signs of pulmonary sclerosis or cardiac deficiency after exposure to dusts, should avoid such conditions henceforward.

69. The Detection of Silicosis.

Colliery Guardian. 149: 1046, Dec. 7, 1934.

Various measures devised to prevent employees from contracting silicosis have been described in these columns, and it is, of course, complete prevention which offers the only entirely satisfactory solution to the problem. The attainment of this ideal, however, is at the present stage still rather remote, but it appears that if the disease could be detected at its onset, serious cases and fatalities could be almost, if not entirely, eliminated.

X-ray examination offers a positive method of obtaining a true conception of the condition of the lungs and at the same time of obtaining a permanent record of this condition. It is for the medical man to say to what extent X-ray examination is effective, but it is significant that Dr. Lewis, in the annual report of the Pontardawe Rural District Council, suggested that periodical X-ray examinations should be made amongst workers. The soundness of this suggestion is, it is believed, fully supported by other medical men who have made a special study of silicosis. The general appearance, and method of using the apparatus will be gathered from the illustration. The X-ray tube and transformer are mounted on the column at the right, the latter acting also as a counterweight for the tube and the mechanism which moves with it. In the illustration the operator is making a visual screen examination, i.e., a shadow of the chest is projected on to a screen coated with a chemical composition which glows brightly on being exposed to X-radiation. By this means an experienced operator is able to apprehend the condition of the lungs without ex-

posing a sensitised film. If any doubtful signs are present, or a permanent record is required, a film may be exposed with great rapidity and ease. A feature which makes the equipment particularly suitable for work at mines and quarries, is that it may be dismantled and packed into three small sections in a few minutes. Thus, instead of employees having to travel to a hospital for an X-ray examination, the apparatus is brought to the pithead in a car—a procedure which can be repeated as many times as necessary in the case of mines scattered over a wide area. Although so small in size, this apparatus will produce extremely good chest radiographs, and may also be used for other classes of work.

The apparatus described is a British product, made under Philips Metalix patents, by Watson and Sons (Electro-Medical), Ltd., Sunic House, Parker-street, W.C.2.

70. Dible, J. H.

Silicosis and Malignant Disease.

Lancet. 2: 982-983, Nov. 3, 1934.

The occurrence of three cases of malignant disease in 14 post-mortem examinations made upon persons suffering from silicosis in East Lancashire since 1930, seems worthy of record for the purpose of directing more attention to the possible presence of an etiological factor in the material causing the pneumoconiosis. In two of the cases to be cited two independent malignant growths of different types were present: in one instance lung and colon were involved and, in the second, bile-duct and prostate. A case of myeloid leukaemia, in a subject who was suffering from advanced silicosis, is also included since there are many reasons for regarding this disease as an example of malignant neoplasm.

The connection between the malignant disease and the silicotic condition would seem clearly indicated in the two cases of pulmonary carcinoma, and also in the case of bile-duct carcinoma, where the tumour originated in an area of liver adherent

to the diaphragm, which in its turn was bound by a greatly thickened pleura to the base of a lung affected with advanced silicosis. The existence of a second and independent tumour in two cases may be variously interpreted, but is at least compatible with the theory of the existence of a diffusible carcinogenic agent. The fact that tuberculosis was only detected in one of these cases would tend to suggest that a degree of resistance to this disease, perhaps unusually well developed, may have permitted an exceptionally prolonged exposure of the tissues to the irritant agent.

71. Differentiation of Noxious Dusts.

Lancet. 1: 903-904, April 28, 1934.

Editorial comment is here offered on the international medico-legal importance of dangerous industrial dusts. At the May meeting in Geneva national representatives were to draw up a preliminary charter securing compensation for those damaged by silicosis, one of the highest industrial disease hazards; it was thought unlikely that asbestosis would yet be granted international status. The sequence leading up to such recognition usually begins with private action: a firm or group of firms conducting an industry admits their workers are liable to a risk; the admission may be made spontaneously or under pressure of local public opinion; research into methods reducing or avoiding the risk usually accompanies. Regulations may follow and a scheme of compensation for those injured in proportion to their damage. The next stage coordinates such schemes in any one country by laws designed to bring all employers up to the standards set by the best (as in South African gold fields); in England the Medical Research Council subsidizes research into the causes, possibilities of prevention and cure of the pathological conditions set up by the dusts. When national conscience in several countries has reached a certain sensitivity, then international action may be hoped for. Silicosis has reached

this stage; other allied conditions will demand similar recognition in the future.

In differentiating dangerous dusts the work of Dr. L. U. Gardner and Staff at Saranac Lake is discussed as highly important. Their technique, however, has the primary disadvantage of requiring years rather than months of exposure to the dust in estimating results. The sericite work of W. R. Jones and that of E. H. Kettle on differential diagnosis of biologically harmful from biologically inert dusts are pertinently epitomized. The editorial desires a more humane method of diagnosing dangerous dusts than merely watching the fate of employees and a more certain method than studying animals in dusting chambers.

72. Discussion on Symposium on Pneumoconiosis.

Illinois M. J. 66: 439, Nov. 1934.

Discussion of the papers on pneumoconiosis and silicosis, appearing in the *Illinois Medical Journal*, Nov. 1934, was participated in by Drs. Don C. Sutton (Chicago), and Dr. Wm. D. McNally (Chicago).

73. Doctors Seek Law's Spread To Silicosis.

Would Have Occupational Diseases Covered by Compensation.

Syracuse N. Y. Herald. Nov. 17, 1934.

Silicosis, a disease which has caused hundreds of disabilities and deaths among miners, quarry men and workers in abrasive and dust-producing industries, will be listed among occupational diseases subject to the Workmen's Compensation Law, if the New York Society of Industrial Medicine wins a legislative battle this winter.

Thousands of claims against employers are now flooding the civil courts of the State as workers seek damages for the contraction of the disease, it was said by Frank Redmond, Buffalo, secretary of the society, at its 14th annual meeting at the Onondaga Hotel Thursday.

Mr. Redmond, who is educational director of the

Associated Industries of New York State, said that the average award for the contraction of silicosis is approximately \$3,500 and cited one instance in which \$14,000 damages was granted.

John L. Train, general manager of the Utica Mutual Insurance Company, recommends that silicosis be brought under the Workmen's Compensation Act, not only to prevent suits for negligence but to induce employers to install suction equipment to remove dust from the air the workers breathe.

As defined by Dr. Leroy U. Gardner, director of the Saranac Laboratory, which was founded by Dr. Edward Livingston Trudeau, silicosis is a predisposing factor in the development of tuberculosis. It is contracted by the breathing of rock dust into the lungs, with the result that the tissues are hardened over a period of years.

The 100 physicians from all sections of the State were urged in another address to be on their guard against the socialization of medicine.

74. Dreessen, W. C.

Effect of Inhaled Marble Dust As Observed in Vermont Marble Finishers.

U. S. Pub. Health Rpts. 49: 724-732, June 22, 1934
also *Reprint* No. 1630.

The author gives the clinico-Roentgenographic findings in 80 marble finishers, to determine the effects of inhaling marble dust. Observations of the dust content of the air at the breathing level and analyses of the dust had been recorded.

Although marble dust when inhaled in the concentrations here observed, produces a mild bilateral, linear fibrosis in a certain number of cases (termed herein "commencing generalized fibrosis"), no serious lung changes were noted, and there was no disability due to the dust, even after many years of exposure. The findings of this study, which was conducted under the direct supervision of Surg. Albert E. Russell, were regarded as essentially negative.

75. Drinker, C. K., Field, M. E. and Drinker, P.

The Cellular Response of Lymph Nodes to Suspensions of Crystalline Silica and to 2 Varieties of Sericite Introduced through the Lymphatics.

J. Indust. Hyg., 16: 296-299, Sept. 1934.

Certain of the lymph nodes of two dogs were injected with two types of sericite and with crystalline silica for control. After 10 and 12 days, the dogs were killed and the nodes containing sericite showed changes similar to those which had received silica. It may thus be inferred that dusts containing either of these sericites will upon inhalation react as does crystalline silica.

76. Dubrow, J. L.

Pneumoconiosis; With Special Reference to Some of its Complications.

Radiol. 22: 202-211, Feb. 1934.

The different types of pneumoconiosis vary in relation to the industry, viz.: anthracosis in coal miners, silicosis in rock drillers, asbestosis in asbestos workers, siderosis in gold miners, and byssinosis in cotton mill workers. Each of the above tend to cause the development of pulmonary fibrosis (pneumoconiosis). The author describes the shape, size, extent of exposure, number of dust particles per cubic feet of air, and the physiochemical properties. He reports six cases illustrative of different types of pneumoconiosis in the various stages.

77. Dust Collection.

Coal Age. 39: 446, Nov. 1934.

The Thermix dust-collecting fan of the Prat-Daniel Corporation, 350 Fifth Avenue, New York City (as also Thermix multi-cyclone dust collectors for dust collection service) consists of a conoidal multi-blade fan with a scroll continued around the axis for an additional 180°. Centrifugal force along the elongated outside scroll sheet (equipped with wearing plates) causes separation as in a highly efficient cyclone. A skimmer operating within the scroll and parallel to it, withdraws the 10 to

15 per cent of gas containing the concentrated dust, at the same velocity as the gas passing through the scroll, thus eliminating eddies which might interfere with efficient separation. The gas bears the concentrated dust to an auxiliary cyclone, where most of it is eliminated. The gas then returns to the system inlet to give a second clean-up. No auxiliary power is required for this but efficiency is markedly increased. The fan can be operated to skim off a lower gas slice at low rating, thus there is constant dust collecting efficiency at all ratings.

The multi-cyclone collector is as clearly described. Both are illustrated by machine drawings.

78. Dust Collection.

Solving a Works Dust Collection Problem.

Removing and Recovering fine Dust from Large Volumes of Air.

Chem. Age. 30: 431-433, May 19, 1934.

A great variety of industries produce large quantities of fine or coarse dust during their manufacturing processes, which must be collected to safeguard the health of workers and prevent nuisance and damage to adjacent machinery; also the dust itself being often valuable its collection is an economic necessity. This article illustrated comprehensively with drawings and photographs shows in detail and to advantage the Visco-Beth dust collector, supplied by the Visco Engineering Company, Ltd., London.

79. Dust-Control Devices Aid Fight Against Silicosis.

Eng. N. 112: 284-287, March 1, 1934.

An investigation into the practical use of the Kelley trap for the control of dust generated by pneumatic rock drills was made on the installments used on two large buildings in New York City. The dust exposure of the drillers is only approximately 5 million particles per cubic foot of air as compared to several hundred million particles per cubic foot without the dust trap, so one can really see the value of this device.

The functioning of the dust separating units is: (1), the first two stages separate out the coarser

due to reflections of light from immense numbers of minute cavities, each containing liquid and a gas bubble. In Hallinger quartz the number of visible cavities per cubic millimeter was on the order of one million, seldom less than 200,000, rarely more than three millions. Mr. Edge reported many cavities as containing two immiscible liquids as well as a gas bubble, the latter kept in perpetual agitation by the Brownian movement of its gas molecules. One of the two liquids was liquid carbon dioxide, identified easily by its critical temperature (31°C). The other liquid appeared to be water or some aqueous solution. With liquid CO_2 present, the pressure existing in these cavities for any particular temperature could be deduced: a piece of this quartz placed in the mouth a few moments, would be raised to the body temperature (37°C), or 6° above the critical temperature of CO_2 . All the quartz being subjected to body temperature, all the CO_2 would be gaseous; pressure within the cavities would be around 84 atmospheres, or half a ton to the square inch. With the pressure suddenly released, say, by the solution and eventual bursting of a cavity, the compressed CO_2 would almost instantaneously expand to 400 times its former cavity volume. Some quartz specimens examined did contain between three and four times their own volume of CO_2 (the latter being released at conditions of normal temperature and pressure). In the Rand quartz Mr. Edge found similar conditions, and he pointed out that when quartz particles taken into the lung, contained a large number of such cavities, something would be bound to happen to the lung when these particles disintegrated and discharged their gases under pressure into surrounding tissue.

89. Efficient Dust Separation.

Advantages of Electro-Static Methods.

Queensland Govt. Min. J. 35: 260, Aug. 15, 1934.

In many industrial fields, including the crushing, roasting, and general treatment of ores, blast-

furnace and boiler plant operation, electrostatic methods are recognized as the most efficient for dust or gas removal from air. In the past an impression prevailed that such methods were costly in power consumption; this has been shown incorrect, as the total net power consumption is considerably less in many cases than either centrifugal or water-washing methods. Due to effective co-operation of the 5 main companies operating the Lodge-Cottrell process in Great Britain, the United States and Germany, with pooling of all patents and experience the electrical power now required averages only $1\frac{1}{2}$ to 2 kw. h. per million cu. ft. of air, combustion gas or other medium. This equipment is represented by over 3000 electro-filters, and 95 to 98 per cent of the total dust is removed from air or gas, irrespective of the size of the particles: This is a highly important practical point as removal of particles below 10 microns (1 micron is 1 thousandth of a mm.) is extremely difficult by ordinary methods. Sir Oliver Lodge is the practical pioneer in this field, his work dating back to 1883; Dr. F. G. Cottrell, of the United States, Dr. Erwin Moller in Germany associated their patents, researches and experience with those of Sir Oliver Lodge. The British Company was Lodge-Cottrell Ltd., of Birmingham (George St., Parale) and London, having developed from the original Lodge Fume Deposit Company. An intelligible description of the Lodge-Cottrell equipment closes the article. Its basic principle consists in the use of chambers or towers, made, say, of concrete, through which pass the dust or fume-laden air or gases.

90. Ellman, P.

Pulmonary Asbestosis.

Brit. J. Radiol. 7: 281-295, May 1934.

Part III.—Pulmonary Asbestosis. While the use of asbestos in industry can be traced to antiquity, it is only during the last 6 or 7 years that the subject of pulmonary asbestosis can be said to have received any real attention as a serious industrial lung disease.

The author gives a detailed report on the radiological and pathological features; the macroscopic and microscopic appearances, and the asbestosis bodies. He concludes: the disease must be grouped with silicosis as a very serious pneumoconiosis. The average length of employment in fatal cases is only one-half that of silicosis.

A diagnosis, which is most likely to be made during the winter months, when acute respiratory complications occur, must depend upon a combination of factors: (a) opportunities for inhaling asbestos dust; (b) the occurrence of asbestosis bodies in the sputum; (c) clinical and radiological findings of the diffuse, pulmonary fibrosis commencing at the bases; the characteristic radiological findings are probably the most important reliable single piece of evidence in early diagnosis.

The clinical features of the disease indicate that the onset of symptoms usually occurs after some 5 to 10 years of exposure to the dust; the degree of dyspnoea and emaciation, the complexion, the absence of haemoptysis and the very scanty expectoration, are all characteristic features.

Inhalation of asbestos dust must be expected sooner or later to produce pulmonary fibrosis, depending upon (a) length of exposure, and (b) nature and concentration of the dust.

The histological features of asbestosis are essentially (a) diffuse chronic interstitial fibrosis of the lungs, with areas of acute catarrhal changes, and (b) the presence of characteristic asbestosis bodies, appearing either singly or in clumps.

The presence of isolated asbestosis bodies in the sputum is indicative merely of previous exposure to asbestos dust, and does not necessarily have any clinical significance; if the bodies occur radially arranged in clumps they suggest disintegration of pulmonary tissue.

Pulmonary asbestosis, once established, is a progressive disease with a bad prognosis; its treatment can only be symptomatic.

The tuberculosis risk in asbestosis must be reckoned

with, even though time has yet to indicate whether it is less, equal to, or perhaps even greater than that in silicosis.

91. Epidemic of Silicosis Suits Raging in Missouri.

Weekly Underw. 130: 453, March 3, 1934.

An epidemic of suits aggregating \$974,000 against one company alone, have been filed in Missouri in which the plaintiffs seek damages on the grounds they contracted silicosis while in the employ of the defendant concerns. A. F. Brinkman, a claim adjuster, testified that 45 suits for an aggregate of \$974,000 had been filed against the Tavern Rock Company and that the company had been informed 200 other suits were contemplated.

92. Evans, D. W.

Dust Control in Traprock Production.

Excavating Eng. 28: 361-364, 368, Aug. 1934.

Anyone who has lived near crushed rock plants knows the thick dust they kick up, especially when a hard rock is handled. The Consolidated Stone & Sand Company, due to their modernizing, affords a welcome relief from this condition. Every conveyor, every screen, is completely enclosed in a housing of matched lumber, each housing with a conduit leading to a powerful suction fan with a capacity of 46,000 feet of air per minute. This fan is the heart of the Sly Dust Arrester system which not only eliminates undesirable working conditions but pays dividends as well. This system was installed last October on the very justifiable grounds that it prevents dust from getting into bearings, and also prevents that dread disease—too prominent in rock plants of late—silicosis.

The suction fan draws dust from all parts of the enclosed screen and conveyor system to a central series of folds of heavy cloth which catch the dust and deposit it in bins for convenient removal. Here two men load the dust into 100 pound bags which find ready market for filler in asphalt shingles, some varieties of paint, and many other industrial uses.

est to test these views by ascertaining experimentally what reactions would be obtained in animals given these silicates, either by inhalation or intratracheal injection.

105. Franks, W. R.

Silica Dust.

Canad. M. A. J. 31: 245-249, Sept. 1934.

The author first describes the methods by which silica particles find entrance into the lung cells. There are many methods used for measuring the dustiness of the air, both gravimetric and chemical. The Konimetric method of counting, used in South Africa, is perhaps the most satisfactory; but this method is both arduous and fatiguing and only about a dozen estimations can conveniently be completed in a day.

The author has recently developed a method which obviates this difficulty. Instead of counting the particles, the amount of light reflected by the dust spot under the dark field is measured photometrically. Under standard conditions this is proportional to the surface of the dust recovered. The light may be measured either by a comparative photometer or a photo-electric cell. The author gives a table to show the comparatively good results obtained by this method.

The highest dust concentrations to which miners are exposed are encountered in drilling and blasting operations. The author discusses and illustrates methods by which such concentrations have been greatly reduced, such as a new type of dust concentrator used by the Ontario Mining Association.

106. Froment, J. and Guichard, A.

Case of Pulmonary Silicosis with Spontaneous Pneumothorax Developing in a Former Miner.

Lyon Méd. 66: 391-395, Oct. 14, 1934.

A former coal miner, 50 years old, in perfect health who had left his calling 9 years before, was suddenly seized with dyspnoea and cyanosis, with symptoms of spontaneous pneumothorax confirmed

by the radiograph. For 23 years he had been a borer in mine pits in various parts of France, Belgium, and England; had always been well; had retired on a pension 9 years before, becoming a farmer. Unsuspected silicotic invasion of his lungs was revealed in the study of his condition. No tuberculous signs appearing, the diagnosis of pneumoconiosis stood—the pneumothorax being resorbed in 30 or 40 days. Interest was taken in the cause of the pneumothorax, which was shown not to be from strain or that of conscripts. An emphysematous vesicle or small tubercle rupturing into the pleura, were possibilities.

107. Fulton, W. B., Houtz, R. L., Dooley, A. and Matthews, J. L.

Asbestosis.—Pt. 1: The Collection and Counting of Dust Encountered in Asbestos Fabricating Plants.

Penn. Dept. Labor and Industry. Special Bull. 37, Oct. 1, 1934.

The Pennsylvania Department of Labor and Industry is engaged in several surveys on the effect of dusts and their relation to the health of the worker.

In a survey now being conducted in the asbestos industries approximately 150 samples have been collected and the data presented in this paper.

Samples of dust, taken in plants for fabricating asbestos products, were collected by a modified form of the standard method, using a 95.0 per cent U. S. P. ethyl alcohol as a medium. In this medium the particles were well distributed and readily counted. When distilled water was utilized as the collecting medium agglomeration occurred immediately and made counting impossible. Dispersion of the dust in alcohol is good. A settling time of at least 30 minutes was required before counting. The particle count in any given field remained constant after this period. A modified holster is described. It is constructed of leather straps, in such a way that the fluid in the impinger flask is visible at all times.

has come in for special award under varying occupational provisions.

The status abroad is discussed in terms of mining statistics of work done, its value, and outlay for silicosis damages. Medical control is indicated for areas that insist upon it.

In the United States silicosis compensation has been hindered by association with forms of racketeering bitterly resented by employers.

The reference value of this series of articles is high and will undoubtedly awaken, as well as maintain, active interest in the subject.

193. Kellogg, F. S.

Silicosis Claims—A New Problem in the Insurance Field.

Address delivered before *Ins. Sect. American Bar Assn. at Milwaukee*. Aug. 27, 1934.

In speaking of the subject "Silicosis Claims—A New Problem in the Insurance Field," the first question to be answered is "What element creates the problem?"

We may take as a definition of silicosis, that it is an injury to the lung tissue caused by the prolonged inhalation of dust, which dust contains the element silicon where such inhalation of dust results in the substitution of scar or fibrous tissue in the place of the normal tissue of the lung.

Apparently the only certain diagnosis is autopsy, which can, of course, only be performed after the disease is ended. The next method of diagnosis on the scale of certainty is by x-ray examination. In addition to the x-ray examination, there is a case history and a clinical examination. The slow rate of progress of the disease is another medical aspect of silicosis which makes it extremely difficult to adjudicate.

Prevailing opinion is that disabling injury is only caused by dusts which contain silicon and oxygen in chemical combination in the form of free silica. I have said that this is the prevailing opinion, but there are other opinions and there are certain de-

velopments which cause one to hesitate to say that silicon dioxide (free silica) is the only cause of silicosis.

Silicosis was first carefully studied in the South African gold mines. The rock there mined contains a large percentage of free silica, say 70 to 80 per cent. This free silica was fixed upon as the cause of silicosis, prevalent among the miners. But gold mining has been carried on also and for many centuries in India. The rock in India contains a higher percentage of free silica than in South Africa. One would expect that the Indian miners would be afflicted with silicosis to a greater degree than those of South Africa, but investigation seems to prove that the Indian miners are practically free from the disease.

It seems reasonably certain that asbestos dust will cause changes in the lung which will appear on an x-ray plate, and that these appearances are somewhat different from those of silicosis. There is further question as to whether asbestos, causing the condition known as asbestosis, produces a disabling effect. Another contention is that asbestos almost always contains a small amount of free silica in addition to the silicates.

Recent opinion seems to be that particles above five microns will not enter the lung, because they are too big. Whether there is any degree of smallness below which particles would be harmless is not known, because the limit of microscopic visibility is reached. At the present time it is thought that dust particles of half a micron are the most injurious.

These factors in connection with silicosis, namely: its extremely slow development, the difficulty of making diagnosis, the further difficulty of determining whether any industrial environment will produce a dust of sufficient fineness and of proper chemical composition to cause the disease—these, among other factors make it so difficult justly to adjudicate claims for injury by silicosis, whether by common law procedure or under the Workmen's Compensation system.

Proper procedure for the adjudication of claims for silicosis must include the submission of every claim to a competent and impartial medical board. The claimant himself should be required to submit to physical examination, including x-ray examination. If the claim be made by the next of kin or dependents of the deceased employee, then there should be in each case an autopsy conducted by a medical man of experience in the diagnosis of silicosis.

194. Kerner, O.

Illinois Occupational Disease Act, Defined. Attorney General Rules Against Exclusions Contained in Policies.

Insur. Field. 63: 10-11, Oct. 25, 1934.

An insurance carrier in Illinois may not, by an endorsement to the standard Workmen's Compensation policy, relieve itself of liability to pay compensation for occupational diseases enumerated in Section 2 of the Occupational Disease Act, Attorney General Otto Kerner of Illinois held October 15 in an opinion.

In view of the large number of claims being filed under both the common law and the Occupational Disease Act the companies had sought to relieve themselves of the occupational disease liability by an endorsement, and legality of this action was submitted to Attorney General Kerner, with the resultant opinion. This leaves the compensation companies in the same unfortunate condition as formerly with occupational disease claims piling up at a ruinous rate and with apparently no remedy except a change in the law. The opinion cited the fact that in 1923 the Illinois legislature provided that employees in occupations enumerated in Section 2 should be placed upon the same plane as employees who suffered accidental injuries arising out of and in the course of their employment. The amendments also removed the right of employees to bring civil suit in case of injury.

"Section 26 of the Workmen's Compensation Act," the opinion said, "provides that any employer who shall come within the provisions of Section 3 of the act and any other employer who shall elect to provide and pay compensation provided for in this act shall, in accordance with sub-paragraph 3, insure his entire liability and said sub-paragraph provides that all policies of insurance carriers insuring payment of compensation under the act shall cover all employees and the entire compensation liability of the insured, and that any provision of the policy attempting to limit or modify in any way the liability of the insurance carried, shall be wholly void.

"Section 3 of the Workmen's Compensation Act provides that the provisions of the act shall apply automatically and without election to all employees engaged in any of the enumerated businesses or enterprises * * *

"When an employer is liable to pay compensation, whether it be under the provisions of the Workmen's Compensation Act or by virtue of the provisions of Section 15 of the Occupational Disease Act, he nevertheless has elected to provide and pay the compensation provided for in the Workmen's Compensation Act * * * and there is only one compensation provided for and that is the compensation as provided in Section 15 (b) 1 of the Occupational Disease Act, namely, if an employee is disabled or dies by reason of an occupational disease arising out of and in the course of his employment, in one or more of the occupations referred to in Section 2, he, or his dependents, 'shall be entitled to compensation in the same manner and subject to the same terms, conditions and limitations as are now or may hereafter be provided by the Workmen's Compensation Act for accidental injuries sustained by employees arising out of and in the course of their employment.'"

"If an employer must compensate his employees

209. Leary, T.

Variation in Lung Lesions Produced by Silica and the Silicates.

Safety Eng. 68: 41-42, July 1934.

The impression exists that tuberculosis is a necessary sequence of silicosis in order to produce death. This is not true, particularly in asbestosis.

The cases of pure silicosis, i. e. without tuberculosis, and of asbestosis without tuberculosis, illustrate the varied picture which the lungs may show. From three asbestosis cases the influence of a widespread focal emphysema, occurring in regions where the asbestosis bodies are massed, makes plain that this lesion together with the fixation of the lung by thickening of the subpleural tissue is responsible for the dyspnea and the relative insufficiency of the respiratory mechanism. Fibrosis within the lung is slight in degree and of minimal importance. On the other hand, fibrosis within the lung, replacing a large part of the vesicular substance, together with subpleural thickening and adhesions, bring about the same interference with respiratory efficiency in the silicotic.

210. *Legge, Sir Thomas.

Industrial Maladies, Lond. Oxford Univ. Press, 1934.

Rev. In: *Monthly Labor R.* 39: 1308, Nov. 1934.

This posthumous work of the writer, who was formerly Senior Medical Inspector of Factories in England, covers most of the important industrial diseases. There is a review of the development of interest in hazardous working conditions and of the regulations requiring notification of industrial disease. A chapter is directed to compensation for industrial diseases.

*Not available for review.

211. **Lehmann, G.**

Dust from the Nasal Passages as Cause of Lung-Silicosis.

Arbeitsphysiol. 8: 218-250, 1934.

In a previous paper (*Arb. physiol.*, 7: 167, 1933) the author described his apparatus by which a large number of persons could be examined for the possibilities of lung infection by dust which passed into the nose and its connections. Since then 700 examinations have been made by the author and his assistants, the test conditions and results being embodied in descriptions and diagrams of the present paper.

503 mountain workers engaged in breaking up stone were examined and the results compared with the figures for existing silicosis diseases. In the higher age classes those working in stone for 16 years or more the men who remained in good health, had nasal connections in good condition, with a very few exceptions. The silicotic workers had bad noses in the majority of cases. In the younger workers those early diseased were characterized by bad nasal connections. Mouth-breathers sickened, as a rule, since they had not the use of nasal passages in good condition.

From the findings the conclusion was drawn that besides duration of the work, the possibility for dust injury in the nasal passages was the decisive predisposing factor in lung silicosis.

Applying the principle of averages the danger begins when the expression

$$\frac{\text{Dust Activity in nasal passages—years of work-}}{2} \text{—ing at the oc-} \\ \text{cupation} = 0.$$

When the danger-index is positive, a silicosis infection is unlikely. The probability of a severe disease infection increases proportionally to the negative value of the index.

Research upon 52 polishers of the Remscheider area led to the conclusion that in carrying on the

tissue measurements in microns and air requirements in cubic feet.

243. Meiter, E. G.

What is a Dusty Factory?

Nat. Safety N. 29: 34, 36, April 1934.

To control the dustiness of the air intelligently, the amount of dust present must be determined. With a given dust two factors are usually considered: (1) the number of dust particles in a given quantity of air; (2) the particle size, usually expressed in a size frequency distribution curve, that is, the percentage less than stated size is plotted against the size of particles.

Many instruments have been devised for determining the dust concentration in air, and the author gives a brief historical review of the subject.

244. Meller, H. B.

Clean Air, an Achievable Asset.

J. Frank. Institute. 217: 709-728, June 1934.

Recent data indicates there is a "safe load" of air pollutants, hygienically speaking. Up to a certain concentration in mines and factories, non-toxic dust is relatively non-injurious to the worker; above these, permanent injury results. Research in out-door pollution is being directed toward ascertaining amounts and kinds of safely-breathed contaminants. In city air common products of fuel combustion are silica and carbon; much recent research has been directed toward the harm to the lungs from breathing silica compounds. Dr. William McNally has found that the normal lung contains 1.13 mgrms., SiO_2 per gram. of dried lung tissue. over 2 mgrs. SiO_2 per grm. of dried lung tissue shows undue exposure to dusty air. The silica content of city dust can exist in concentrations amounting to "undue exposure." Dr. McNally cites 8 cases of persons in varied occupations dying of different diseases where silica amounts breathed in, were believed harmful; 2 were houseworkers; 1, an

automobile painter; 1, a man who did part-time as a farmer, and part-time as steel mill worker. This last showed 7.06 mgrms. of silica per gram of dried lung tissue. A building worker showed 2.37 mgrms.; a coke shoveler in a coal yard 9.75 mgrms. or 8 times the normal amount as stated by Dr. McNally.

The age at which workers take employment in one dusty occupation is stated as a large factor in determining the length of time required to develop a first stage in one dust disease (Dr. R. R. Sayers, U. S. Public Health Service). Workers under 40, will average 13 years in contracting it; over 40, will average 7.8 years. Age thus may bring a progressive decrease in bodily resistance or else a dust build-up has occurred in the lungs. Dust is a sizable agent in cutting down the productive capacity of workers, such a loss being permanent, whereas many other forms of common illness may be only temporarily disabling.

Eventually it may become possible to state the average amounts of dust that street air can safely carry. The amount can then be delimited at the source of smoke and the dust-production. Indoor air-conditioning has been greatly hampered in development because of aggravated outdoor air pollution. This industry is proceeding on the assumption that damaging dust lodging in the lungs, ranges in size from 6 microns to 0.4 micron in greatest diameter.

245. Merewether, E. R. A.

A Memorandum on Asbestosis.

Tubercle. 15: 69-81, Nov. 1933; 109-119, Dec. 1933; 152-159, Jan. 1934.

Asbestosis may be defined as a specific occupational disease of the lungs due to the inhalation of asbestos dust and characterized by progressive replacement of the essential active functioning tissue of the lung by inactive non-functioning fibrous or scar tissue. It is really a pneumoconiosis, a fibrosis of the lungs, and there-

fore is in the same category as silicosis, a disease it resembles in some respects, while differing considerably in others. Only recently has asbestosis been recognized as an entity of serious import.

The fibrous minerals commercially known as asbestos are silicates, the silica being combined with metallic bases, mainly magnesium or iron and, to a less extent, calcium, sodium or aluminum. The term is a collective name applied to a variety of silicate minerals which differ from each other in chemical composition and physical properties but resemble one another in their finely fibrous nature and flexibility.

The number of workers employed in the quarrying of asbestos and in the manufacture of asbestos-containing materials and articles is considerable, but the industry is very small as compared with, for example, the coal mining industry and, fortunately, only a small proportion is exposed to risk from the inhalation of asbestos-containing dust.

The first recorded case of asbestosis was that of a patient who died in 1900 in the Charing Cross Hospital, London. Post-mortem examination revealed extensive diffuse pulmonary fibrosis with no evidence of pulmonary tuberculosis.

Asbestosis—the pulmonary fibrosis of asbestos workers—is insidious in its onset, irregular in its course, and variable in its mode of termination. It is helpful to visualize the disease, as the slow growth of fibrous tissue (scar tissue) around the bronchioles or smaller air tubes of the lungs and between the air cells wherever the inhaled dust comes to rest: in contrast to the depots used in silicosis the bronchiole is the important site of deposition of asbestos in the lungs. While new fibrous tissue is being laid down like a spider's web, that deposited earlier gradually contracts. This fibrous tissue is not only useless as a substitute for the air cells, but with continued inhalation of the causative dust, by its invasion of

new territory and consolidation of that already occupied, it gradually, and literally, strangles the essential tissues of the lungs.

In common with other essential organs of the body the lungs have a large reserve of tissue for use in emergencies and to permit of a diminution in functional capacity due to advancing age or disease. For this reason and because fibrosis of the lungs is essentially a local disease, it is only when the fibrosis progresses to the extent of obliterating this reserve, that undue shortness of breath on any extra effort draws the worker's attention to the fact that his health is not what it should be. The other symptoms of the disease, such as cough, are equally unassuming, and are readily ascribed to some common and trivial cause.

From this point the progress of the disease is more rapid, since it is now encroaching on the remaining sound tissue of the lungs, already only just sufficient to maintain the worker in his ordinary daily activities. Ultimately, if no acute respiratory infection has precipitated a fatal termination, a stage is reached when the lungs can do little more than maintain life, and the shortness of breath becomes extreme.

Usually the fatal issue is determined by the onset of some acute infection with which the remaining undamaged lung tissue is quite unable to cope: this is commonly a low grade bronchopneumonia, but may be a lobar pneumonia, bronchitis, influenza, or, less often, a subacute tuberculous infection. With the exception of a mild degree of bronchitis which is more often mechanical in origin and not infective, being due to irritation of the retained dust, there is no evidence that the existence of developed asbestosis predisposes to the onset of such acute infections; but if, however, an acute infection does supervene, the presence of the asbestosis seriously impairs the chance of recovery. Details of cases in which the terminal infection was tu

berculous could be given which demonstrate this added risk unequivocally: in these cases the onset of the tuberculosis was indubitably recent in origin and undoubtedly precipitated the fatal termination, but on post-mortem examination the extent of the tuberculous lesions was clearly insufficient to have caused death under ordinary circumstances.

In the absence of intercurrent infections the fibrosis may progress to an extreme degree; bronchiectasis, non-tuberculous cavitation and spontaneous pneumothorax may occur, but 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.

Intercurrent attacks of dry pleurisy, which are partially responsible for the considerable thickening of the pleura which occurs, are common, but usually only cause slight and temporary disablement.

Clinical Signs.—The most important single clinical sign is that of diffuse bilateral impairment of the percussion note: this is slight in degree and best elicited by very light and rapid percussion of the back of the chest from apex to base on each side, and is associated with a slight sense of resistance. The other physical signs do not differ materially from those presented by silicosis and need not be detailed here. The symptoms exhibited also, as might be expected, closely resemble silicosis. For a considerable period the disease causes almost no inconvenience to the worker, and for a long time the symptoms may pass almost unnoticed. Between 50 and 60 per cent. of cases of asbestosis complain of cough and of undue shortness of breath on exertion, and show a duskeness or slight blueness of the lips which contrasts with the general pallor of the face not uncommonly seen.

The radiographic appearances of the developed or advanced stages of the disease are distinctive, although they are not specific. While, as is the case with silicosis, certain radiographic appearances may be looked upon as typical of the disease, frequently modifications of and departure from the typical picture occur.

Badham was the first to describe the fine type of fibrosis which can be caused by an inorganic dust containing no free silica, and to draw attention to its serious nature.

An inquiry in the United States showed that in cases of silicosis in granite cutters the radiographic features were atypical.

In workers exposed to the insulating material known as "magnesia" (85 per cent. magnesium carbonate and 15 per cent. asbestos approximately), with signs of pulmonary fibrosis, one finds a type of radiogram which is totally distinct from either typical silicosis or typical asbestosis, and from appearances intermediate between the two. It is impossible to say what the underlying lung lesion may or may not be, as no autopsy findings are available. Haynes however, states that magnesia is a dangerous dust, so the radiographic picture may truthfully reflect a modification of the asbestos fibrosis effected by the introduction of a preponderance of a more quickly soluble dust.

Radiograms of asbestos workers are very puzzling, more so than in the case of silicosis, when it comes to assessing the degree of asbestosis present, particularly in the earlier stages, and also in women, owing to the shadows cast by the breast tissue. Keating, of the Silicosis and Asbestosis Medical Board, has pointed out that pregnancy may produce a mottling suggestive of asbestosis.

In the light of present knowledge, therefore, one can say that asbestosis cannot be diagnosed with absolute certainty on either physical examination or radiological examination alone:

with the aid of both, the pneumonokoniosis can be diagnosed with certainty, if present in some degree, although not necessarily to an extent sufficient to cause either symptoms or any disablement. But it is not possible to say from physical and radiographical examination that an asbestos worker has not, trapped in the lungs, an amount of dust sufficient to cause the development of serious fibrosis after a lapse of time.

The greatest possible aid to the diagnosis of asbestosis is to have available for comparison a series of films of asbestos workers, together with their whole history and the post-mortem findings if death has occurred.

Asbestosis bodies are not found in asbestos dust, but McDonald came to the definite conclusion that they were derived from asbestos fibres modified by residence in the lungs. The asbestos fibre forming the central core in the asbestosis body was identified by Gloyne. The typical bodies have not been found in any other disease, but forms superficially resembling them have been found in a coal miner.

Since they may be found in the lungs, at any rate up to fourteen years after the last exposure to asbestos dust, their presence does not give any clue as to whether the exposure has been recent or long past.

Any manipulation of asbestos, produces dust, which is easily projected into the air. Different varieties and grades of asbestos also differ materially in dustiness.

Exposure to asbestos for less than five years can result in the development of a degree of asbestosis sufficient to cause death. Commonly, however, cases of definite asbestosis are not discovered on examination within five years of commencing work, although a few are found.

Particulars of cases seem to show that with high concentrations of dust the minimum period of time which must elapse between the commence-

ment of exposure and the production of a serious degree of asbestosis is approximately seven years—which include not only the trapping period of the fibrosis-producing dose of dust, but also the maturation period of the fibrosis, which periods, of course, overlap.

The amount of disablement produced by the development of asbestosis is surprisingly slight for a number of years. This is partly due to the character of the disease, and partly due to the nature of the work, which, in the majority of processes in the industry in which there is a risk of asbestosis, does not involve much physical exertion. Those affected may, and often do, continue at work with occasional intermissions laterally, due to exacerbations of bronchitis, until the condition is advanced, although increasing inconvenience from shortness of breath on exertion is experienced.

Usually these cases cease work a year or more before death, but sometimes a terminal bronchopneumonia, or other acute infection, commences while they are still at work, and there is no long period of invalidism.

It is remarkable to what extent the lungs can be affected and yet life in a fair degree of comfort remain. The reserve is, however, so slender that the addition of any burden to the system, in the form of a disturbance of health which would only slightly inconvenience a normal person, may overcome the remaining resistance and precipitate the fatal outcome. It is apparent, therefore, that while the onset of an acute respiratory infection is most ominous, the development of any disease which directly or indirectly throws an added strain on the heart or lungs is of serious import.

Generally on post-mortem examination of the lungs of asbestos workers one finds that the cases showing active tuberculosis fall into three groups: (1) Those with evidence of rapid tuberculosis and very slight or no signs of asbestosis;

(2) those with much fibrosis due to both diseases; (3) those with advanced asbestosis and a slight amount of active tuberculosis.

246. Miller, J. W. and Sayers, R. R.

The Physiological Response of the Peritoneal Tissue to Dusts Introduced as Foreign Bodies. *U. S. Pub. Health Rpts.* 49: 80-88, Jan. 19, 1934.

The physiological response of the body tissues to dusts of various kinds has been a subject of much interest in the past few years, and the opinion that the injury due to dust is chemical rather than physical in action has recently gained greater ground. Mavrogordato, Gardner, Gye and others have conducted experiments on the action of inhaled dusts. Kettle has studied the response to dusts injected into the subcutaneous tissues and intratracheally, and Policard has used the cornea and conjunctiva in his recent studies. In 1924, experiments were begun at the Pittsburgh station of the United States Bureau of Mines to determine the action and fate of various dusts when injected into the peritoneal cavity of guinea pigs. The conclusions reached at that time were that live animal tissue in all parts of the body tends to react in essentially the same manner to foreign bodies and that fibrous tissue is formed in the peritoneal cavity by quartz and is not formed by limestone and coal. This paper reports a continuation and elaboration of these earlier studies.

1. A definite quantity (0.2g) of dust in suspension was injected intraperitoneally into guinea pigs.
2. Dusts of two particle-size groups were used—one of screened material with particles less than 43 microns (325 mesh), and the other of air-separated material with particles varying from less than 2 to 8 microns in size.
3. The animals injected with the coarser material were examined 7, 14, 30, 56, and 112 days

after injection, and those treated with the air-separated material were examined after 7, 14, 30, 56, and 90 days.

4. The response caused by the dust in the peritoneal cavity was constant in all of the animals injected with an individual dust and could be classified as an absorption, a proliferative, or inert reaction.

5. In the absorption reaction the injected dust disappeared from the peritoneal cavity without the production of scar tissue.

6. In the proliferative reaction the nodules produced by the dust continued to increase in size up to 112 days after injection, the maximum duration of the tests in this series.

7. In the inert reaction the amount of injected dust remained approximately the same in the peritoneal cavity throughout the various periods, but the nodules became more flattened and fine particles of dust were carried over rather extensive areas in the peritoneum by phagocytes.

8. Calcite, limestone, precipitated calcium carbonate, gypsum, and cement exhibited an absorption reaction.

9. Quartz, chert and flint produced a proliferative reaction.

10. Soapstone, carborundum, jeweler's rouge, anthracite coal, bituminous coal, and precipitator ash were inert in reaction.

The tissue of the peritoneal cavity responds actively to a dust introduced as a foreign body, and this response is of such a character that it may be used as a basis for the classification of industrial dusts from a physiological standpoint. In this report, dusts of uniform chemical composition or those definitely known to produce or not to produce silicosis were used, and for this group the reaction occurring in the peritoneal cavity was uniform and constant for each dust. It seems probable, in view of the nature of the reactions, that dusts of mixed chemical

and 6 per cent finely ground clay from pitcher or broken ware. The free silica content approximates 20-25 per cent.

The casting room constitutes the essential dust hazard, because each caster must shake silica dust into his own moulds, dry sandpaper them and brush off the dust from his own moulds. He is constantly inhaling silica dust, and wears no mask. No forced ventilation system. Occasionally the floor is sprinkled with water.

106 employees, exclusive of the office-workers, were examined physically. In 58 employees, with an average duration of exposure to silica-laden air of $7\frac{1}{2}$ years, x-rays of the chest were taken; of these 5, or 8.6 per cent showed third stage or advanced fibrosis, the average duration of exposure being 16 years. 22, or 38 per cent, had second-stage fibrosis. The remaining 31, or 53.4 per cent, showed only slight fibrosis; five of the latter were confined principally to the hila. This report shows that

1: Inhalation of silica-laden air results in pneumoconiosis, a progressive, incurable occupational disease of the lungs.

2: In 58 cases reported from the pottery industry in which the concentration of dust is low, silicosis develops slowly—symptoms are latent, and appear many years after physical and x-ray signs of silicosis are demonstrable.

3: In advanced silicosis, total and permanent disability for work, and a high susceptibility to pneumonia and tuberculosis are being recognized by Industrial Accident Commission.

4: Prevention of inhalation and reduction in concentration of silica-laden air should receive more attention from the industries concerned, state legislatures and the medical profession.

287. Quarelli, G. and Velicogna, A.

Trachitis From Asbestos.

Med. d. lavoro, 25: 218-223, June 30, 1934.

This case of trachitis particularly interested Dr.

Quarelli as the only one of its kind ever published. The patient for a long time had attacks of convulsive coughing, variously diagnosed by physicians previously seen, none of whom had ever asked about her occupation. She was a single woman 42 years old, who worked in an asbestos factory as a spinner of the greenish variety of that mineral. Her only previous illness was a broncho-pneumonia following influenza in the 1918 epidemic; later she had a few light attacks of angina. The past three years she had suffered from this violent coughing, which had begun originally in a tickling of the throat. Later the coughing was accompanied by a feeling of retrosternal oppression. She had burning sensations, like those after a severe chill, followed by trachio-bronchial catarrh. Breathing was difficult at times and pre-cordial pain was then noticeable. These annoying symptoms had lasted three years, now lessening, now reviving in full force. She entered the University of Turin Clinic Jan. 10, 1934, and stayed till Jan. 29, all the time in bed in a half-sitting position, and not talking, to keep down the then almost incessant convulsive coughing. She insisted she had no fever; however, did have it. She was admitted on a diagnosis of bronchial asthma, but after the first few days auscultation showed no bronchial asthma whatever. In the factory she had worked an 8-hour day before a machine which spun the asbestos. No respirators were in use and she and her companion workers were "tormented by angry coughs," so she worked with her mouth firmly closed against the fine asbestos particles filling the air and penetrating the bronchi. Very numerous and admirably detailed examinations were given her at the clinic, the results, largely normal, are carefully recorded in this paper. She was a short woman with rosy complexion and well nourished. During her stay at the Clinic she complained of thoracic pain on right. The

coughing was continuous, morphine alone giving relief.

The objective and radiological examinations showed a slight heart enlargement toward the right, slight mobility at the pulmonary bases due to emphysema, accented hilary shadows in the pulmonary outlines, and tributary vessels with bilateral signs from tiny little speckles uniformly diffused throughout both lungs, the reaction thickening toward the hila, observable in pneumoconioses.

In the sputa were brownish-black particles, some of them needle-shaped, all of varying sizes, probably derived from the asbestos-filled air.

The case presented the signs of acute trachitis, with pulmonary emphysema, and heart insufficiency on the right side. There were no signs at all of tuberculosis or syphilis. From inhaling asbestos dust the patient had evidently an unusual form of asbestosis, hence the diagnosis, *Trachitis asbestos*.

Since the International Congress at Lyons in 1929 pneumoconioses have received especial attention in Italy, promoted by the interest and initiative of the Corporation Ministry and the Minister of Labor. Asbestosis has been particularly studied. Two interesting papers have been lately prepared upon it by Luisetto of Turin and Mussa of Cirie, doctors of industrial medicine in Piedmont, with original radiological contributions. In 1908 Scarpa of Turin was deeply impressed by the very serious asbestos statistics then existing and relating it to tuberculosis. In the 20 years since Scarpa, great advances have been made in the hygienic conditions of industry, the healthful homes and improved manner of life on the part of the industrials, helped on during and since the War by grants of money.

Investigations recently completed in Forno Rivara, a little region in Piedmont, by Dr. Quarelli and his colleague Bordi Trotti, will be

presented at the next International Geneva Conference. Carried out by numerous visits and with radiological control, observations have been made on an entire generation of workers in metal objects, which have been made in this region for a hundred years. The products are refined by sharpening on grind-stones, by the smoothing of rough edges, polishing with quantities of emery, these processes filling the air with clouds of silica dust. Through hygienic measures put through in this generation the silicosis mortality in the former tuberculous statistics of 70 per cent, has been reduced in current data to exactly 0 per cent.

The very fineness of asbestos fibres makes it possible to spin them much as glass is spun at Murano where the needle-like spicules often enter the skin of the glass workers. Asbestos is a mineral found in nature in fine silky fibres varying in color according to their chemical combination; often white asbestos may also be grayish, and even green or azure. Important strata occur in Europe: in Corsica, in Hungary, the Alps, the Pyrenees and elsewhere. It is a magnesium silicate usually containing also silicates of calcium and aluminum, with traces of oxide of iron. The Canadian asbestos which dominates international business is a crystalline hydrated silicate of magnesium, containing protoxide of iron. The Italian asbestos of Valtellina, the best for spinning into thread because formed of long fibres, is a silicate of magnesium and calcium ($\text{CaO.SiO}_2 + \text{MgO.SiO}_2$). As practically a non-conductor of heat and electricity, its special value is its non-combustibility, giving it multiple adaptations in industry during the last 30 years especially. But the ancient Greeks knew it, weaving it into textiles. In modern factories asbestos is comminuted by power-machines, separated by aspirators (usually in adjoining buildings) into long fibres deposited in certain rooms for spinning and weaving, while

the short threads are made into paper and cardboard.

Our patient improved but only on giving up her work and moving far away from the region, as we advised. She was to be observed from time to time lest asbestos particles penetrating deeply into the pulmonary alveoli, should inevitably cause lung asbestosis. These areas are below the defence provided by the violent coughing which sooner or later expelled the particles.

We also advise all asbestos workers to be protected by masks, and to wear an enveloping uniform, especially preventing their heads and clothing from carrying back to their homes any dangerous particles.

288. Quintana Otero, F. and Garcia Martinez, S.

Action of Inhaled Dust on the Morphology of the Blood.

Arch. cardiol. y hemat. 15: 98-108, March 1934.

In perusing the literature the authors have not found any work having reference to hematology in pneumoconiosis.

They illustrate monocytes and histiocytes found in the sputum of a cement worker. In the peripheral blood there is an increase of monocytes or rather of elements derived from the reticulo-endothelial system. Under normal conditions most hematologists consider these as monocytes.

There are certain differential characteristics of these cells in the circulating blood in pneumoconiosis. There are three distinct types: (1) Monocytes, characterized by nuclear chromatin and occasionally with nucleoli. (2) Cells which we call histiocytes, presenting a structure similar to the monocytes but with a more marked chromatic rete. The nuclear forms vary. The radical difference from the monocytes resides in the protoplasm. These types of cells occur more especially in advanced cases of pneumo-

coniosis. (3) Endothelial cells, a rather incorrect name since, more correctly, these cells are histiocytes in the final stage of decomposition. All three types are of reticular origin. The authors found them in studying the morphology of the blood in 56 workers in cement and other dusty occupations.

289. Rao, S. S.

Silicosis in India.

Brit. M. J. 1: 1094, June 16, 1934.

The Mysore Government recently appointed a Committee to investigate the prevalence of silicosis among the Kolar Gold Field miners. The Committee comprises the Chief Medical Officer of the Mysore Government, the local Medical Officer, and a physician of the Krishnarajendra Hospital, Mysore. The preliminary investigation reported silicosis as existing among the underground workers; this report submitted to Dr. L. G. Irwin, Chairman the Miners' Phthisis Medical Bureau South Africa, was confirmed on the pathological and radiographic evidence supplied. The Committee found the disease in incidence much smaller in the Kolar Gold Fields and the disease is longer in showing signs and symptoms than in South Africa. The percent of free silica contained in Kolar rock is 5-20, as compared with the much larger per cent, 43-98, obtaining in South Africa. The Senior Surgeon S. Subba Rao conveys these figures to correct a statement previously made in the *Journal* (April 14) that silicosis was "inexplicably unknown in the Kolar area, although quantities of quartz occur in the rock."

290. Rapado Pozas, A.

Pneumoconiosis and tuberculosis.

Progresos de la Clin, 42: 31-38, Jan. 1934.

This author distinguishes between simple silicosis and complicated silicosis in which it is complicated with tuberculosis (infectious silicosis of South Africa). The latter is divided into 2 sub-

307. Sappington, C. O.

Laboratory Methods for the Determination of Atmospheric Pollution Causing Pneumoconiosis. *Illinois M. J.* 66: 417-421, Nov. 1934.

The author gives a brief description of the laboratory methods used in the determination of atmospheric pollution causing silicosis, the most frequent specific type of pneumoconiosis. To secure the real value in assessing the relative health hazard in any given industrial environment, these laboratory methods must be used in connection with the information gained through a physical and hygienic survey of the working conditions, together with occupational analyses of employees thus exposed. When adequate information concerning environmental conditions has been gained by use of the above-mentioned procedures, comparisons may then be made with the criteria which have been suggested by official groups, thereby providing a basis for an estimation of the hazards involved in any given problem.

There are ample reasons for believing that laboratory and survey methods can only be properly used and interpreted by those who have had sufficient special training and experience to permit of real expertness. Similar study and survey procedures will be used more widely regarding various occupational disease exposures in evaluating the essential hazard, the prevention of disease and the rating of the efficiency of control devices.

To those who have had experience with the medical and legal aspects of silicosis, there has been considerable difficulty in establishing etiological relationships with reference to the kind and type of dust exposure; this same situation has obtained in reference to other occupational diseases, chiefly because the physician has had no incentive to adequately study environmental conditions in the various industrial plants, nor has he had the specialized training which is necessary to make such studies.

308. Sappington, C. O.

Pneumoconiosis.

Internat. J. Med. & Surg. 47: 399-403; 424, Oct. 1934.

There are two large groups concerned with silicosis: (1) persons occupied in taking products from the earth and (2) those engaged in processing or refining—as in sand-blasting, grinding, buffing, cleaning and polishing. There are probably in excess of one-half million persons exposed in American industry to this hazard.

The vital factors of the industrial environment concerned in the production of silicosis are 4: (1) The concentration of the dust; (2) the particulate size distribution; (3) mineralogical composition; and (4) period of occupational exposure.

Collected clinical experience points to the inevitable fact that the majority of silicotics sustain tuberculous infection.

Dyspnea appears to be the most common symptom. The use of serial Roentgenograms of the chest probably constitutes the most important single diagnostic procedure.

There is no specific treatment for silicosis. Some recent figures have shown that after removal from exposure, 20 per cent of silicotics in the first stage of the disease still progressed; in the second stage, 40 per cent, and all of those in the third stage.

The successful prevention of silicosis partakes both of medical and engineering supervision measures. There are two general methods in use, that of personal protection of the individual employed and, second, dust control.

Regarding the legal compensation aspects, it is the consensus that it is futile to set up legislation without the use of special medical boards to be used in conjunction with the laws.

309. Sappington, C. O.

Problems in Industrial Medicine.

Indust. Med. 3: 329-338, June 1934.

A perspective is given to industrial medicine, com-

monly thought of as a new form of medical work, by this author's reminder that it began in Padua in 1700 with Prof. Ramazzini's account of 43 different occupations and their accompanying diseases—a chapter being required for each occupation. Translated into English in 1705 and into French in 1711, this book remained the sole textbook for 100 years. In recent years International Congresses emphasize the subject's importance; the 6th was held in Geneva in 1931. Current problems are taken up by Dr. Sappington under 12 heads and treated in turn: (1) diseases from dust and (2) from chemical hazards; (3) legal medicine; (4) protective procedures; (5) evaluation of health hazards; (6) pre-employment and periodic examination; (7) the small industry; (8) health information for employees; (10) organization of industrial physicians; (11) undergraduate and post-graduate medical instruction; (12) cost of service. The "two definite dust diseases clearly recognized at the present time" are silicosis and asbestosis, a brief treatment being given on the problems presented by each. The industrial executive is hopefully mentioned as one of the major problems in industrial medicine, with responsibility for his reluctances placed on the industrial physician.

310. Sappington, C. O.

Use of Scientific Methods in Prevention, Control and Defense of Silicosis Cases.

Indust. Med. 3: 168-170, March 1934.

The author emphasizes improvements in the silicosis outlook due to application of improved scientific methods.

In dust analysis the impinger was devised in 1922 and a report made on its value as an instrument for collecting dust. In 1925 the relative merits of dust collecting instruments such as the sugar tube, the konometer, and the Owens' jet dust counter were compared with the impinger and the results published. Important dust survey counts and studies have been published.

Inspection alone of a plant will not give scientific information regarding the quantity and quality of dust and chemical exposures. A hygienic survey of the plant should first be made, including the kinds of machines or processes producing dust or toxic emanations; the equipment used to control these conditions and the type of work done by the man exposed. Physical and chemical determinations as to quantity and quality of fumes and dusts may then be made. The hazard may be definitely determined through comparison with the minimum amount of materials to which an employee may be exposed without harm or at least the amount to which good industrial practice can keep them.

Employers have incentives to know what their working conditions are in terms of scientific results. The tendency to compensate for occupational diseases, the establishment of specific codes in some states in an attempt to prevent such diseases, and wider knowledge of the effects of industrial environment on health and efficiency, have all aroused the interest of employers to find out whether potentially hazardous processes are being used in their plants and whether the working environment is affecting the health of their employees.

Certain standards of dustiness have been established by studies undertaken by the U. S. Public Health Service. There is a technique of dust sampling and dust analysis sponsored by this Service, constituting a national standard for comparisons with the results of various investigators and for use in standardizing plants.

The author points out four factors bearing on the contraction of silicosis from dust inhalation. (1) amount of dust in the breathing atmosphere, the concentration, that is; (2) the percentage of quartz contained in the aerial dust, the mineralogical composition of the dust, that is; (3) the size of dust particles, since particles beyond a certain size do not penetrate the air sacs of the lungs; (4) the

primary cause of silicosis, the production of fibrosis of the lungs by inorganic dusts, other than free silica, is now well recognized. The 1933 Factory Inspector's Report recorded results of a preliminary inquiry into the effects of French-chalk as producing lung fibrosis. French-chalk, a granular form of talc or steatite is essentially a hydrated magnesium silicate, extensively used in many industries, is very light, with powder readily flying. The literature on its possibilities as causing lung fibrosis is remarkably scanty, and indicates at present nothing like the fibrosis-producing power of asbestos dust under ordinary industrial conditions. The few examinations made so far do not disclose any appreciable disablement after exposure ranging from 9 to 32 years.

329. The Silicosis and Asbestosis (Medical Arrangements) Amendment Scheme, 1934.

Ministry of Labour Gaz. 42: 341, Sept. 1934.

The Home Secretary, August 4, 1934, made a further Scheme under Section 47, Workmen's Compensation Act, 1925, as extended by the Workmen's Compensation (Silicosis and Asbestosis) Act, 1930. [Statutory Rules and Orders, 1934, no. 889, H. M. Stationery Office, price 1d. net, 1½ and post free]. This order came into force September 1, 1934, amends that part of the Silicosis and Asbestosis (Medical Arrangements) Scheme 1931, which relates to the initial and periodic medical examination of workers in certain industries and processes involving exposure to silica or asbestos dust, which are included in the first schedule to the Scheme.

330. Silicosis and Carcinoma of the Lung.

Med. J. Australia, II: 168-169, Aug. 4, 1934.

Dr. Maxwell presented a case before the Victorian Branch of the British Medical Association at Melbourne, May 16, 1934, of a gold-miner, 59 years old, who had been a mine driller 10 years. Six years before presentation he had be-

gun to lose strength and weight gradually, 50 lbs. in the six years. The last 4 years he had developed symptoms, which after various detailed tests and the X-ray examination warranted diagnosis of third-stage silicosis. Biopsy after bronchoscopy revealed a probable lung carcinoma as cause of a dense area situated at the base of the right upper lobe of the lung.

331. Silicosis and Sand Blasting.

Foundry Trade J. 51: 91-92, Aug. 9, 1934.

Extremely important information on the silicosis evidence among sand blasters is here abstracted from the Annual Report of the Chief Inspector of Factories and Workshops for 1933. No other silicosis-producing industry shows such grave figures for the average working life-time of fatal cases, or any figures comparable to the sand-blasting trade, except the manufacturers of scouring powders, where incidentally, it is only fair to remark preventive measures extensively applied in the past few years have materially reduced the risk in that industry. Quoting American authoritative observations (Bloomfield and Greenburg) the atmosphere in a sand-blasting chamber may contain up to 3,104 million cu. ft. of particles. The general air of a work room in which sand-blasting chambers are located, may contain up to 118 million particles per cu. ft. The abrasive sand is highly siliceous in nature, some being pure flint. With the pressure up to 100 lbs. per sq. in., all chambers, cabinets, sand-collecting apparatus and helmets inevitably sustain serious and menacing leakage. The average duration of sand-blasters who had died of silicosis, is 11.8 years, as compared with 34.4 years for all fatal cases of silicosis irrespective of the causative occupation. The scouring powder workers last 11.1 years or even only 6.4 years. In the English Midlands there are 272 factories blasting with sandshot or with sand and shot mixtures. 618 persons are engaged in this work in that area. Details and statistics as to fatality, underlying causes, main

fire-brick clay; 30 per cent, too, from glass, porcelain workers and pottery workers. These data in detail constitute the basis of the author's conclusions.

360. Stewart, M. J.

Concentration Method for the Demonstration of Asbestos Bodies in the Sputum.

J. Tech. Methods. 13: 70-71, March 1934.

In 1929 the author published a brief note of a simple concentration method for demonstrating asbestos (asbestosis) bodies in the sputum of asbestos workers and others. The method, with modifications, has been in continuous use in his laboratory since that time, having given very satisfactory results.

About half an ounce of sputum is dissolved in an equal quantity of undiluted antiformin in a wide test-tube. This takes from a few minutes to an hour or so, and is aided by heat (37° C. incubator) and gentle agitation. Two or three ounces of water are then added and the mixture allowed to stand for a few hours or over night. The bulk of the supernatant fluid is now poured off and the remaining 12 to 15 cc. centrifuged at moderate speed for ten minutes. The amount of deposit which comes down varies in different specimens, but in any case the whole of the supernatant fluid may be poured off by simply inverting the centrifuge tube. The deposit and the trace of fluid which remain are thoroughly mixed by means of a fine pipette and transferred to an albuminised slide or slides. Too much force must not be used in the mixing process or at any previous stage, in order not to disintegrate any clumps of asbestos bodies which may be present. As a rule the whole deposit from half an ounce of sputum may spread over an area of one square inch of a microscope slide, or less; in any case a thick film is desirable. After thorough drying on a hot plate and final fixation over a Bunsen, the film is very gently washed in water, dried and mounted in Canada

balsam. The washing in water must be done with the greatest care, as in spite of the albuminisation of the slide, the film is very easily detached, especially if it is thick.

After washing and drying, the film may be treated with ferrocyanide of potassium and hydrochloric acid to demonstrate the Prussian blue reaction given by the bodies. It is then dried and mounted in balsam as before. Alternatively the centrifuged deposit may be covered with a small quantity of a 5 per cent solution of ammonium sulphide, when the bodies are colored black. No special staining is required, however, as the natural greenish-yellow, golden or brown color of the bodies renders them sufficiently conspicuous even under the low power. It is the practice of the author to examine films with the $\frac{1}{2}$ inch objective and $\times 10$ ocular, confirming the presence of asbestos bodies with the $\frac{1}{6}$ inch objective.

It is necessary to cleanse thoroughly all test-tubes, centrifuge tubes and pipettes after use, otherwise there is a risk of contamination of subsequent specimens from a positive sputum.

Asbestos bodies are demonstrable by this method in the great majority of asbestos workers, provided they have any expectoration. In the older workers, especially during a bronchitic attack and in the later stages of pulmonary asbestosis, bodies are present in profusion, but even in those who have only been engaged in the industry for a year or two, they may be demonstrable in small numbers. Their presence indicates merely that asbestos dust has been inhaled into the lung and that it has remained there long enough for "bodies" to form around individual fibres, a period of months, at least. It does not necessarily mean that there is pulmonary fibrosis. The finding of radially arranged clumps of bodies similar to those demonstrable histologically within the lung alveoli, has a much more serious significance. It indicates disintegration of living tissue, either by suppurative bronchopneumonia or tuberculous caseation, and

may be taken also as presumptive evidence of an underlying asbestosis.

Bodies somewhat similar to asbestos bodies but much coarser and more irregular have been recovered from the lungs of coal miners who had never worked in asbestos. The morphological differences are so great, however, that there is little or no risk of confusion.

361. Stewart, M. J.

Silica in Relation to Pulmonary Disease.
Edinburgh M. J. 41: 226-232, March 1934.

The author states that the 4 chief forms of pneumoconiosis encountered in England are silico-anthracosis, silicosis, asbestosis, and silico-siderosis—all due to the action of silica in some form or other. In silico-anthracosis and silico-siderosis it is suggested that the carbon and haematite dust, respectively, exert some sort of modifying effect on the chemical action of the silica or on the fibrotic process, which latter is diffuse and not nodular as in ordinary silicosis. Asbestosis is definitely due to the fibrous silicate, asbestos; in the other three the recent work of W. R. Jones suggests that another fibrous silicate, sericite, may be the causative agent, rather than uncombined silicon dioxide, as has hitherto been universally believed.

362. Stewart, M. J. and Faulds, J. S.

The Pulmonary Fibrosis of Haematite Miners.
J. Path. & Bact. 39: 233-253, July 1934.

A number of haematite miners in the West Cumberland mine field have died with a grossly fibrotic lesion of the lungs in which both silica and haematite dust are present in large amounts. 15 cases have been investigated in the present study, all of whom have died within the last three years. The lesion is a highly characteristic one, mainly on account of the haematite dust present, which causes the densely fibrosed areas to assume a bright brick-red colour. The fibrosis is diffuse rather than nodular, often massive, and always most intense in the upper

half of each lung. An associated or superimposed tuberculosis is the rule, being present in 11 of the 15 cases. A very characteristic clinical and radiological picture is produced, and the diagnosis can usually be made with reasonable certainty a year or more before the fatal issue.

The silica content of the lungs averaged 1.66 per cent of the dry weight in 14 cases. This may be compared with figures of 1.72 for a group of 8 cases of ordinary silicosis and 1.78 in a series of 7 sand-blasters examined by the same method. We are of the opinion that this lesion is a form of silicosis (sidero-silicosis) resulting from the inhalation of silica-containing haematite dust, generated by dry-drilling and shot-firing and that serious trouble only commenced when the old "hammer and jumper" gave place to the dry mechanical drill.

The use of the wet drill has diminished the risk to a certain extent only, as the speeding up of the drilling process has allowed blasting to take place at much shorter intervals, greatly increasing the dustiness of the atmosphere as compared with the period prior to 1913.

It is not possible at present to give even an approximate idea of the frequency of this condition. The number of persons under risk for varying periods of time during the past twenty years must run into some thousands and it is clear from enquiry in the mine-field that a certain proportion of them are suffering from this disease. The 15 fatal cases here recorded have died within the past three years, and it is unlikely that this includes all deaths from this cause during that period.

363. Stratton, R. C.

Industrial Dusts and Their Control.

Safety Eng. 68: 38, July 1934.

In the author's opinion one of the most important considerations for an engineer is, Housing. Where practicable, dusty manufacturing processes should be separately housed and isolated; every effort should be made to arrest dust at its source to pro-

Workmen's Compensation or other laws which are the legal basis of awards of compensation for occupational disease.

379. U. S.—Public Health Service.

Pulmonary asbestosis held compensable under Workmen's Compensation Act; North Carolina Supreme Court, *Rept.* 49: 1273-1274, Oct. 26, 1934.

The plaintiff had brought an action for damages due to pulmonary asbestosis contracted during 5 months of the 15 months he had worked for his employer, who had maintained no dusting or suction system such as was approved and in general use in other asbestos plants. The plaintiff's contention of occupational disabling disease not coming under the Workmen's act, the suit was brought at common-law as his only remedy. The Court discussed the several definitions of occupational disease, taking the view the plaintiff had not such a disease, but had sustained an accidental injury, compensable under the compensation law. The element of time in the Court's opinion was negligible. Had the employee accidentally breathed noxious fumes during work hours for 5 days, the injury would be accountable as such; similarly if he breathed such fumes five months, more or less.

380. U. S. Surgeon-General's Office.

Annual Report of the Surgeon General of the Public Health Service of the United States, 1934. 143 pp.

A series of injections of dusts of known chemical composition have been made intraperitoneally into guinea pigs to determine the physiological response. The investigation has shown that the response is uniform and constant for a single dust, but different for each of three groups of dusts, and sufficiently differentiated to afford a means of classifying them from the standpoint of biological reaction. Other dust studies have included health of anthracite coal

miners, silicosis among granite quarriers, pulmonary infection in pneumoconioses, effect of inhaled marble dust as observed in Vermont marble finishers, and a study of talc workers.

Health of anthracite and coal miners.—On request of the Governor of Pennsylvania, an investigation has been made of the effect of the dusts of hard-coal mining on the health of the workers. Studies were made of the occupational environment, including determinations of the dust concentrations for different operations. Twenty-seven hundred persons, the entire number at 3 mines located in the districts of the anthracite fields, were given medical examination and x-rayed. A supplementary study was made of disabled miners.

The physiological response of the peritoneal tissue to dusts introduced as foreign bodies.—A series of injections of dusts of known chemical composition (some definitely known to produce and others not to produce silicosis) was made intraperitoneally into guinea pigs to determine the nature of the response. The first part of the study was completed during the year and appeared in the Public Health Reports for January 19, 1934.

It was concluded that dusts of the absorption group could be considered least harmful, those of the proliferation group as being productive of silicosis, and those of the inert group as responsible for the various other pneumoconioses in which there is a large deposition of mineral material in the lungs with comparatively little fibrosis.

380. U. S. Surgeon-General's Office.

Annual Report of the Surgeon General of the Public Health Service of the U. S.

Silicosis among granite quarriers.—The effects of the inhalation of granite dust generated in granite quarrying were studied and a report published in the Public Health Reports for June 8, 1934.

Diseases Committee of the Medical Research Council and in other quarters. This I believe to be the best method of attacking the problem of this disease and at the present stage of the investigations I do not think the suggested committee would serve any useful purpose. At a later stage, however, I will take the suggestion of the Hon. Member into consideration.

Mr. T. Smith asked whether there was any evidence of stone dusting being a contributory cause of silicosis, and whether it was regarded as such.

Mr. E. Brown: Not that I know of.

Mr. Turton asked if it would not be simpler to make silicosis an industrial disease under the Workmen's Compensation Act.

Mr. Brown: No. There are many other issues involved.

383. Williams, J.

Silicosis in South Wales. House of Commons, sitting of July 4, 1934.

Lancet. 2: 110, July 14, 1934.

Dr. John Williams asked the Secretary for Mines the number of men certified for silicosis in the county of Carmarthen and in the rest of the South Wales coalfield; and the number of such cases where death had resulted from this disease.—Mr. Ernest Brown: I am informed by the Home Office that between June 1st, 1931, when the Medical Board under the compensation scheme was appointed, and May 31, 1934, 105 miners were certified by the Board as disabled from silicosis in the county of Carmarthen and 279 in the remainder of the South Wales coal field; and that the number of deaths certified by the Board were 26 and 57 respectively.

384. Withdraw Appearance.

Ottawa Ill. Repub.-Times. Nov. 22, 1934.

Sol Andrews, Chicago attorney, today withdrew his appearance as counsel for the plaintiffs in the suits that had been started against the

Ottawa Silica Sand Company, and Gilbert J. Ward of this city entered his appearance as attorney for the plaintiffs.

One suit was started by the late Lyman Jameson of Grand Ridge prior to his death to collect damages for an alleged case of silicosis. The second suit was started by Mrs. Cora Jameson, widow of Lyman Jameson after his death, asking a death claim against the company, charging that Jameson's death was caused by silicosis contracted as a result of his employment.

385. Wood, W. B.

Pneumoconiosis. (A discussion held at the Institute January 19, 1934.)

Paper II. Pulmonary Asbestosis.

Brit. J. Radiol. 7: 277-280, May 1934.

Pulmonary asbestosis might be described as mono-symptomatic, for one symptom—dyspnoea—dominates to such a degree that it is of this alone that the patient complains. Physical signs are slight except in the later stages. The sputum contains the characteristic golden-yellow asbestos bodies. The lungs are the seat of a diffuse fine fibrosis, especially at the lung bases. The nodules characteristic of silicosis are not found. The pleura is often adherent, and the adhesions tough. Apical adhesions are often found.

Pulmonary asbestosis results in a fine fibrosis which specially affects the bases of the lower lobes and radiology fails to reveal its earliest manifestations.

Pulmonary tuberculosis is so often associated with asbestosis that it seems probable that the association is more than accidental. The radiographic appearances of the combined diseases present few difficulties in diagnosis. Bronchitis or broncho-pneumonia are the common causes of death, but even the later complication is not always fatal when it attacks a sufferer from advanced asbestosis.

386. Wood, W. B. and Gloyne, S. R.

Pulmonary Asbestosis. A Review of 100 Cases. *Lancet*. 2: 1383-1385, Dec. 22, 1934.

In 1928 the authors published a preliminary report of their studies, and in 1930 reported their findings in a series of 37 asbestos workers, and are now reporting on 100 cases.

Of 67 women in their series, 24 were spinners, weaving, 5; mattress making, 9; carding and opening, 11; and miscellaneous or mistated, 18. 22 began their employment before the age of 18. The shortest exposure was 6 months and the longest 15 years.

Of 33 men—about two-thirds of them were employed in the same factory as the women. The others included an elderly man, the owner of a small factory, a van boy—who assisted in his spare time mixing powdered asbestos in an open yard; a middle-aged boiler riveter who had served his apprenticeship as a youth in a shop when asbestos was used for logging pipes, and a man who had been employed handling asbestos mattresses in the open air at an aerodome. The shortest exposure was 11 months and the longest 40 years (clinical and administrative work). Apart from pneumonia and pulmonary sepsis and complications arising from tuberculosis, there have been none definitely attributable to the asbestosis with the exception of two instances of spontaneous pneumothorax diagnosed by x-ray, and apparently unassociated with tuberculosis, which may have been due to the rupture of emphysematous bullae.

387. Workers' Compensation at Broken Hill.

Chem. Eng. & Min. R. 26: 261, April 5, 1934.

As the result of an investigation by the N. S. W. government into payments made under the compensation act, the ministry decided to stop payments to dependents of beneficiaries whose death was not caused by the statutory diseases under the act, viz., pneumoconiosis or tubercu-

losis. The dependents of 29 men were affected. The total number of recipients is 654 and the amount disbursed annually is £140,000 of which the mining companies and the government each contribute half. In addition payments to 157 partially incapacitated beneficiaries were reduced to the amount of the basic wage declared by statutory authority in the state or country in which the workers reside. In protest of these amendments in payments a one-day strike took place in Broken Hill on March 20 and all mines were idle. The government is now reconsidering the question.

388. Workmen's Compensation Acts, 1925-34.

Various Industries (Silicosis) Amendment Scheme, 1934.

Ministry of Labour Gaz. 42: 426, Nov. 1934.

The Home Secretary, on 22nd October, 1934, made a further Scheme under section 47 of the Workmen's Compensation Act, 1925, as extended by the Workmen's Compensation (Silicosis and Asbestosis) Act, 1930, the effect of which is to extend the Various Industries (Silicosis) Scheme, 1931, to cover employment in any operation underground in a coal mine.

The Order applies to workmen employed in any such operation at any time on or after the date of the Order.

389. Workmen's Compensation (Occupational Diseases) Convention (Revised) 1934.

Labour Gazette (Canada). 34: 672-674, July 1934.

The International Labour Organization of the League of Nations at its 18th Session, June 4, 1934, adopted a Draft Convention "which may be cited" under the heading given above. Articles 1 and 2 are given by the Labour Gazette (p. 672 loc. cit.) showing what numbers of the Organizations agree to in ratifying this Convention. The Schedule following lists diseases and toxic substances, under which silicosis appears "with or without pulmonary tuberculosis, pro-