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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PART I

International Abstracts, Extracts and Reviews

mistakenly classified as "first stage" or "ante-primary" were really not silicotic at all under the limitations imposed by the law. Due to several circumstances the Company presently found itself at the end of 1932 with an estimated liability of several million dollars, most of it outstanding. Their honest efforts to co-operate with the law and the government, complicated with inexperience on the part of those diagnosing the silicosis stages, brought about situations in which nobody was satisfied, and the details of which deserve wider publication. Finally in 1933 the Act was amended. "Ante-primary" and "primary" classifications were detailed, compensation was made payable only for disability, which means actual impairment of working capacity; men with only a moderate degree of silicosis were allowed to continue underground work which was the only kind they knew, and which they had been transferred from, under the previous Act, to continual discontent and even misery; the Industry was relieved of unlimited liability in respect of progression, by providing that the claimant must make and establish his claim for disability within two years of leaving the employment at which he was exposed to dust, to entitle him to compensations. To this writer, silicosis is essentially an employer's problem. The reasons for this open stand are interestingly and usefully given. Efforts made in Ontario to allay the dust and combat the incidence of silicosis, also tuberculosis, are treated in closing, together with the physical requirements now insisted on for men undertaking to work in the industry. A rigid initial physical examination, and by the x-ray, eliminates those likely to be susceptible to silicosis or tuberculosis.

18. **Baybutt, J. W.**

A Cloth Filtering Separator for Very Fine Dust. *Amer. Artisan.* 103: 12-13, Dec. 1934.

For all really efficient dust removal, this engineer considers some form of cloth-filtering material

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 outcrop 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 fac-

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. **Bénassy, M. P.**

Tuberculous Infection and Silicosis.

Bull. Méd. 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;

84. Complete Silicosis Hearings.
Hurley, Wis., Miner. Oct. 19, 1934.

Another series of compensation hearings involving silicosis was completed at the Court House on Wednesday afternoon. Nine cases were heard, the complainants all being employees or former employees of the Montreal Mining Co. Al Flint, a representative of the State Industrial Commission, presided at the hearings.

85. Cooke, W. E.
Silicosis: Modern Views.
Brit. M. J. 2: 878-879, Nov. 1934.

This paper was presented at a pathological meeting of the Liverpool Medical Association, held Oct. 25, 1934, and urged reconsideration of the terminology applying in the present numerous conditions included under the title, silicosis. Causative factors were discussed in the light of the recent work of W. R. Jones and W. Tideswell. The effect of the grain-size on SiO_2 solubility, the size of lung alveoli, presence of insoluble minerals i. e. asbestos fibers and fusain spicules in asbestosis and coal miners' lung, and the production of the curious bodies in these diseases and in normal lungs were considered. Photomicrographs were shown of potash felspar undergoing metamorphosis into secondary white mica, sericite, and coal-measure shale and mine dust demonstrating sericite, and quartz grains present, Badham and Taylor's views on sillimanite as producing silicosis in New South Wales were presented and their chemical analyses of the lungs of various workers were compared with lung analyses of other observers. Lung sections as examined by polarized light were exhibited and results of repeating Watkins-Pitchford and Moir's work in this connection, offered.

Conclusions arrived at, indicated a balance of probability, in present insufficient data, for chemical rather than mechanical action in the several forms of pneumoconiosis. Until the silicates

(sericite, sillimanite, asbestos) and silica in its forms of quartz, chert, flint, etc. were isolated in a pure state and investigated as to their plasma solubilities, together with tissue reactions to their presence, no progress in silicosis etiology was possible.

Prof. J. Henry Dible discussed the paper in detail especially in relation to the impressive sericite views of W. R. Jones. He was convinced that silicosis, arose in other situations than those with only sericite present, an example cited being the recently published cases of sidero-silicosis of Professor M. J. Stewart, in which sericite could not be demonstrated.

Dr. C. O. Stallybrass referred to the radical changes in view-point respecting silicosis occurring in the last ten years, as largely due to Dr. Cooke's work on asbestosis, Dr. Cooke was also complimented not only as a skilled pathologist but for his exceptional knowledge of microscopy and mineralogy enabling him to deal with this border line disease of science and medicine.

56. Copeland, R. S.

Pneumoconiosis.

Chicago Herald and Examiner. Nov. 26, 1934.

Within recent years adequate legislation and protective measures have been provided for the safety of miners and other workers in hazardous industries. For a long time occupational diseases were a menace to the health of the individual. "Silicosis" is one of these formerly dreaded diseases.

When miners are engaged in work on siliceous rock, as in metal mining, coal mining, quarrying or sandstone or granite, tunneling, excavating, sand blasting, stone carving, grinding or polishing, they breathe dust laden with silica. If this dust is inhaled and deposited within the lungs, a severe irritation or inflammation of the lung tissues occurs.

Victims of this disorder become susceptible to chronic bronchitis. This soon leads to repeated

removal apparatus to collect the dust from swing-frame grinders in foundries, without restricting the movement of equipment, a problem long confronting foundries. An illustration with description and explanation, accompanies. In connection with its good house-keeping program, this same company employs a portable vacuum cleaner of the industrial type to prevent accumulation of dust and dirt in its powdered coal plant and other foundry departments. The electrically driven unit is used to clean off walls, tops of bins, runways of buildings and other places difficult to reach. The walls of the building have been painted white.

11. **Dust Research-Insurance Company Support.**
Nat. Underw. 38: 25; 28; July 19, 1934.

One of the three places in the United States where actual research is being conducted on the dust hazard and dust diseases, is Saranac Lake, New York, where Dr. Le Roy Gardner and colleagues have such a Laboratory. The Metropolitan Life, the American Mutual Alliance have decided to contribute financially to the support of this Laboratory. The American Mutual Alliance is already contributing $15,000. The National Bureau of Casualty and Surety Underwriters is considering a contribution. Dr. Gardner and his Assistant Director, Donald Cummings held a National Insurance Conference by invitation at their Laboratory. Insurance men in considerable numbers came, representing life insurance, mutual and stock insurance; scientists, doctors, technicians were also present. The state of Wisconsin sent all the members of its Industrial Commission, and an Assistant Attorney General.

The part of Dr. Gardner's work especially arousing the interest of practical insurance men seems to be his experiments based on the theory that certain oxides in contact with free silica may form a mixture counteracting or modifying the injurious effects of silica.

Another experiment of Dr. Gardner seeks to determine whether a person who is silicotic before

becoming tuberculous is more dangerous than a person tuberculous before he becomes silicotic.

85. Dyson, J. M.

Pulmonary Heart Disease in Pneumoconiosis.

Am. Heart J. 9: 764-770, Aug. 1935.

This paper is concerned with pulmonary heart disease resulting from pneumoconiosis in anthracite coal miners.

A series of 18 cases of its occurrence in pneumoconiosis is presented to illustrate incidence and symptomatology.

The disability and incidence indicate that pulmonary heart disease is not a rarity in anthracite coal miners, and must not be neglected.

It is urged that pneumoconiosis, because of its great economic and industrial importance, be more generally considered an important cause of pulmonary heart disease.

No attempt is made to explain why pulmonary heart disease occurs in some cases of marked pneumoconiosis, but not in others equally marked.

86. E. R. H.

The Dust-Testing Equipment Migos.

Am. J. Pub. Health. 24: 940, Sept. 1934.

The manufacturer of the instrument Migos (Arthur Goldberg, Praha, XII, Caslavska 3), which is described herein, claims that it may be used for determining the quantity, quality, and density of a (suspended) dust, while at the same time making it possible to register dust tests so that comparisons may be made, and control and supervision of the hazard put on a scientific basis. It is stated that the equipment as described will take 15 consecutive tests at a given operation. In addition to the instrument itself, a microscope must be used for examining the dust fields secured at tests.

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 1½ 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-ladened air or gases.

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 pneumonoconiosis. 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.

11. 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.

12. 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.

93. Evans, W. A.

Silicosis.

Chicago Daily Tribune. March 28, 1934.

Two great groups are very much interested in silicosis. One is the employers who are conducting industries in which there is exposure to dust. The other groups consist of the thousands of people who work in these industries. Lawyers and legislators make up another group—one that buzzes around the other two.

The industries, especially interested, are: pointing of stone and brick houses, sandblasting, foundry work, mills, castings, sand mining, sandstone work, glass, mining, particularly underground work in quartz [and, in lesser measure, granite], coal mining, asbestos mining, and metal grinding and polishing.

The main interest is in exposure to silicates, a sand compound. However, under such names as anthracosis, siderosis, asbestosis, and pneumonoconiosis, are embraced disorders due to exposure to various kinds of dust, some of which contain no silicon or silicates. Organic dusts such as mill dust, flour dust, and cotton dust are far less harmful. The dust from lime rock, limestone, and lime seems to be far less so.

At one time special stress was put on the number of dust particles in a cubic foot of air, the size of the particles, and the sharpness of their edge. These qualities, while still thought to be of some importance, are not considered as determining as they were once thought to be. However, we still stress the importance of small particles as against large ones.

Opinion now is toward the chemical properties of dust. The theory is that it acts much as does lead. It does most of its harm when it goes into solution. Like lead, it is thrown out of solution and deposited in certain parts of the body. Later it is absorbed and secreted.

As is the case in lead poisoning, the progress of the disease, silicosis, does not stop at once when expo-

sure to dust is stopped. It may go on during the period of subsequent absorption and excretion. The harm done in silicosis is done to the lungs. It causes the fibrous tissue of the lungs to thicken up. Breathing becomes rapid and difficult. In time, extra work is thrown on the heart as well as on the lungs. The heart beat, as well as the breathing, becomes labored. Eventually the digestion suffers. In many cases the ground work is laid for consumption and the pneumonias.

16. **Evans, W. A.**
Silicosis.
Chicago Daily Tribune. May 23, 1934.

The most interesting of all the newer industrial diseases is silicosis. In the aggregate, in all the world, a billion dollars is being sought through the courts as damages for silicosis. We hear of suits for an aggregate of thirty million dollars in one state alone. The disease is not on the list of those that come under the compensation act and, in consequence, suits for damages have become the principal racket of the times. Probably nine-tenths of the suits are fakes, but they must be vigorously and expensively fought. When a large verdict is awarded a very small part of it reaches the person who alleges that he has been harmed. Certainly ninety per cent of all awards in and out of court goes to racketeers.

But there could not be so much smoke without some fire. There must be genuine disease caused by dust, and it must do harm, and there should be just compensation. On this point there is no question among honest men, whether they are employers, employees, or lawyers.

A fairer way to compensate those who are injured must be found and racketeers must be eliminated. Furthermore, a way must be found to prevent the disease, and when found it must be employed. Health departments are engaged in studying the question of prevention.

Twenty years ago it was thought that all rock dust

caused it. An investigation made by Hayhurst in Illinois proved that lime rock dusts did not.

The next theory was that it was caused by exposure to dust that contained sand or silicon and silicates. Now comes Dr. W. R. Jones with a theory that it is caused by only one kind of silicate. This is a silicate of aluminum and potassium. The crystals that cause it, he thinks, are very fine fibers. The tendency to fiber arrangement of crystals we know best in the form of asbestos. And one of the better known forms of silicosis is due to exposure to asbestos and is known as asbestosis. Dr. Jones thinks the presence of very fine fibrous crystals is responsible for much of the harm. The particular chemical substance responsible is known to geologists as secondary mica.

In his investigation he found that in mining fields where there was much secondary mica in the dust the workers had a great deal of silicosis. In other fields rich in other forms of silicates and silicon in the rocks and dust there was little or no silicosis. Silicosis is a disabling disease, but it is not fatal until after it has been present many years. It lays the foundation for consumption, and this secondary disease causes a good part of the deaths among silicotic subjects.

Dr. Jones secured a fair number of autopsies on persons who had died either from silicosis or silicotic consumption. He examined the lungs for peculiar appearances due to secondary mica. He also burned these lungs and weighed the ash and searched for fiber crystals of silicate of aluminum. He found them in quantities great enough to confirm his theory. If it is found that he is correct the field is still further narrowed. Every time we narrow it we concentrate attention on the cause and increase the chance of finding a method of prevention.

95. Exhaust Code Committee Urges Representation of All Interests.

Safety Eng. 68: 212, Nov. 1934.

The Exhaust Code Sectional Committee of the American Standards Association was organized

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